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Celiac Disease

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Preface

The Agency for Healthcare Research and Quality (AHRQ), through its Evidence-Based Practice Centers (EPCs), sponsors the development of evidence reports and technology assessments to assist public- and private-sector organizations in their efforts to improve the quality of health care in the United States. This report on *Celiac Disease* was requested and funded by the Office of Medical Applications of Research, National Institutes of Health (NIH) for the Consensus Development Conference on Celiac Disease as well as the National Institute of Diabetes and Digestive and Kidney Diseases, NIH. **Marian D. James, Ph.D., served as AHRQ's Task Order Officer in charge of overseeing the report development process.** The reports and assessments provide organizations with comprehensive, science-based information on common, costly medical conditions and new health care technologies. The EPCs systematically review the relevant scientific literature on topics assigned to them by AHRQ and conduct additional analyses when appropriate prior to developing their reports and assessments.

To bring the broadest range of experts into the development of evidence reports and health technology assessments, AHRQ encourages the EPCs to form partnerships and enter into collaborations with other medical and research organizations. The EPCs work with these partner organizations to ensure that the evidence reports and technology assessments they produce will become building blocks for health care quality improvement projects throughout the Nation. The reports undergo peer review prior to their release.

AHRQ expects that the EPC evidence reports and technology assessments will inform individual health plans, providers, and purchasers as well as the healthcare system as a whole by providing important information to help improve health care quality.

We welcome written comments on this evidence report. They may be sent to: Director, Center for Outcomes and Evidence, Agency for Healthcare Research and Quality, 540 Gaither Road, Rockville, MD 20850. **Questions regarding this report should be sent to epc@ahrq.gov.**

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Author Contribution

Dr. Alaa Rostom was the lead investigator. He was involved in all aspects of the study design, management, planning, analysis and write-up, including article screening, data extraction, quality assessment, statistical analysis, and report write-up. Drs. Catherine Dubé and Ann Cranney were the second investigators. Dr. Catherine Dubé was involved in all aspects of study design, and planning and organization, including task management, article screening, data extraction, and quality assessment. She was the lead writer of Celiac 2 and 3. Dr. Ann Cranney was involved in all aspects of study design and planning, including article screening, data extraction, and quality assessment. She was the lead writer of Celiac 4, and oversaw the screening and data extraction for Celiac 3, 4, and 5. Dr. Navaaz Saloojee was involved in study planning, article screening, data extraction, and quality assessment, and was the lead writer of Celiac 5. Dr. Richmond Sy was involved in study planning, article screening, data extraction, and contributed to the writing of Celiac 4. Drs. David Mack and Dilip Patel were involved in study planning and article screening, in addition to being content experts in pediatric and adult celiac disease, respectively. They also reviewed and advised on the report write-up. JoAnne McNeal was involved in article screening and data extraction for Celiac 1 (serology) and Celiac 2 (prevalence).

Dr. David Moher was involved in all aspects of study design, management, planning, analysis, and write-up. He was the methodological content expert and reviewed and advised on all report conduct and documents. Chantelle Garrity was involved in all aspects of project planning and management, including liaison with all key partners. She oversaw the screening progress, document retrieval, and assisted in report management, review and write-up. Margaret Sampson was the lead information specialist and was involved in all aspects of the search strategy/key word design and refinement, in association with the information specialists at the NLM. She was involved in all aspects of article database management, including article retrieval, set-up of the online computerized SRS article screening and extraction system, and the development and write-up of the QUOROM Flow. Li Zhang was the information specialist involved in all aspects of SRS system management, article retrieval, and implementation of the SRS system. Dr. Vasil Mamaladze and Fatemeh Yazdi performed the data extraction of Celiac 1–serology and Celiac 2–prevalence. Irene Pan performed data extraction and quality assessment of Celiac 2–prevalence.

Structured Abstract

Context. Celiac disease (CD) is a disorder of small bowel malabsorption. It is characterized by mucosal inflammation, villous atrophy and crypt hyperplasia that occur upon exposure to gluten, and clinical and histological improvement with withdrawal of gluten from the diet. The classical presentation of CD has now been shown to be less common than silent or atypical presentation, in which patients do not have intestinal symptoms. Untreated CD is associated with multiple important short- and long-term complications including nutritional derangements, anemia, reduced bone density, as well as intestinal lymphoma. In the vast majority of patients, CD is effectively treated with dietary modifications that eliminate gluten. Mounting evidence suggests that CD is actually considerably more common than previously believed and, therefore, this disorder warrants consideration for screening of at-risk patients, as well as possibly the general population.

Objectives. To conduct a comprehensive systematic review on five areas of CD: (1) sensitivity and specificity of serological tests; (2) prevalence and incidence of CD; (3) CD associated lymphoma; (4) consequences of testing for CD; and, (5) interventions for the promotion and monitoring of adherence to a gluten-free diet (GFD).

Data Sources. Staff of the National Library of Medicine performed a series of searches in support of the literature review of CD. Searches were run in the MEDLINE® (1966 to Oct 2003) and EMBASE (1974 to Dec 2003) databases for each of the five objectives and their respective sub-objectives separately.

Study Selection. Study selection for each objective was performed using three levels of screening with predetermined increasingly more strict criteria to ensure that all relevant articles were captured. Following a calibration exercise, two reviewers independently screened all studies using a web-based system allowed automatic identification of review disagreements. These disagreements were resolved by consensus.

Data Extraction. For each CD objective, a detailed and standardized data abstraction form was developed. For each objective, data abstraction was conducted by one reviewer and verified by another. The extracted data was further verified by one of the principal investigators. Quality assessments were performed using specific instruments for each of the included study types.

Data Synthesis. The data obtained from this review fell into several broad categories, which correspond in large part to the individual study objectives. Data for the sensitivity and specificity of each serological marker was considered separately, and studies were further divided according to the age group of the study population. Attempts were made to identify, explain, and minimize clinical and statistical heterogeneity in the included studies. A Pearson's Chi Square with n-1 degrees of freedom, where n represents the number of included studies in an analysis, was calculated to assess statistical heterogeneity. Pooled estimates were only calculated if clinically and statistically appropriate. In situations where pooling was not performed, a qualitative systematic review was conducted.

To produce clinically useful pooled statistics, a weighted mean of the overall sensitivity and specificity from the included studies was calculated, along with 95% confidence intervals (CIs). The pooled estimates for the sensitivity and specificity were compared with a summary receiver operating characteristic (ROC) curve, calculated for the same group of studies as a second check of the estimates.

Results/Conclusions. This report has provided a systematic review of five broad areas (and corresponding sub-areas) of CD. Perhaps one of the most important findings of this report is the significance of how one chooses to define CD in the era of serological testing, and how this apparently clear-cut task has profound implications on all the results presented in this report. Specifically, can CD be diagnosed solely on the basis of serology? Is some degree of villous atrophy necessary for a diagnosis of CD. These questions have important implications downstream of the diagnosis as well. For example, do CD patients without symptoms or villous atrophy have the same risk of complications as those with villous atrophy. Is serological improvement on a GFD sufficient to reduce CD complications, or must there be documented histological improvement, and what degree of histological improvement is necessary?

The results of the Celiac 1 objective suggest that in the era of EMA and tTG antibody testing, AGA antibody testing in both children and adults has a limited role. The sensitivity and specificity of EMA and tTG are quite high (over 95% for sensitivity, and close to 100% for specificity), as are their positive and negative predictive values; however, one has to be aware that the reported diagnostic parameters are taken from studies in which the prevalence of CD was, for the most part, much higher than that seen in usual clinical practice. The positive predictive values reported for these tests will certainly not be as high as that reported when these tests are used to screen the general population. The bulk of the evidence on the diagnostic characteristics of these tests was derived from studies that defined CD as having at least some degree of VA.

HLA DQ2/DQ8 testing appears to be a useful adjunct in the diagnosis of CD. The test has high sensitivity (in excess of 90%-95%), however, since approximately 30% of the general population, and an even higher proportion of "high-risk" subjects (e.g., diabetics and family members) also carry these markers, the specificity of this test is not ideal. The greatest diagnostic utility of this test appears to be its negative predictive value.

Biopsy itself, when used with a strict cut-off requiring villous atrophy, appears to have high specificity, but poor sensitivity. Using a lower grade cut-off clearly improves sensitivity, but because of the wide differential of causes of histological lesions similar to Marsh I to IIIa, the specificity suffers. The use of histomorphometric measures such as quantification of gamma delta positive intraepithelial lymphocytes ($\gamma\delta$+ IELs) are likely to allow for the use of lower grade cut-offs, while maintaining reasonable specificity. Ultimately, a trial utilizing multiple diagnostic tests in an attempt to capture as many CD patients in a clinically-relevant population as possible, along with a time dimension such as a response to a GFD or gluten challenge, is required to fully assess the diagnostic characteristics of biopsy alone. This type of study would be able to characterize the false-positive and false-negative rates, provided that all studied patients are followed forward in time.

The included prevalence studies demonstrated important differences between the studies including, execution, tests for prevalence assessment, and patient sampling. Thus, results have to be interpreted in the light of some of the limitations that have been identified regarding the diagnostic performance of the tests for CD. Nonetheless, the results of this report suggest that

CD is a very common disorder with a prevalence in the general population that is likely close to 1:100 (1%). Several high-risk groups with a prevalence of CD greater than that of the general population have been identified and include: those suspected of having CD; family members of CD patients; type I diabetics; and, those with iron-defiency anemia (IDA) or low bone mineral density (BMD). Additionally, the review identified many other high-risk groups, including those with Down Syndrome, short stature, and infertility, to name a few. Their inclusion was however, beyond the scope of this report

The results of this report confirm that, apart from a few limitations, there is a strong association between CD and GI lymphoma. The report identified standard incidence ratios (SIR) for lymphoma that ranged from 4 to 40, and standard mortality ratios (SMR) that ranged from 11 to 70. A diagnostic delay—in particular a diagnosis of CD in adulthood as apposed to in childhood—is associated with poorer outcomes. Fortunately, several studies suggest that adherence to a GFD reduces the risk of lymphoma in CD patients.

The consequences of testing for CD in at-risk and symptomatic patients appears to be more straightforward, since these patients appear to be more compliant with a GFD and would be expected to benefit from this intervention. The data is less clear for asymptomatic screen-identified patients, particularly those who have truly silent CD and/or don't have fully-developed villous atrophy. On the one hand the outcome of such patients has not been extensively studied, and on the other hand compliance with a GFD appears problematic, particularly for those diagnosed in adulthood.

Finally, no specific interventions have been identified that promote adherence to a GFD, but education of patients and family members about CD and about the intricacies of a GFD, and participation in local celiac societies, has been shown to improve compliance. Although somewhat controversial, biopsy monitoring of adherence to a GFD appears to be important, since improvement in histological grade has been associated with improved BMD, IDA, and nutritional status. The serological markers appear to be adequate for detecting gross dietary indiscretion, and respond to a gluten challenge, but appear to have poor sensitivity for detecting lesser degrees of dietary indiscretion, and inadequately correlating with histological improvement at least in the short-term. It should, however, be noted, that we could not identify a controlled study that objectively determined the level of histological improvement that would be associated with improved outcomes, and this is an area for future study. Nonetheless, based on this report it would appear that follow-up biopsy, at least 1 year after a GFD in adults to document improvement of the histological grade, would be valuable.

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Celiac Disease

Summary

Introduction

Celiac disease (CD) is a disorder of small bowel malabsorption. It is characterized by mucosal inflammation, villous atrophy, and crypt hyperplasia, which occur upon exposure to gluten, and clinical and histological improvement with withdrawal of gluten from the diet.[1-4] CD—also referred to as celiac sprue, gluten-sensitive enteropathy, non-tropical sprue, in addition to a host of other names—is thought to result from the activation of both a cell-mediated (T-cell) and humoral (B-cell) immune response upon exposure to the glutens (prolamins and glutenins) of wheat, barley, rye, and oats, in a genetically susceptible person.[5,6] Genetic susceptibility is suggested by a high concordance among monozygotic twins of close to 70 percent,[7] and an association with certain type II human leukocyte antigens (HLA).[8,9] HLA DQ2 is found in up to 95 percent of CD patients, while most of the remaining patients have HLA DQ8.[8-10] However, there is only a 30 percent HLA concordance among siblings, suggesting that other genetic factors are also at play.[11] More recent evidence suggests that the presence of auto-antibodies to a connective tissue element surrounding smooth muscle called endomysium is highly specific for CD. The target of this autoantibody is now known to be an enzyme called tissue transglutaminase (tTG). This enzyme may play a prominent role in the pathogenesis of CD by modifying gliadin, resulting in a greater proliferative response of gliadin specific T-cells, which contributes to mucosal inflammation and further B-cell activation.[5,6,12,13]

CD appears to represent a spectrum of clinical features and presentations. Although "classical" CD (i.e., fully developed gluten-induced villous atrophy and classical features of intestinal malabsorption) is most commonly described, it appears that most patients have atypical CD (i.e., fully developed gluten-induced villous atrophy found in the setting of another presentation such as iron deficiency, osteoporosis, short stature, or infertility) or silent CD (i.e., fully developed gluten-induced villous atrophy discovered in an asymptomatic patient by serologic screening or perhaps an endoscopy for another reason). Other authors describe a latent form of CD that is characterized by a previous diagnosis that responded to a gluten-free diet (GFD) and retained a normal mucosal histology upon later introduction of gluten. Latent CD can also represent patients with currently normal intestinal mucosa who will subsequently develop gluten-sensitive enteropathy.[13,14]

The true prevalence of CD is difficult to estimate because of the variable presentation of the disease, particularly since many patients can have little or no symptoms. With this limitation in mind, the prevalence of the disease is highest in Celtic populations where estimates of 1:300 to 1:122 have been described. The prevalence of CD in North America has been estimated to be 1:3000, but a recent American study found the prevalence among the general not-at-risk population to be 1:105, while the prevalence in at-risk groups such as first-degree relatives of CD patients was 1:22, suggesting that CD is greatly under diagnosed. CD can affect persons of many ethnic backgrounds, but appears to rarely affect persons of purely Chinese, Japanese, or Afro-Caribbean decent.[13]

The diagnosis of CD in adults is classically made on the basis of clinical suspicion—that is, recognizing atypical presentations such as isolated iron deficiency, combined iron and folate deficiency, and osteoporosis—compatible with a duodenal biopsy while taking a gluten-containing diet, followed by clinical and histological improvement following commencement of a GFD.[2,4] However, several serologic markers have become available that have altered the classic

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diagnostic pathway. The sensitivity of IgA anti-gliadin antibodies (AGA) is reported to range from 70 to 85 percent, whereas the specificity ranges from 70 to 90 percent. IgA anti-endomysial (EMA) and anti-tissue transglutaminase (tTG) antibodies have sensitivities in excess of 90 percent and specificities of over 95 percent.[14] Significant variability seems to exist in the reported values among the different studies, and these IgA-based tests can be negative in IgA-deficient patients, accounting for about 3 percent of CD cases.

The sensitivity and specificity of the anti-EMA and anti-tTG antibodies, along with the perceived under diagnosis of CD, has led to suggestions of using these tests for population screening. Aside from the recognized influence of CD prevalence on the predictive value of a serologic test result, little consensus exists regarding the value of population screening. Furthermore, specific questions regarding clinically important outcomes resulting from screening remain unclear. In particular, little data is available on adherence to a GFD in asymptomatic CD patients detected by screening.

The major complications of CD include intestinal and extraintestinal malignancies, ulcerative jejunoileitis, and collagenous sprue. Unlike most gastrointestinal (GI) lymphomas that are typically of B-cell origin, lymphomas associated with CD appear to be most commonly of T-cell origin. Unfortunately, the prognoses for patients with CD-associated T-cell lymphomas, ulcerative jejunoileitis, and collagenous sprue, appear grim. It is widely believed that strict adherence to a GFD reduces the risk of these complications. It is suggested that by 5 years of dietary adherence the risk of lymphoma in CD patients approaches that of the general population.[14]

The challenge of CD remains to determine which patient populations should be screened, the best means of screening, and whether early detection of patients with CD leads to improved patient outcomes. For patient outcomes to improve as a result of screening, the degree to which "positively" screened individuals, particularly those who were asymptomatic, adhere to the stringent GFD, needs to be determined.

Methods

We completed a series of systematic reviews on five areas of CD: (1) sensitivity and specificity of serological tests; (2) prevalence and incidence of CD; (3) CD-associated lymphoma; (4) consequences of testing for CD; and (5) interventions for the promotion and monitoring of adherence to a gluten-free diet (GFD). Staff at the National Library of Medicine performed a series of searches in support of the literature review of CD. Searches were run in the MEDLINE® (1966 to Oct 2003) and EMBASE (1974 to Dec 2003) databases for each of the five objectives and their respective sub-objectives separately. Furthermore, for the 4th and 5th objectives, PsycINFO (1840 forward), AGRICOLA (1970 forward), CAB (1972 forward), and Sociological Abstracts (1963 forward) database searches were run in December 2003. Study selection for each objective was performed using three levels of screening with predetermined increasingly more strict criteria to ensure that all relevant articles were captured. Following a calibration exercise, two reviewers independently screened all studies using a Web-based system that allowed automatic identification of review disagreements. These disagreements were resolved by consensus. For each CD objective, a detailed and standardized data abstraction form was developed. For each objective, data abstraction was conducted by one reviewer and verified by another. The extracted data was further verified by one of the principal investigators. Quality assessments were performed using specific instruments for each of the included study types. The data obtained from this review fell into several broad categories, which correspond in large part to the individual study objectives. Data for the sensitivity and specificity of each serological marker was considered separately, and studies were further divided according to the age group of the study population. Attempts were made to identify, explain, and minimize clinical and statistical heterogeneity in the included studies. A Pearson's Chi Square with n-1 degrees of freedom, where n represents the number of included studies in an analysis, was calculated to assess statistical heterogeneity. Pooled estimates were only calculated, if clinically and statistically appropriate. In situations where pooling was not performed, a qualitative systematic review was conducted.

To produce clinically useful pooled statistics, a weighted mean of the overall sensitivity and specificity from the included studies was calculated, along with 95 percent confidence intervals (CIs). The pooled estimates for the sensitivity and specificity were compared with a summary receiver operating characteristic (ROC) curve, calculated for the same group of studies as a second check of the estimates.

Results and Discussion

Perhaps one of the most important findings of this report is the significance of how one chooses to define CD in the era of serological testing, and how this apparently clear-cut task has profound implications on all the results presented in this report. Specifically, can CD be diagnosed solely on the basis of serology? Is some degree of villous atrophy necessary for a diagnosis of CD? These questions have important implications downstream of the diagnosis as well. For example, do CD patients without symptoms or villous atrophy have the same risk of complications as those with villous atrophy? Is serological improvement on a GFD sufficient to reduce CD complications, or Must there be documented histological improvement? What degree of histological improvement is necessary?

Out of 3,982 citations identified by the search strategy for the Celiac 1 objective, 60 studies fulfilled the level 3 inclusion criteria. Overall, the quality of the diagnostic studies assessed in the Celiac 1 objective was quite good, due largely to our stringent inclusion criteria. However, 59 percent of the

included studies reported using a selected patient population that may not be representative of a clinically relevant population. This is likely related to study design. In addition, only 11 percent of the studies reported on whether the reference test was reported without knowledge of the index test. However, we felt that this was not a major threat to the validity of the studies.

Two other factors that affect the interpretation of these results, are (1) the threshold effects for determining the positivity of a serological test and (2) the high prevalence of CD in these studies (see above). With these considerations in mind, the overall strength of the evidence is quite good.

To minimize clinical and statistical heterogeneity, the included articles of a particular antibody test were divided into groups by age of the included population (adults, children, mixed), the study design (case control, or relevant clinical population/cohort), by antibody type (IgA or IgG), and by test methodology (e.g., monkey esophagus [ME] or human umbilical cord [HUC]). Within these groups, further differences in study population, country of origin, and biopsy definitions (especially whether or not mild grades without villous atrophy were included) were assessed systematically. Studies that reported using the ESPGAN criteria for the diagnosis of CD were categorized as including patients with some degree of villous atrophy. Other potential causes of heterogeneity, such as the cut-offs used to define a positive test, were assessed. The results of the Celiac 1 objective suggest that in the era of EMA and tTG antibody testing, AGA antibody testing in both children and adults has a limited role. The sensitivity and specificity of EMA and tTG are quite high (over 95 percent for sensitivity, and close to 100 percent for specificity), as are their positive and negative predictive values; however, the reported diagnostic parameters are taken from studies in which the prevalence of CD was, for the most part, much higher than that seen in usual clinical practice. The positive predictive values reported for these tests will certainly not be as high as that reported when these tests are used to screen the general population. The bulk of the evidence on the diagnostic characteristics of these tests was derived from studies that defined CD as having at least some degree of villous atrophy.

HLA DQ2/DQ8 testing appears to be a useful adjunct in the diagnosis of CD. The test has high sensitivity (in excess of 90 to 95 percent); however, since approximately 30 percent of the general population, and an even higher proportion of "high-risk" subjects (e.g., diabetics and family members) also carry these markers, the specificity of this test is not ideal. The greatest diagnostic utility of this test appears to be its negative predictive value.

Biopsy itself, when used with a strict cut-off requiring villous atrophy, appears to have high specificity, but poor sensitivity. Using a lower grade cut-off clearly improves sensitivity, but because of the wide differential of causes of histological lesions similar to Marsh I to IIIa, the specificity suffers. The use of histomorphometric measures such as quantification of gamma delta positive intraepithelial lymphocytes (gd+ IELs) are likely to allow for the use of lower grade cut-offs, while maintaining reasonable specificity. Ultimately, a trial utilizing multiple diagnostic tests in an attempt to capture as many CD patients in a clinically relevant population as possible, along with a time dimension such as a response to a GFD or gluten challenge, is required to fully assess the diagnostic characteristics of biopsy alone. This type of study would be able to characterize the false-positive and false-negative rates, provided that all studied patients are followed forward in time.

The literature search yielded 2,116 references to address the Celiac 2 objective. Studies were included if they reported the prevalence and/or incidence of CD in the following groups: (1) general populations from North America or Western Europe; (2) first-degree relatives of patients with CD; (3) patients with type 1 diabetes; (4) patients being investigated for anemia; (5) patients with osteoporosis or osteopenia; and (6) patients with suspected CD on the basis of their clinical presentations. We did not use any geographic restriction for the studies of populations at risk (first-degree relatives and type 1 diabetics) or of associated clinical presentations (suspected CD, anemia, or metabolic bone disease). Studies of prevalence or incidence that used AGA tests conducted prior to 1990 were excluded after discussion with AHRQ because of potential problems with the reliability of older AGA assays. One hundred and nineteen studies were included.

The overall quality of reports of the included studies in the Celiac 2 objective was found to be marginal to fair. For example, most of the studies did not report on whether the patients were consecutively enrolled, a factor that could contribute to selection bias. However, setting aside the quality of individual studies, from a policy perspective, the strength of the evidence is fairly good in that the study populations were selected to reflect that of a North American/Western European descent, that should reflect the demographics of the U.S. population.

The crude incidence of CD in adults varied from lows of 1.27 in Denmark[15] and 3.08 in England,[16] to a high of 17.2 cases per 100,000 patient years in Finland,[17] where specific efforts had been untaken to encourage screening for CD (see Table 34). The crude incidence of CD in children age 0 to 15 years varied from 2.15 to 51 cases per 100,000 patient years.[18-20,21,16,22] When reported, the relative risk (RR) of CD was greatest for the 0- to 2-year age group, as well as for women, and varied from 32.26 to 42.4 [18,19,22] and from 1.9 to 3.34, [23,18,20] respectively. The cumulative incidence at age 5, when reported, varied between 0.089 and 9 cases per 1,000 live births.[23,24,25,26]

The included prevalence studies demonstrated important differences between the studies including execution, tests for prevalence assessment, and patient sampling. Thus, results have to be interpreted in light of some of the limitations that have been identified regarding the diagnostic performance of the tests for CD. Nonetheless, the results of this report suggest that

CD is a very common disorder with a prevalence in the general population that is likely close to 1:100 (1 percent). Several high-risk groups with a prevalence of CD greater than that of the general population have been identified and include: (1) those suspected of having CD; (2) family members of CD patients; (3) type I diabetics; and (4) those with iron-defiency anemia (IDA) or low bone mineral density (BMD). Additionally, the review identified many other high-risk groups, including those with Down Syndrome, short stature, and infertility, to name a few. Their inclusion was, however, beyond the scope of this report.

Out of 379 references resulting from the literature search on CD and lymphoma, our third objective, eight cohort studies and one case-control study were selected for data extraction. The studies included in the Celiac 3 objective were found, overall, to be of good quality. Again, the overall strength of the evidence is due largely to the stringent inclusion criteria, such as the requirement for the reporting of standardized rates for the outcomes based on rates from the local general population, and the overall good quality of the included studies.

Out of 1,199 citations that were identified by the search strategy for the Celiac 4 objective, 35 articles satisfied the screening criteria. The majority of studies included in this objective were single group "before–after" studies, although some also had a comparative healthy control group. We could not identify any quality instruments for this type of study design and, in general, this type of study is considered weak, particularly in the absence of a control group. Overall, however, the strength of the evidence for this objective is fair to good and suggests that the results can be used for policy decisions with the understanding that this area of CD research is still relatively new and requires further high-quality studies.

The results of this report confirm that, apart from a few limitations, there is a strong association between CD and GI lymphoma. The report identified standard incidence ratios (SIR) for lymphoma that ranged from 4 to 40, and standard mortality ratios (SMR) that ranged from 11 to 70. A diagnostic delay—and possibly a diagnosis of CD in adulthood as opposed to in childhood—may be associated with poorer outcomes. Fortunately, several studies suggest that adherence to a GFD reduces the risk of lymphoma in CD patients.

The consequences of testing for CD in at-risk and symptomatic patients appears to be more straightforward, since these patients appear to be more compliant with a GFD and would be expected to benefit from this intervention. The data are less clear for asymptomatic screen-identified patients, particularly those who have truly silent CD and/or don't have fully developed villous atrophy. On the one hand, the outcome of such patients has not been extensively studied; on the other hand, compliance with a GFD appears problematic, particularly for those diagnosed in adulthood.

Out of 502 citations identified by the search strategy for the Celiac 5 objective, 20 studies met level 3 inclusion criteria. The majority of studies in this objective were also of a "before–after" design. However, in this setting, this design may not pose a major limitation, since the purpose of the study is to assess the change in serology and histology after introduction of a GFD. In this regard, the strength of the evidence for monitoring adherence to a GFD is fairly good. However, there is almost a complete absence of studies of interventions for the promotion of adherence to a GFD.

No specific interventions have been identified that promote adherence to a GFD, but education of patients and family members about CD and about the intricacies of a GFD, and participation in local celiac societies, has been shown to improve compliance. Although somewhat controversial, biopsy monitoring of adherence to a GFD appears to be important, since improvement in histological grade has been associated with improved BMD, IDA, and nutritional status. The serological markers appear to be adequate for detecting gross dietary indiscretion and respond to a gluten challenge, but appear to have poor sensitivity for detecting lesser degrees of dietary indiscretion and inadequately correlate with histological improvement, at least in the short-term. Children, on the other hand, show more rapid and complete histological improvement on a GFD. Therefore, monitoring adherence using serology is reasonable in this age group. It should, however, be noted, that we could not identify a controlled study that objectively determined the level of histological improvement that would be associated with improved outcomes; this is an area for future study. Nonetheless, based on this report it would appear that followup biopsy at least 1 year after a GFD in adults to document improvement of the histological grade would be valuable.

This review has allowed us to identify several areas in need of future research. Perhaps the most important of these is a need for the development of a consensus on the definition of CD in the era of advanced serological testing. As discussed in the report, this distinction of what one calls CD has profound implications for each of the requested task order objectives. Do screen-positive patients without villous atrophy have CD? Certainly, the preliminary evidence suggests that this is the situation in many cases. However, what is required is a new definition of a gold standard for the diagnosis of CD. This new gold standard may include a combination of serology, biopsy, and HLA testing. Such a gold standard, when used in studies with a time dimension (e.g., response to a GFD or gluten challenge; extended followup), would help answer some of the uncertainties identified in this report including: the real performance of the serological tests when low-grade lesions are considered CD; the diagnostic performance of biopsy alone; the outcomes of patients with these low-grade lesions; and those that would be "missed" using current screening strategies. Even in the absence of a new gold standard, we could not identify a well-conducted study of the diagnostic performance of the various serological markers when applied to an average population (i.e., one with a prevalence of CD in keeping with the range identified for average risk), with the entire cohort

being investigated equally (i.e., all are biopsied). Such a study would at least be able to shed light on the performance of these tests in average-risk patients, and since all patients are biopsied, the relationship of histology to serology could be further assessed.

On a similar theme, we have identified multiple studies that suggest the importance of histological improvement on a GFD. This is a controversial area because in common clinical practice clinicians are moving away from routine followup biopsy. It seems reasonable to believe that improvement in clinical parameters with loss of serological markers is adequate evidence of response to a GFD. In children, this issue may be less important since histological improvement is much more rapid and complete than in adults, and correlation with serology seems better. However, we have identified multiple studies in adults that suggest poor correlation between serology and improvement of histology on a GFD, and other studies that suggest that serology is useful for detecting gross dietary indiscretion, but not minor occurrences. Therefore, the questions that arise are What constitutes adequate improvement on a GFD?, and What are the criteria to define this improvement? Based on the lymphoma literature that suggests that this malignancy may arise from chronic antigenic stimulation and immune activation, what are the outcomes of adults with clinical improvement, yet persistent histological abnormalities? Are some histological features, such as reduction of mucosal lymphocytes, more important markers of improvement and possibly prognosis than other features such as villous height?

Availability of the Full Report

The full evidence report from which this summary was taken was prepared for the Agency for Healthcare Research and Quality (AHRQ) by the University of Ottawa Evidence-based Practice Center, under Contract No. 290-02-0021. It is expected to be available in July 2004. At that time, printed copies may be obtained free of charge from the AHRQ Publications Clearinghouse by calling 800-358-9295. Requesters should ask for Evidence Report/Technology Assessment No. 104, *Celiac Disease*. In addition, Internet users will be able to access the report and this summary online through AHRQ's Web site at www.ahrq.gov.

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Evidence Report

Chapter 1. Introduction

Overview

Celiac disease (CD) is a disorder of small bowel malabsorption. It is characterized by mucosal inflammation, villous atrophy and crypt hyperplasia, which occur upon exposure to gluten, and clinical and histological improvement with withdrawal of gluten from the diet.[1-4] CD—also referred to as celiac sprue, gluten-sensitive enteropathy, non-tropical sprue, in addition to a host of other names—is thought to result from the activation of both a cell-mediated (T-cell) and humoral (B-cell) immune response upon exposure to the glutens (prolamins and glutenins) of wheat, barley, rye, and oats, in a genetically susceptible person.[5,6] Genetic susceptibility is suggested by a high concordance among monozygotic twins of close to 70 percent,[7] and an association with certain type II human leukocyte antigens (HLA).[8,9] HLA DQ2 is found in up to 95 percent of CD patients, while most of the remaining patients have HLA DQ8.[8-10] However, there is only a 30 percent HLA concordance among siblings, suggesting that other genetic factors are also at play.[11] More recent evidence suggests that the presence of auto-antibodies to a connective tissue element surrounding smooth muscle called endomysium is highly specific for CD. The target of this autoantibody is now known to be an enzyme called tissue transglutaminase (tTG). This enzyme may play a prominent role in the pathogenesis of CD by modifying gliadin, resulting in a greater proliferative response of gliadin specific T-cells, which contributes to mucosal inflammation and further B-cell activation.[5,6,12,13]

CD appears to represent a spectrum of clinical features and presentations. Although "classical" CD (i.e., fully developed gluten-induced villous atrophy and classical features of intestinal malabsorption) is most commonly described, it appears that most patients have atypical CD (i.e., fully developed gluten-induced villous atrophy found in the setting of another presentation such as iron deficiency, osteoporosis, short stature, or infertility) or silent CD (i.e., fully developed gluten-induced villous atrophy discovered in an asymptomatic patient by serologic screening or perhaps an endoscopy for another reason). Other authors describe a latent form of CD that is characterized by a previous diagnosis that responded to a gluten-free diet (GFD) and retained a normal mucosal histology upon later introduction of gluten. Latent CD can also represent patients with currently normal intestinal mucosa who will subsequently develop gluten-sensitive enteropathy.[13,14]

The true prevalence of CD is difficult to estimate because of the variable presentation of the disease, particularly since many patients can have little or no symptoms. With this limitation in mind, the prevalence of the disease is highest in Celtic populations where estimates of 1:300 to 1:122 have been described. The prevalence of CD in North America has been estimated to be 1:3000, but a recent American study found the prevalence among the general not-at-risk population to be 1:105, while the prevalence in at-risk groups such as first-degree relatives of CD patients was 1:22, suggesting that CD is greatly under-diagnosed. CD can affect persons of many ethnic backgrounds, but appears to rarely affect persons of purely Chinese, Japanese, or Afro-Caribbean decent.[13]

Note: Appendixes and Evidence Tables are provided electronically at http://www.ahrq.gov/clinic/tp/celiactp.htm

The diagnosis of CD in adults is classically made on the basis of clinical suspicion—that is, recognizing atypical presentations such as isolated iron deficiency, combined iron and folate deficiency, and osteoporosis—compatible with a duodenal biopsy while taking a gluten-containing diet, followed by clinical and histological improvement following commencement of a GFD.[2,4] However, several serologic markers have become available which have altered the classic diagnostic pathway. The sensitivity of IgA anti-gliadin antibodies (AGA) is reported to range from 70 to 85 percent, whereas the specificity ranges from 70 to 90 percent. IgA anti-endomysial (EMA) and anti-tissue transglutaminase (tTG) antibodies have sensitivities in excess of 90 percent and specificities of over 95 percent.[14] Significant variability seems to exist in the reported values among the different studies, and these IgA-based tests can be negative in IgA-deficient patients, accounting for about 3 percent of CD cases.

The sensitivity and specificity of the anti-EMA and anti-tTG antibodies, along with the perceived under diagnosis of CD, has led to suggestions of using these tests for population screening. Aside from the recognized influence of CD prevalence on the predictive value of a serologic test result, little consensus exists regarding the value of population screening. Furthermore, specific questions regarding clinically important outcomes resulting from screening remain unclear. In particular, little data is available on adherence to a GFD in asymptomatic CD patients detected by screening.

The major complications of CD include intestinal and extraintestinal malignancies, ulcerative jejunoileitis, and collagenous sprue. Unlike most gastrointestinal (GI) lymphomas that are typically of B-cell origin, lymphomas associated with CD appear to be most commonly of T-cell origin. Unfortunately, the prognoses for patients with CD-associated T-cell lymphomas, ulcerative jejunoileitis and collagenous sprue, appear grim. It is widely believed that strict adherence to a GFD reduces the risk of these complications. It is suggested that by 5 years of dietary adherence the risk of lymphoma in CD patients approaches that of the general population.[14]

The challenge of CD remains to determine which patient populations should be screened, the best means of screening, and whether early detection of patients with CD leads to improved patient outcomes. For patient outcomes to improve as a result of screening, the degree to which "positively" screened individuals, particularly those who were asymptomatic, adhere to the stringent GFD, needs to be determined.

Definition of CD

As briefly described in the Overview, CD can take on a variety of forms. Paramount to the conduct of this review and subsequent interpretation of the literature is the identification of clear definitions of the many faces of CD. Implicit to a definition of CD (with a few exceptions that are detailed below) is the concept that the clinical and the small intestinal pathological features are present in patients who consume a gluten-containing diet, normalize with the introduction of a GFD, and recur with the re-introduction of dietary gluten.[2,4] The historical tendency to rely on biopsy features as part of the definition of CD, creates difficulties (as discussed below) in accurately addressing the sensitivity and specificity of biopsy for the diagnosis of CD, and in assessing the sensitivity and specificity of the serologic

markers, if different studies use different criteria to define CD. For the purpose of this review, the following definitions have been used.

General Definitions

1) **Classical CD**. The most commonly described form. It describes patients with the classical features of intestinal malabsorption who have fully developed gluten-induced villous atrophy and the other classic histological features. These patients present because of GI symptoms, and are identified as CD sufferers through the investigation of these symptoms. This group can also be said to have symptomatic CD.

2) **Atypical CD**. Appears to be one of the most common forms. These patients generally have little to no GI symptoms, but seek medical attention because of another reason such as iron deficiency, osteoporosis, short stature, or infertility. These patients generally have fully developed gluten-induced villous atrophy. Because these patients are "asymptomatic" from the GI perspective, if their atypical CD feature is not recognized, they may be difficult or impossible to distinguish from "true" silent (asymptomatic) CD patients.

3) **Silent CD**. A very common form of CD. Refers to patients who are asymptomatic but are discovered to have fully developed gluten-induced villous atrophy after having undergone serologic screening or perhaps an endoscopy and biopsy for another reason. These patients are clinically silent, in that they do not manifest any clear GI symptoms or associated atypical features of CD such as iron deficiency or osteoporosis. These patients can be confused with atypical CD if their atypical features are not recognized in an early stage. As well, Fasano et al.[15] have shown that many of these patients do not manifest fully developed villous atrophy.

4) **Latent CD**. Represents patients with a previous diagnosis of CD that responded to a GFD and who retain a normal mucosal histology upon later re-introduction of gluten. Latent CD can also represent patients with currently normal intestinal mucosa who will subsequently develop gluten-sensitive enteropathy.

5) **Refractory CD.** For the purpose of this review, patients with refractory CD are patients with true CD and villous atrophy (i.e., not a misdiagnosis) who do not, or no longer, respond to a GFD. Although the most common reason for failure to respond to a GFD is dietary indiscretion or unknown exposure to gluten, refractory CD also occurs in patients on a GFD who have developed a complication such as ulcerative-jejunoileitis, or enteropathy-associated lymphoma. Patients with refractory CD do not necessarily have positive serology for CD. Refractory CD was reviewed in the context of the requested objectives.

In order to utilize the above definitions, there needs to be clear and valid histological criteria for the diagnosis of CD. The histological patterns, particularly the more mild lesions, are not specific for CD and can be seen in a variety of other disorders (Table 1, Appendix A).

To help standardize the histological criteria for the diagnosis of CD, several scoring systems have been developed. The classic Marsh criteria,[1] and its modification by Rostami,[16] are presented in Table 2 (Appendix A). The revised ESPGAN criteria[4] use histological, serological and clinical criteria (Table 3, Appendix A).

Report Purpose and Target Population

The purpose of this report is to systematically review the available CD literature in order to provide organized evidence relating to a number of objectives put forth by the AHRQ. The findings of the report are intended to assist an assembled group of American and world experts in the field of CD in the development of a National Institute of Health (NIH) Consensus Development Conference Guidelines sponsored by AHRQ and OMAR.

Methodological Considerations

At first glance, the determination of the sensitivity and specificity of the various diagnostic modalities for CD seems straightforward. There are a multitude of studies that have assessed the diagnostic characteristics of each of the serological markers using a variety of different laboratory methods. However, these studies are remarkably heterogeneous on a number of levels.

For example, there appears to be notable heterogeneity in the actual definition of CD, an issue that has important consequences on all of the task order objectives. Central to the classic definition of CD is the recognition that biopsy is the gold standard for diagnosis. However, it has become clear over the years that the majority of patients with CD do not have the classically described features of intestinal malabsorption, and that a large proportion of patients do not have the classic flat mucosa (sub-total or total villous atrophy). To further aid in the diagnosis of CD, multiple authors have devised and modified histological criteria to grade the mucosal lesions of patients with CD. But still at issue is the broad differential of disorders that can cause villous atrophy, particularly the milder histological grades. To help address this issue, others have attempted to address specific features of the biopsy, such as the number of intraepithelial lymphocytes (IELs), the number of gamma delta positive ($\gamma\delta+$) IELs and other lymphocyte subtypes, as well as the localization of IELs towards the villous tip, just to name a few.

The serological screening studies, together with the recognition that a low-grade histological lesion can be consistent with CD, have helped bring to light the concept of a spectrum of CD and the so-called "celiac iceberg." In brief, it is recognized that classic CD with the typical symptoms of malabsorption and a fully developed mucosal lesion represents a small proportion of patients. The majority of patients are asymptomatic and are classified as having either atypical CD, silent CD, or less commonly latent CD. Some authors question whether most, if not all cases of silent CD, are in fact atypical CD, although the associated consequence of this has not been recognized. To further complicate the issue, Fasano[15] has clearly characterized patients with silent CD without fully developed mucosal lesions, and found that only 34 percent of the patients had subtotal or total villous atrophy.

It should be recognized that the majority of studies assessing the diagnostic characteristics of the serological markers have defined CD by a biopsy with Marsh III or modified IIIa

lesions or greater. These studies have reported a high sensitivity and specificity for these tests, particularly for the anti-EMA and anti-tTG antibody tests. However, some studies have looked at the characteristics of these tests in lower-grade lesions, and have found that while 100 percent of patients with Marsh IIIc histology show antibodies to endomysium, only 60 percent of patients with Marsh IIIa histology have anti-EMA antibodies.[17,18] Furthermore, it is apparent that serological markers can be used to monitor adherence to a GFD; for example, EMA and tTG antibodies fall to normal or non-diagnostic levels on a GFD, but the correlation with improvement of villous height is not as clear-cut. Finally, with the discovery by Sollid et al.[8] and others, that over 95 percent of patients with CD have HLA DQ2 and most of the remainder having HLA DQ8, it became hopeful that a reliable confirmatory test based on HLA typing would be available. Unfortunately, up to 40 percent of the general population and a much higher proportion of those with autoimmune disorders such as type I diabetes also have HLA DQ2 and/or HLA DQ8. Therefore, the specificity of this test can be quite low, making its positive predictive value relatively low. It is also becoming apparent that HLA DQ2/8 may not be the true risk-genes, and researchers are actively studying other candidate genes that may be associated with DQ2/8, or in patients without DQ2/8, other genes altogether.

The preceding overview was presented to simply illustrate the complexity involved in separately assessing the sensitivity and specificity of the serological markers, HLA typing, and biopsy itself, in the diagnosis of CD. Over time, the status of the biopsy as the gold standard for the diagnosis of CD has been eroded. Yet at the same time, most of what we know about the sensitivity and specificity of serological markers and HLA typing rely on biopsy as the gold standard. Therefore, one is locked in a circular argument of how best to choose the gold standard test(s), when each has important shortcomings and is dependent on another to define its own diagnostic characteristics. The major problem in accurately evaluating the diagnostic characteristics of these tests, is the issue of identifying all possible CD patients in a general screened population to use as a benchmark. Serology would be the most convenient strategy, but appears to loose sensitivity in patients with low-grade lesions. Screening a general population with biopsy has significant practical/cost issues, as well as potential ethical problems; however, if such a study was performed along with measuring the serological and HLA status of patients, this would allow for identification of Marsh I or II lesions that would need to be characterized further. HLA DQ2/8-negative patients could likely be excluded from having CD. But those patients with Marsh I-II lesions would have to be followed, whether or not they were serology positive or HLA DQ2/8 positive, to see if CD develops; alternatively, they could be tested with a GFD and subsequently rechallenged to see whether they truly have CD. Only in this way can the true sensitivity of biopsy be determined. Using this multi-test gold standard with follow-up of equivocal cases, would also be the best way of assessing the sensitivity and specificity of serology markers and HLA DQ2/DQ8 typing.

Finally, a question which needs to be addressed is: “What are the implications of identifying a truly asymptomatic individual, for example with serological screening, who has no other obvious complications such as iron deficiency or osteoporosis, and is then found to have a Marsh I or II lesion?” This returns the circular argument back to “What is truly CD?”—a question that is beyond the scope of this review.

Chapter 2. Methods

Overview

The UO-EPC's evidence report on CD is based on a systematic review of the scientific-medical literature to identify, and synthesize the results from studies addressing the key questions put forth by the AHRQ. The Celiac Review Team, together with content experts, identified specific issues integral to the review. A Technical Expert Panel (TEP) refined the research questions, as well as highlighted key variables requiring consideration in the evidence synthesis. Evidence tables presenting the key study characteristics and results were developed. Summary tables were derived from the evidence tables. The methodological quality of reports of the included studies was appraised, and individual study results were summarized. For some objectives a narrative interpretation of the literature was provided.

Key Questions Addressed in This Report

The AHRQ task order requested answers to the questions outlined below:

1) Objective 1 – Sensitivity and specificity of tests for CD (Celiac 1)

a) What is the sensitivity and specificity of the following tests for CD:
   i) AGA;
   ii) EMA;
   iii) human tTG lgA antibodies;
   iv) HLA (DQ2/DQ8);
   v) duodenal/jejunal biopsy (see section below on celiac definition)
b) Do sensitivity and specificity vary in different target populations (e.g., symptomatic vs. asymptomatic; geographic populations)?

2) Objective 2 – Prevalence and incidence of CD (Celiac 2)

a) What is the prevalence and incidence of symptomatic and "clinically silent" CD in:
   i) the general population;
   ii) high-risk populations:
      (1) family member of patient with CD;
      (2) type 1 diabetes mellitus;
      (3) iron deficiency anemia (IDA);
      (4) osteoporosis?
b) How does prevalence and incidence in the general population vary in different geographic and racial/ethnic populations?

3) Objective 3 – Celiac associated lymphoma (Celiac 3)

Note: Appendixes and Evidence Tables are provided electronically at http://www.ahrq.gov/clinic/tp/celiactp.htm

a) What is the association between CD and GI lymphoma?
   i) What is the cumulative risk of developing GI lymphoma in patients with CD?
   ii) Does the cumulative risk vary with clinical presentation?

4) Objective 4 – Expected consequences of testing for CD (Celiac 4)

a) What are the expected consequences of testing for CD in the following populations:
   i) patients with symptoms suggestive of CD;
   ii) asymptomatic, at-risk populations (affected family members, patients with type 1 diabetes);
   iii) the general population?

b) "Consequences" include:
   i) false-positive results;
   ii) follow-up testing;
   iii) invasive procedures (biopsies);
   iv) cases diagnosed;
   v) patients complying with treatment; and
   vi) response to treatment.

5) Objective 5 – Promoting or monitoring adherence to a GFD (Celiac 5)

a) What interventions are effective for promoting or monitoring adherence to a GFD?

Study Criteria Used in this Review

Histological

From the preceding discussion in the methodological consideration section it is clear that current histological criteria using a cut-off grade to define CD have important shortcomings. We therefore adopted an open histological definition of CD when selecting a study for inclusion, as long as the authors' explicitly stated or described the criteria used to define CD (see inclusion criteria below)**.** ***However, with the help of the TEP, we defined a "standard" histological definition of CD as a biopsy grade showing a modified Marsh IIIa or greater***. This definition was NOT used as an inclusion/exclusion criterion, but simply to frame our results and to allow for the evaluation of the effect of different histological criteria on the performance of the various CD tests.

The choice of biopsy criteria and/or histological grade "cut-off" used to define CD has important implications for the interpretation of the studies of serology, HLA, and biopsy. It is recognized that some patients with CD may have Marsh I or II lesions, and by definition patients with latent CD have Marsh 0 lesions. However, as emphasized by Marsh,[1] and as is discussed further below, in order to correctly interpret these early lesions, prospective follow-up studies are required, and an individual patient follow-up and documented response to gluten withdrawal would be required to firmly establish the diagnosis of CD.

The practical importance of the histological definition is evident from our preliminary review of articles that demonstrated considerable heterogeneity in the histological criteria used within the studies to define CD. Some used strict definitions, whereas, others accepted milder grade lesions. Furthermore, since the existence of latent CD and some silent CD without fully developed histology is now recognized, a study that aims to assess the sensitivity and specificity of biopsy itself in CD needs to use a design that incorporates the most sensitive and specific serologic and HLA tests available. The biopsy and serology should be performed simultaneously, with patients having discordant test results being further evaluated. Those with normal biopsy and positive serology would have to be followed over time to see if they have a latent form of CD. Conversely, patients with positive biopsies and normal serology would have to demonstrate improvement in histology on a GFD, and ideally, certification of relapse by biopsy with reintroduction of gluten. This type of study design was sought in order to address the objective of the sensitivity and specificity of biopsy.

Populations

1) **Unselected general population**. The unselected general population implies a representative sample of a given population, such as a random sample of healthy blood donors or healthy school children. Some unselected populations are better than others for determining the true prevalence or incidence of CD. For example, blood donors are required to have normal hemoglobin and no iron deficiency, and therefore may underestimate the true numbers of patients with CD.

2) **Suspected CD.** Patients with suspected CD include patients with GI symptoms, such as diarrhea or symptomatic malabsorption, who are being investigated for the possibility of CD. These patients are typically undergoing other investigations in addition to being worked-up for CD.

3) **High-risk populations.** High-risk populations include populations with an expectedly higher prevalence of CD. Such populations include asymptomatic family members of patients with CD, patients with type I diabetes where identified CD would likely be silent or latent, and populations such as those with iron deficiency or osteoporosis where identified CD would be in the atypical CD classification.

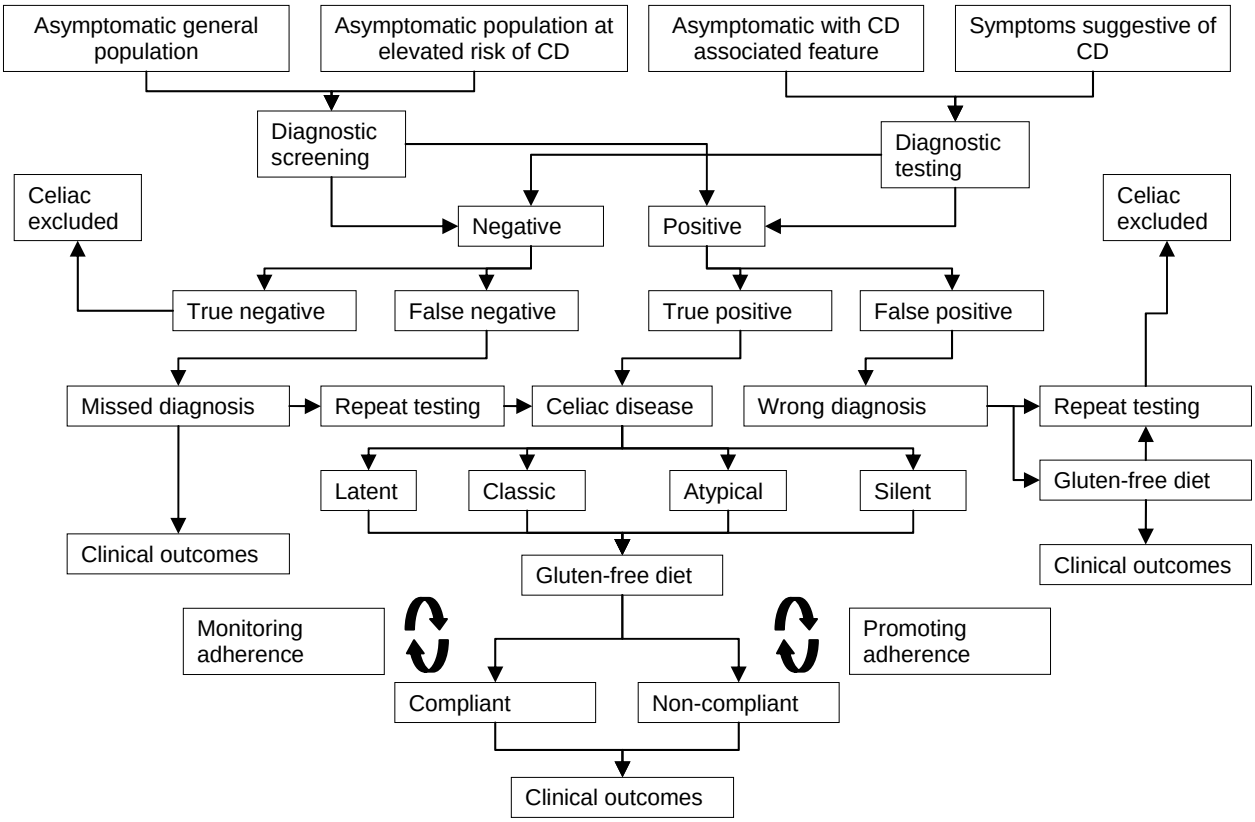
HLA DQ2/DQ8

The HLA DQ2 haplotype represents the occurrence of HLA class II heterodimer alleles DQA1*0501 and DQB1*0201. These typically occur in a cis position as HLA DR3-DQ2 or in a trans position as HLA DR5/DR7-DQ2. The HLA DQ8 haplotype DQA1*0301/DQB1*302 typically occurs in association with DR4.

Analytical Framework

The analytical framework is presented in Figure 1. In this framework, we wanted to represent the diagnostic pathways and the potential outcomes of testing various populations for CD. Each step of the pathway represents a portion of this systematic review, starting with the identification of the populations of interest, their diagnostic pathways, and ultimately the clinical outcomes, as well as consequences of testing.

Figure 1: Analytic framework



Study Identification

Although the objectives of this task order are contained within a request for a single evidence report, we conducted five separate reviews, from the literature search onwards, as the objectives of this mandate were more orthogonal than overlapping.

Search Strategy

A series of searches were performed by National Library of Medicine staff in support of the literature review for CD. Strategies were developed using the guidelines supplied by the UO-EPC, and were divided into the five questions posed by AHRQ. All searches were limited to human studies published in English language journal articles. The specific strategies used for each search are located in Appendix B.

1. What is the sensitivity and specificity of the following tests for CD:
    a. EMA
    b. human tTG IgA antibodies
    c. AGA
    d. HLA DQ2/DQ8
    e. small bowel biopsy

Searches were run in the MEDLINE® and EMBASE databases for each of the five tests. With the exception of the search for small bowel biopsy, a reference to CD or its synonyms was not a requirement for retrieval in order to obtain the widest possible information on these tests. Because of their complexity, a separate search was run for each test, then the results combined into one Pro-Cite file and duplicates eliminated. Individual case reports and letters to the editor were also removed.

The MEDLINE® searches were run in October 2003 for the year 1966 forward and yielded a total of 2885 citations, with a follow-up search for HLA DQ2 and DQ8 performed in November 2003 that yielded an additional 390 citations. The EMBASE searches were run in December 2003 for the year 1974 forward and yielded a total of 1,046 citations after duplicates to MEDLINE® were removed.

2. What is the prevalence and incidence of symptomatic and clinically silent CD in the general population and in the following identified high-risk populations:
    a. patients with an affected family member
    b. type 1 diabetes mellitus
    c. IDA
    d. osteoporosis

Searches were run in the MEDLINE® and EMBASE databases. The MEDLINE® search was performed in October 2003 for the year 1966 forward and retrieved a total of 1,584 citations. The EMBASE search was run in December 2003 for the year 1974 forward and yielded 467 citations after duplicates to the MEDLINE® retrieval were removed. Individual case reports and letters to the editor were also removed from both searches.

3. What is the association between CD and GI lymphoma?

Searches were run in the MEDLINE® and EMBASE databases. The MEDLINE® search was performed in October 2003 for the year 1966 forward and retrieved a total of 230 citations. The EMBASE search was run in December 2003 for the year 1974 forward and yielded 97 citations after duplicates to the MEDLINE® retrieval were removed. Individual case reports and letters to the editor were also removed from both searches.

4. What are the expected consequences of testing for CD in the following populations:
    a. patients with symptoms suggestive of CD
    b. asymptomatic, at-risk populations
    c. general population

Searches were run in the MEDLINE®, EMBASE, PsycINFO, AGRICOLA, CAB, and Sociological Abstracts databases. In order to obtain the widest possible retrieval, all articles on screening for celiac and its synonyms were included, not just those discussing consequences.

The MEDLINE® search was performed in October 2003 for the year 1966 forward and retrieved a total of 917 citations. The EMBASE (1974 forward), PsycINFO (1840 forward), AGRICOLA (1970 forward), CAB (1972 forward), and Sociological Abstracts (1963 forward) database searches were run in December 2003 and yielded a combined total of 204 citations after duplicates to the MEDLINE® retrieval were removed. Individual case reports and letters to the editor were also removed from both searches.

5. What interventions are effective for promoting or monitoring adherence to a GFD?

Searches were run in the MEDLINE®, EMBASE, PsycINFO, AGRICOLA, CAB, and Sociological Abstracts databases. Because of the small number of citations retrieved, a few selected articles discussing adherence to dietary limitations for other conditions were included. The MEDLINE® search was performed in October 2003 for the year 1966 forward and retrieved a total of 152 citations. The EMBASE (1974 forward), PsycINFO (1840 forward), AGRICOLA (1970 forward), CAB (1972 forward), and Sociological Abstracts (1963 forward) database searches were run in December 2003 and yielded a combined total of 168 citations after duplicates to the MEDLINE® retrieval were removed. Individual case reports and letters to the editor were also removed from both searches.

Some citations fulfilled the criteria of more than one celiac objective. Duplicates within each celiac objective were electronically removed. The obtained citations were uploaded into an internal web-based review system (SRS) for online collaborative citation screening and abstraction. Articles passing the first level screen were retrieved in full for further screening (see below).

Reference lists of included studies, book chapters, and narrative or systematic reviews retrieved after having passed the first level of relevance screening, were manually searched to identify additional unique references. Through contact with content experts, and the TEP, attempts were made to identify other studies not identified by the search.

Study Selection and Eligibility Criteria

Study selection was performed using three levels of screening with increasingly more strict criteria to ensure that all relevant articles were captured (Table 1). Each celiac objective had its own selection criteria for each level of screening and, as discussed previously, each celiac objective was treated as a separate sub-review. Following a calibration exercise, two reviewers

independently screened all studies using the SRS web-based system. This system allows automatic identification of review disagreements. Any disagreements were resolved by the two reviewers by consensus; rarely, a third reviewer was used to break an impasse. The specific screening questions for each screen level are included in Appendix C.

Level 1 broad screening. Level 1 screening was used to identify any potentially relevant citation, based on review of the title, abstract and key words. For each objective, the SRS system displayed the corresponding task order questions alongside the citation details. Reviewers answered a broad question of whether the citation potentially related to the current objective. Furthermore, the SRS system was set-up in such a way that articles which were identified in one celiac objective silo, that could also be relevant to another objective, could be identified and moved/copied to the other silo. The review team was divided up so that two members could be simultaneously reviewing each objective.

Level 2 refined screening. Potentially relevant articles identified at level 1 were obtained in full for level 2 screening. Again, using the SRS system with the actual articles on hand, reviewers selected articles that related to each of the specific objectives. The reviewers were asked to err on the side of inclusion for this level, and to classify articles as "original" or "review". Original articles meeting level 2 inclusion also had basic demographic data—such as screening test used, celiac definition, and study population identified—recorded into the SRS system.

Level 3 final screening. Level 3 screening identified articles that specifically allowed for the answering of the task order questions. These articles fulfilled the final inclusion/exclusion criteria, allowed actual extraction of the required data, and did not have fatal methodological flaws.

Table 1: Inclusion/exclusion criteria by level of screening

Objective	Level	Inclusion	Exclusion
Celiac 1	1	Any article reporting sensitivity/specificity of AGA, EMA, tTG, HLA DQ2/DQ8, or biopsy.	Clearly unrelated citation.
	2	For serology and HLA – articles where sensitivity and specificity could be extracted. For biopsy – articles were included if some measure of diagnostic utility could be obtained.	
	3	Articles that allowed determination of sensitivity or specificity for all tests were included.	• Articles with major methodological flaws excluded • Control group did not have gold standard test (biopsy) applied • No description of biopsy criteria given • Celiac group known to be positive for test under evaluation • Control group known to be negative for the test under evaluation • Control groups included patients with Marsh I or II biopsy lesions • AGA test performed without commercial ELISA kit or before 1990
Celiac 2	1	Any potential citation of prevalence or incidence of CD in general and high-risk populations or association of CD with other disorders	Clearly unrelated citation.
	2	Citations limited to those that gave evidence of the prevalence or incidence of CD in the general population or the AHRQ identified high-risk populations (e.g., diabetes, relatives, iron deficiency, osteoporosis). Countries: North America, western Europe, Australia, New Zealand.	Any studies of other CD-associated disorders not identified by the task order. Citations of the prevalence of specific disorders in patients with celiac (i.e., reverse of the inclusion). Any other country.
	3	Incidence and/or prevalence could be extracted from the article.	Serious methodological flaws: • patients identified by surveys, through solicitation of celiac societies • incidence studies without a population density denominator

Table 1 (cont'd): Inclusion/exclusion criteria by level of screening

Objective	Level	Inclusion	Exclusion
Celiac 3	1	Any potential citation of the association, prevalence or risk of lymphoma in CD, including articles on outcome of refractory sprue and ulcerative jejunoileitis.	Clearly unrelated citation.
	2	Measure of risk or prevalence/incidence of lymphoma in a population with CD.	Prevalence of CD in a population of lymphoma. Case reports and non-comparative case series.
	3	Extractable prevalence, incidence, or cumulative risk of lymphoma in CD.	Clonality of lymphocytes in ulcerative jejunoileitis-ileitis not determined or stated (as per TEP). Serious methodological flaw.
Celiac 4	1	Any potential citation of possible consequences of testing for CD.	Clearly unrelated citation.
	2	Consequences extractable from article.	
	3	Consequences limited to the AHRQ list.	Consequences obtainable from the other celiac objective sub-review – i.e., false positive and negative results, etc.
Celiac 5	1	Any potential citation of interventions for the monitoring or promotion of adherence.	Clearly unrelated citation.
	2	Studies of monitoring adherence were included if they assessed monitoring, by biopsy, serology (AGA publication date 1990 or later, EMA, tTG), or both. Any promotion intervention.	Serology prior to 1990.
	3	Data from article could be extracted. Data included follow-up by biopsy alone or serology with biopsy confirmation.	Articles assessing adherence through the measures of intestinal permeability. Studies that reported changes in mean serological titers with a GFD or gluten challenge, but did not address the potential usefulness of a serologic test to assess compliance.

Important articles answering a stated objective but not meeting inclusion criteria (i.e., containing potential threats to internal validity), were presented and discussed in the discussion section.

Data Abstraction

For each objective, a detailed and standardized data abstraction form was developed with the assistance of content experts and the TEP panel. The data abstraction forms included baseline study characteristics as well as questions allowing for the abstraction of all relevant study results and characteristics. The electronic data extraction forms began with basic study and patient

demographic questions that were common across the five sub-review forms. These included reviewer name, author name, publication year, publication type, study design type, and basic study population demographics such as race, age, gender, and type of CD population. The extraction forms then moved to specific questions geared at extracting data to answer the respective objective's questions. The individual data abstraction forms are included in Appendix C.

Celiac 1 (sensitivity and specificity) data abstraction form. Separate data abstraction forms were developed for serology, HLA, and the biopsy sub-questions. Two-by-two tables were used to abstract data on sensitivity and specificity, and to determine positive and negative predictive values and the prevalence of CD in the tested population. The biopsy studies were quite heterogeneous, and did not allow for direct numeric extraction of data.

Celiac 2 (prevalence and incidence) data abstraction form. For this objective, the data extraction form included questions for detailing the screened study population, the number of individuals screened, the number of CD cases identified and how CD was confirmed. For incidence studies, the comparison population and time period were recorded.

Celiac 3 (lymphoma) data abstraction form. In addition to the basic demographic, and study design data, the extraction form contained fields for the extraction of risk data linking GI lymphoma to CD. Types of data sought were prevalence and incidence of lymphoma in CD in the setting of comparison data from a control population. Fields for extracting standardized incidence, morbidity, and mortality ratios were included.

Celiac 4 (consequences of screening) data abstraction form. The extraction forms for this objective included text fields to detail the consequences of testing for CD. The form contained fields that identified the specific consequence of testing which was addressed by the study, as well as a data field to report the study findings. The general field approach was chosen to allow extraction of the expected varied data for this objective.

Celiac 5 (monitoring and promoting adherence) data abstraction form. For this objective, standard demographic data was collected, as well as the methods used to monitor adherence to a GFD, the response of those measures to the diet, and the correlation of serological methods with biopsy findings. Space was provided to detail the sensitivity and specificity of the monitoring method when that data was available. For the objective of promoting adherence to a GFD, a text-based form was used to allow the extractor to describe the intervention and the results of its use.

Electronic forms. The abstraction forms were developed in Microsoft Excel to allow for electronic data entry and recording, and to allow exporting the evidence table data into Microsoft Word. For each celiac objective, data abstraction was conducted by one reviewer and verified by another. The extracted data was further verified by one of the principal investigators.

Quality Assessment

The quality of reporting of diagnostic test studies was assessed using the QUADAS tool.[19] This tool is the first to be published that allows for the assessment of the quality of studies of diagnostic tests. The instrument was developed using a Delphi procedure. The Delphi panel consisted of nine experts in diagnostic research who refined an initial list of items in four rounds, after which agreement was reached on the items to be included in the tool. The QUADAS tool consists of 14 questions that are answered "yes," "no," or "unsure." The tool addresses the items individually and does not incorporate an overall quality score (Appendix D).

Cohort and case-control study reports were assessed using the Newcastle-Ottawa scale (NOS; Appendix D). The NOS is an ongoing collaboration between the Universities of Newcastle, Australia and Ottawa, Canada. It was developed to assess the quality of non-randomized studies with its design, content and ease-of-use directed to the task of incorporating the quality assessments in the interpretation of meta-analytic results. A "star system" has been developed in which a study is judged on three broad perspectives: the selection of the study groups; the comparability of the groups; and the ascertainment of either the exposure or outcome of interest for case-control or cohort studies, respectively. The goal of this project is to develop an instrument that provides an easy and convenient tool for quality assessment of non-randomized studies for use in a systematic review.

The inter- and intra-rater reliability of the NOS have been established. The face content validity of the NOS has been reviewed based on a critical review of the items by several experts in the field, who evaluated its clarity and completeness for the specific task of assessing the quality of studies to be used in a meta-analysis. Furthermore, the validity of the NOS criteria has been established by comparisons to more comprehensive but cumbersome scales. An assessment plan is being formulated for evaluating its construct validity, with consideration of the theoretical relationship of the NOS to external criteria and the internal structure of the NOS components.[20]

Quality assessments of cross-sectional reports were assessed using a 19-item instrument adapted from Ophthalmology (Appendix D).[21]

We did not conduct any sensitivity analysis of quality assessments on the observational studies, as there is little by way of guidance to suggest what a poor quality study score would be based on for these assessment instruments.

One reviewer assessed the quality of an entire celiac objective to maintain internal consistency. Quality assessment was not performed under masked conditions.

Data Synthesis and Analysis

The data obtained from this review fell into several broad categories, which correspond in large part to the individual study objectives. These will be addressed in turn.

Data for the sensitivity and specificity of each serological marker was considered separately. In addition, studies were subdivided by the population age group (adults, children, mixed population), and by study design (case control, relevant clinical population/cohort).

Attempts were made to identify, explain, and minimize clinical and statistical heterogeneity in the included studies. Heterogeneity was assessed graphically by plotting receiver operator (ROC) curves for each of the included studies in a given analysis. A Pearson's Chi Square with

n-1 degrees of freedom, where n represents the number of included studies in an analysis was calculated to assess statistical heterogeneity.

Pooled estimates were only calculated if clinically and statistically appropriate. In situations where pooling was not performed, a narrative systematic review was conducted.

There are several potential ways to pool the results of studies of diagnostic tests, each having both advantages and disadvantages. The simplest and most intuitive is to simply perform a weighted mean of the sensitivity and specificity for the studies in question. This method provides a pooled estimate that is easy to interpret by clinicians. Several other techniques involve the pooling of diagnostic odds ratios or likelihood ratios. These methods have the distinct disadvantage of difficulty in interpretation, and the inability to derive a pooled sensitivity or specificity from the resulting estimates. Lastly, one can use one of several methods to produce a summary ROC curve. The method described by Littenberg and Moses,[22,23] has the advantage of being able to produce a summary curve while taking into account a threshold effect. This can occur when different studies use different thresholds to define a positive test, or even from differences in labs using the same cut-off. To interpret summary ROC curves it is necessary to know the sensitivity or specificity of the test in question in the population in which it will be applied. Since neither of these values is estimable without conducting yet another diagnostic accuracy study for the given population, the clinical usefulness of using this method alone is limited.[24,25]

In order to produce clinically useful pooled statistics, we calculated a weighted mean of the sensitivity and specificity from those of the included study. For both sensitivity and specificity, this pooling relies on the assumption that the test statistic is the same in all of the included studies. For each pooled estimate, a 95% confidence interval (CI) was calculated using both a fixed and random effects model. The results of which were compared as a further test for heterogeneity. The pooled estimates for the sensitivity and specificity were also compared with a summary ROC curve calculated for the same group of studies as a second check of the estimates (summary ROC Curves are included in Appendix E).

The prevalence and incidence data from the Celiac 2 objective, and the CD-lymphoma data from the Celiac 3 objective, were anticipated to be quite heterogeneous considering the different, countries, age groups, and risk characteristics of the studied patients. Attempts were made to group studies of prevalence by age group, study population, and serological screening method. If the grouped studies did not show evidence of heterogeneity, pooled estimates of the prevalence were produced for that group of studies, otherwise a descriptive presentation of the data with a qualitative systematic review was conducted. Likewise, the outcome measures of the Celiac objectives 4 and 5 were presented in a qualitative systematic review, except in cases where it was possible to pool the sensitivity and specificity data as measures of monitoring of patients at various stages of recovery on a GFD.

Chapter 3. Results

Celiac 1: Sensitivity and Specificity of Tests for CD

Serology

Out of 3,982 citations identified by the search strategy for the Celiac 1 objective, 907 met level 2 screening criteria. Of these, 204 diagnostic test studies of one or more of the serological markers of interest (AGA, EMA, tTG) were identified. Sixty studies fulfilled the level 3 inclusion criteria (Appendix F; Evidence Table 1, Appendix I).[26-85] The most common reasons for failing level 3 inclusions were AGA studies conducted before 1990, studies utilizing an improper or an unbiopsied control group, or studies that did not give any description of the biopsy criteria defining CD. Five pairs of duplicate publications were identified.[27,28,45,46,58,65,73,74,84,86] Out of each duplicate pair, the study with the most complete data was abstracted,[27,45,46,58,74] bringing the total of included unique studies to 55. The majority of these studies assessed more than one serological marker, and some studied more than one age group. Of the included articles, 20 were conducted in or included an adult population, 33 were conducted in a population of children, and eight in a mixed population of adults and children of varying proportions. The statements in this section that relate to mixed studies or studies in children and adults refer to these eight studies, and not to a sample that we pooled from different studies.

To minimize clinical and statistical heterogeneity, the included articles of a particular antibody test were divided into groups by age of the included population (adults, children, mixed), the study design (case control, or relevant clinical population/cohort), by antibody type (IgA or IgG), and by test methodology (e.g., monkey esophagus [ME] or human umbilical cord [HUC]). Within these groups, further differences in study population, country of origin, and biopsy definitions (especially whether or not mild grades without villous atrophy were included) were assessed systematically. Studies that reported using the ESPGAN criteria for the diagnosis of CD were categorized as including patients with some degree of villous atrophy. Other potential causes of heterogeneity such as the cut-offs used to define a positive test were assessed.

Two articles were identified that assessed the diagnostic value of various antibodies in children[64] and in mixed-age populations[40] with IgA deficiency. As well, one study enrolled biopsy-proven CD patients who were known to be EMA negative.[66] These studies were considered separately from the others. Studies of using antibodies in combination were also assessed separately.

Pooled statistical estimates (with 95% CIs) are provided for studies without clinical and statistical heterogeneity, and summary ROC curves for the studied antibodies are provided in Appendix E. Sensitivity analyses by study design did not show a significant difference except for the analysis of IgA-tTG-guinea pig (GP) in adults. Therefore, apart from studies of IgA-tTG-GP in adults, pooled estimates, when available, included data from both study designs.

Note: Appendixes and Evidence Tables are provided electronically at http://www.ahrq.gov/clinic/tp/celiactp.htm

AGA. The diagnostic characteristics of IgA were assessed in 35 studies and the diagnostic characteristics of IgG-AGA were assessed in 30 studies. Of the 35 IgA-AGA studies, 11 were conducted in an adult population,[30,33,45,50,54,61-63,71,77,80] 21 in a population of children,[26,27,29,31,34,36,38,42,43,50,52,56,59,60,64,67,68,83,85,87,88] and five in a mixed population.[27,37,40,74,75]

Of the 30 IgG-AGA studies, seven were conducted in an adult population,[30,33,54,62,63,71,80] 19 were conducted in population of children,[26,27,29,31,34,36,38,42,43,50,52,58,59,64,66,68,69,83,85] and five in a mixed population.[27,37,40,74,75] Some studies provided data for more than one age group.

Some studies only provided summary statistics without the raw two-by-two table results,[33,34,54,58,59,69] however, the raw data was calculated from the presented sensitivity and specificity, and from the group sizes.

One study[66] was conducted in CD patients who were known to be IgA-EMA negative, and was not included in the main analysis. In this study of children, the sensitivity for IgA-AGA was 22% and the sensitivity for IgG-AGA was 33%, whereas, the specificity for IgA-AGA was 67% and the specificity for IgG-AGA was 58%; these values are considerably lower than those reported in other studies. Another two studies were conducted in patients with IgA deficiency.[40,64] The first demonstrated a sensitivity of 0% using IgA-AGA, but a sensitivity and specificity of 100% using IgG-AGA,[40] whereas the second showed a sensitivity of 0% with IgA-AGA, but a sensitivity of 100% and a specificity of 80% using IgG-AGA.

Despite clinical subdivision of the identified studies, significant heterogeneity was identified for each of the pooled AGA subgroup results (Tables 2 to 7). Heterogeneity can be visualized graphically in the ROC curves (Figures 2 to 4) and suggests that the heterogeneity is in part related to a serological test cut-off threshold effect. As well, two studies included CD patients with less than a Marsh IIIa grade;[37,45] these studies had lower than average sensitivities (61% and 67% for IgA-AGA) then that reported in other studies. The remaining heterogeneity likely represents a combination of the effects of different test kits, inter-lab variability, and differences in the study groups. For example, within the child population, two of the outlier studies were conducted in Turkey,[26,85] although apparently using standard methodology. Therefore, overall pooled estimates do not represent true summary statistics in these situations.

IgA-AGA. Despite the apparent heterogeneity, one can make some broad statements regarding the diagnostic value of AGA antibodies. IgA-AGA appears to offer fair to good performance in children (Table 2; Figure 2).[26,27,29,31,34,36,38,42,43,50,52,58,59,64,66,68,69,83,85] Ten of the 19 studies demonstrated a sensitivity of IgA-AGA of greater than 80%, and six of the studies demonstrated a sensitivity of greater than 90%. However, nine studies demonstrated sensitivities of less than 80%. The specificity was greater than 80% in 15 of the 19 studies, and greater than 90% in 11 studies. Only four studies showed a specificity of less than 80%.

Ten studies assessed IgA-AGA in adults (Table 3; Figure 3).[30,33,54,62,63,71,80] Five of the ten studies demonstrated sensitivities greater than 80%, and three of the studies demonstrated sensitivities of greater than 90%. However, four studies demonstrated sensitivities of less than 65%. The specificity was greater than 80% in eight studies and greater than 90% in three. Five studies had specificities between 80% and 90%, and only two studies had specificities less than 80%.

Among the studies that assessed IgA-AGA in a mixed population of adults and children,[27,37,74,75] two demonstrated poor sensitivities of less than 70% but with specificities between 90% and 92%, one demonstrated a sensitivity of 85% and a specificity of 85%, and the last demonstrated a sensitivity of 91% and a specificity of 98% (Table 4; Figure 4).

Table 2: Included studies for IgA-AGA in children

Author, year; country	Study type	Biopsy criteria	Sens	Spec	PPV	NPV	Prev
Picarelli, 2000; Italy	Case-control	ESPGAN	22.2*	66.7*	50*	36.3*	0.60*
Gaetano, 1997; Italy	Case-control	ESPGAN	92	68	85.2	80.9	0.67
Carroccio, 1993; Italy	Case-control	Biopsies confirmed at diagnosis, on GFD, and rechallenge (severity grade - not reported)	68	91.7	86.1	79.7	0.43
Hansson, 2000; Sweden	Case-control	ESPGAN	95.5	73.9	77.8	94.4	0.49
Berger, 1996; Switzerland	Case-control	ESPGAN revised with complete villous atrophy	76	67	74	59	0.55
Lerner, 1994; USA, Israel	Case-control	Criteria of Townley modified by Ingkaran	52	94	87	74	0.52
Bahia, 2001; Brazil	Relevant clinical population	Severe villous atrophy	95.5	95.6	91.3	97.9	0.31
Russo, 1999; Canada	Relevant clinical population	ESPGAN	83.3	84.5	64.5	93.8	0.25
Bode, 1993; Denmark	Relevant clinical population	ESPGAN	64	99	90	97	0.07
Poddar, 2002; India	Relevant clinical population	ESPGAN (villous atrophy and unequivocal response to GFD)	94	91.5	92	93.5	0.52
Ascher, 1996; Sweden	Relevant clinical population	ESPGAN	100	94.4	95.7	100	0.55
Lindberg, 1985; Sweden	Relevant clinical population	ESPGAN; Alexander grading	88	88			0.31
Altuntas, 1998; Turkey	Relevant clinical population	Subtotal or total villous atrophy, crypt hyperplasia, increased IEL	23	90	75	48	0.55
Artan, 1998; Turkey	Relevant clinical population	ESPGAN ;	58	51	42.4	66.7	0.38
Rich, 1990; USA	Relevant clinical population	Not recorded - state "severe" lesion	53	93	72.7	85.7	0.25
Gonczi, 1991; Australia	Relevant clinical population (184 children with suspected CD)	ESPGAN no details on biopsy findings	95	92.4	76	98.6	0.20
Wolters, 2002; Netherlands	Relevant clinical population (identified retrospectively)	Subtotal villous atrophy with crypt hyperplasia	83	86	81	81	0.51
Lindquist, 1993; Sweden	Relevant clinical population (suspected celiac)	ESPGAN; subtotal or partial villous atrophy	86.5	92.7	93.7	85	0.55
Chirdo, 1999; Argentina	Relevant clinical trial	Total or subtotal villous atrophy	75	87.1	84	80	0.47
Chartrand, 1997; Canada	Relevant clinical population	ESPGAN - with flat mucosal biopsy	80	92	67	96	0.17
Meini, 1996; Italy	Relevant clinical population	Partial villous atrophy or total villous atrophy	0	100	0	91.7	0.08
*30 IgA-EMA-negative patients suspected of CD; 9 of 18 CD patients IgA deficient							

Table 3: Included studies for IgA-AGA in adults

Author, year; country	Study type	Biopsy criteria	Sens	Spec	PPV	NPV	Prev (%)
Sategana-Guidetti, 1995; Italy	Case-control	Roy-Choudhury criteria; partial or total villous atrophy	55	100	100	55.9	35.0
Dahele, 2001; Scotland	Case-control	Included 6 with IEL, rest partial villous atrophy or greater	61	86	88.5	42.7	43.6
Bode, 1994; Denmark	Relevant clinical population	Crypt hyperplasia, villous atrophy and increase inflammatory cells	46	98	75	92	25.7
Kaukinen, 2000; Finland	Relevant clinical population	Villous height to crypt ratio <2.0; IEL and HLA also tested	83	45	75	92	57.0
Maki, 1991; Finland	Relevant clinical population	Severe pathology with crypt hyperplasia to total villous atrophy; mild changes considered normal	30.8	87.2	22.2	91.3	14.8
McMillan, 1991; Ireland	Relevant clinical population	Revised ESPGAN	100	100	100	100	31.5
Bardella, 2001; Italy	Relevant clinical population	Marsh; no grade reported	95	89	76	98	33.3
Gonczi, 1991; Australia	Relevant clinical population (184 children with suspected CD)	ESPGAN no details on biopsy findings	92	88.2	85.2	93.8	45.8
Valdimarsson, 1996; Sweden	Relevant clinical population+ a few dypeptic controls	Alexander's classification; partial or subtotal villous atrophy	79	70	28	96	36.8
Vogelsang, 1995; Austria	Relevant study population	Modified ESPGAN; flat mucosa; crypt hyperplasia raised IELs	81.6	83	81.6	83	48.0

Table 4: Included studies for IgA-AGA in studies including both children and adults

Author, year; country	Study type	Biopsy criteria	Notes	Sens	Spec	PPV	NPV	Prev
Cataldo, 2000; Italy	Case-control	Original & revised criteria?	20 IgA-deficient CD vs healthy IgA-deficient non-CD	0	100	0	33.3	0.7
Sulkanen, 1998; Finland	Case-control	ESPGAN		84.5	81.6	75.2	89	0.4
Ascher, 1996; Sweden	Relevant clinical population	ESPGAN		90.9	98.5	98	92.7	0.5
Carroccio, 2002; Italy	Relevant clinical population	Marsh, broken down by criteria; CD was diagnosed as enlarged crypts and/or villous atrophy-with normalization on GFD		67	90	86	75	0.5
Tesei, 2003; Argentina	Relevant clinical population	Marsh II to IV - with confirmation		64	92	92	64	0.6

Figure 2: IgA-AGA in children with CD

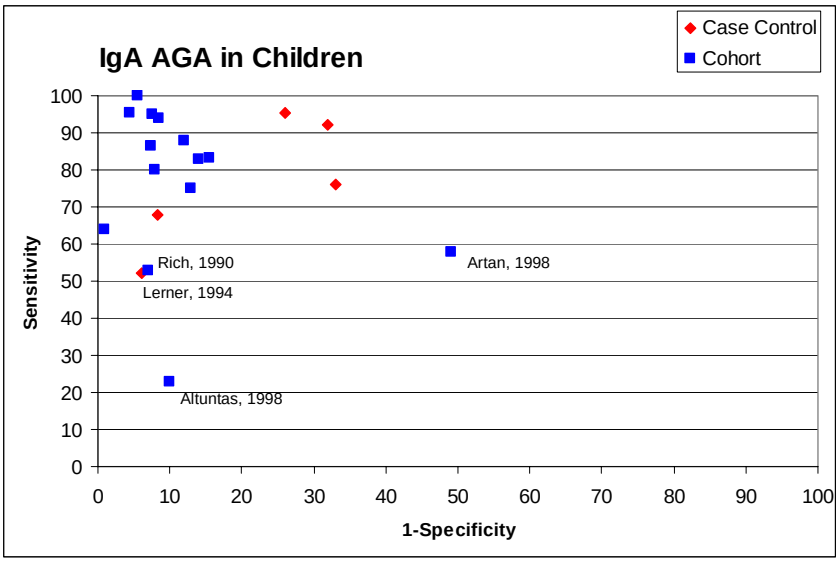


Figure 3: IgA-AGA in adults with CD

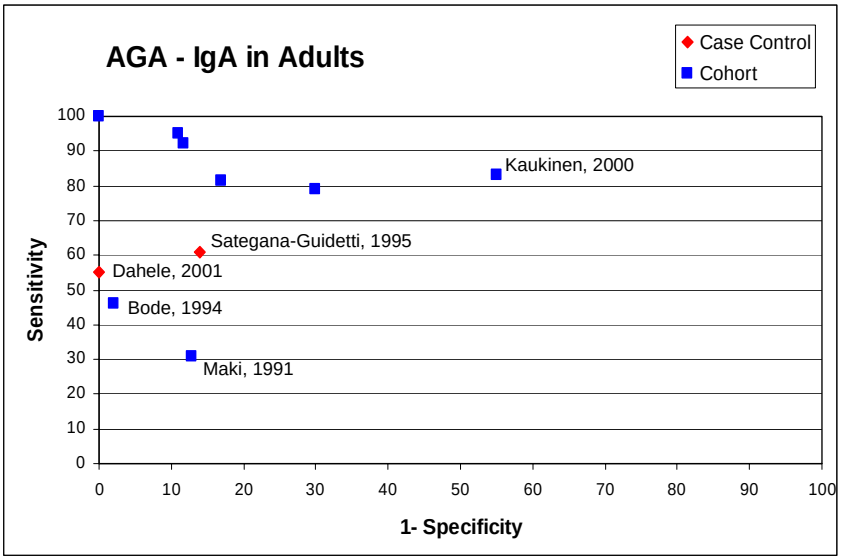
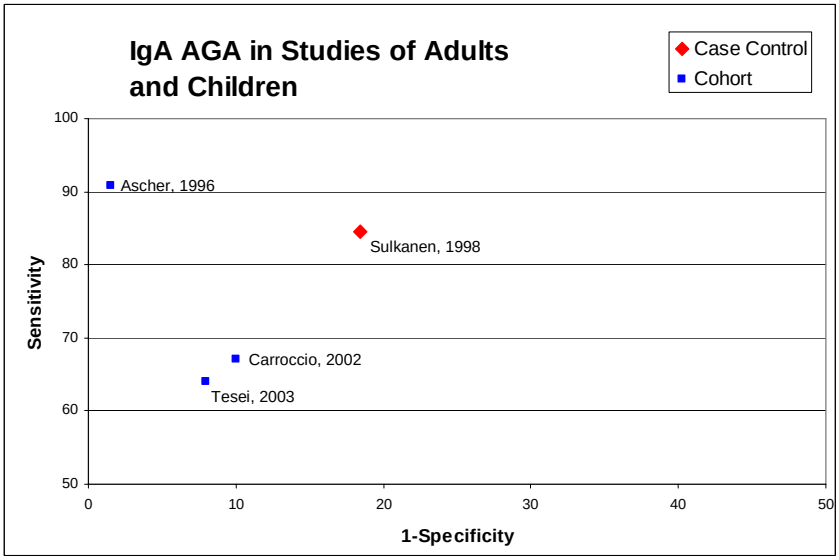


Figure 4: IgA-AGA in adults and children with CD



IgG-AGA. The seven studies of IgG-AGA in adults demonstrated considerably greater heterogeneity.[30,33,54,62,63,71,80] The sensitivity ranged from 17% to 100%, with little study grouping. However, there was less variation in the reported specificities. Five of the seven studies demonstrated specificities greater than 80%, whereas, the remaining two studies had specificities of greater than 70%. (Table 5; Figure 5)

In contrast, among the 17 analyzed studies (non-IgA deficient) of IgG-AGA conducted in children,[26,27,29,31,34,36,38,42,43,50,52,58,59,68,69,83,85] there seemed to be greater variability in the specificity than in the sensitivity (Table 6; Figure 6). Fifteen of the 17 studies demonstrated sensitivities that were greater than 80%, and six demonstrated sensitivities greater than 90%. Only two studies showed a sensitivity of less than 80%. In contrast, with regards to specificity, two groupings of studies become apparent. The first group consists of 11 studies, all of which had specificities greater than 79%, and except for one study, had sensitivities that were greater than 80%. In contrast, the second group of six studies all had specificities below 70%, and with the exception of one study, had sensitivities greater than 80%. (Tables and figures)

Four studies looked at IgG-AGA in a non-IgA-deficient mixed population of adults and children.[27,37,74,75] Two of these demonstrated sensitivities greater than 80%, one showed a sensitivity of 84%, whereas the second had a sensitivity of 96%. However, only the first study had specificity greater than 80%. In total, three of the four studies had specificities less than 80% (Table 7; Figure 7).

Table 5: Included studies for IgG-AGA in adults

Author, year, country	Study type	Biopsy criteria	Sens	Spec	PPV	NPV	Prev (%)
Sategana-Guidetti, 1995; Italy	Case-control	Roy-Choudhury criteria; partial or total villous atrophy	78	80.7	87.6	67.6	56.7
Bode, 1994; Denmark	Relevant clinical population	Crypt hyperplasia, villous atrophy and increase inflammatory cells	62	97	73	94	34.8
Kaukinen, 2000; Finland	Relevant clinical population	Villous height to crypt ration <2.0; IEL and HLA also tested	17	86	14	93.5	15.1
Maki, 1991; Finland	Relevant clinical population	Severe pathology with crypt hyperplasia to total villous atrophy; mild changes considered normal	46.2	89	33.3	93.3	14.8
McMillan, 1991; Ireland	Relevant clinical population	Revised ESPGAN	57	85	64	81	28.1
Gonczi, 1991; Australia	Relevant clinical population (184 children with suspected CD)	ESPGAN no details on biopsy findings	100	69.7	69.4	100	61.0
Vogelsang, 1995; Austria	Relevant study population	Modified ESPGAN; flat mucosa; crypt hyperplasia raised IELs	73.5	73.6	72	75	49.0

Table 6: Included studies for IgG-AGA in children

Author, year; country	Study type	Biopsy criteria	Sens	Spec	PPV	NPV	Prev
Picarelli, 2000; Italy	Case-control	ESPGAN	33.3	58.3	54.5	36.8	0.60
Gaetano, 1997; Italy	Case-control	ESPGAN	100	36	75.7	100	0.67
Carroccio, 1993; Italy	Case-control	Biopsies confirmed at diagnosis, on GFD, and rechallenge (severity grade – not recorded)	88.9	46.7	55.6	84.8	0.43
Hansson, 2000; Sweden	Case-control	ESPGAN	81.8	82.6	81.8	82.6	0.49
Berger, 1996; Switzerland	Case-control	ESPGAN revised with complete villous atrophy	69	59	68	53	0.55
Lerner, 1994; U.S.A, Israel	Case-control	Criteria of Townley modified by Ingkaran	88	92	88	92	0.52
Bahia, 2001; Brazil	Relevant clinical population	Severe villous atrophy	90.9	97.8	95.2	95.7	0.32
Russo, 1999; Canada	Relevant clinical population	ESPGAN	83.3	85.9	66.7	93.8	0.25
Bode, 1993; Denmark	Relevant clinical population	ESPGAN	71	99	100	98	0.07
Ascher, 1996; Sweden	Relevant clinical population	ESPGAN	100	66.7	75.6	100	0.55
Lindberg, 1985; Sweden	Relevant clinical population	ESPGAN; Alexander or Perea et al.	93	89	93.1	88.6	0.31
Altuntas, 1998; Turkey	Relevant clinical population	Subtotal or total villous atrophy, crypt hyperplasia, increased IEL	100	0	55	0	0.55
Artan, 1998; Turkey	Relevant clinical population	ESPGAN	83	59	55.6	85.2	0.38
Rich, 1990; USA	Relevant clinical population	Not reported - state "severe" lesion	100	58	44	100	0.25
Gonczi, 1991; Australia	Relevant clinical population (184 children with suspected CD)	ESPGAN no details on biopsy findings	100	92.4	76.9	100	0.20
Wolters, 2002; Netherlands	Relevant clinical population (identified retrospectively)	Subtotal villous atrophy with crypt hyperplasia	83	80	86	82	0.51
Chirdo, 1999; Argentina	Relevant clinical trial	Total or subtotal villous atrophy	85.7	80.6	80	86	0.47
Chartrand, 1997; Canada	Relevant clinical population	ESPGAN - with flat mucosal biopsy	83	79	45	96	0.17
Meini, 1996; Italy	Relevant clinical population	Partial villous atrophy or total villous atrophy	100	80	31.2	100	0.08

Table 7: Included studies for IgG-AGA in studies including both children and adults

Author, year; country	Study type	Biopsy criteria	Notes	Sens	Spec	PPV	NPV	Prev
Cataldo, 2000; Italy	Case-control	Original and revised criteria?	20 IgA-deficient CD vs healthy IgA-deficient non-CD	100	100	100	100	0.7
Sulkanen, 1998; Finland	Case-control	ESPGAN		69	73.4	63	78.3	0.4
Ascher, 1996; Sweden	Relevant clinical population	ESPGAN		96.4	69.2	72.6	95.7	0.5
Carroccio, 2002; Italy	Relevant clinical population	Marsh-broke down by criteria; CD was diagnosed as enlarged crypts and/or villous atrophy - with normalization on GFD		76	75	73.4	77.3	0.5
Tesei, 2003; Argentina	Relevant clinical population	Marsh II to IV - with confirmation		84	86	89	79	0.6

Figure 5: IgG-AGA in adults with CD

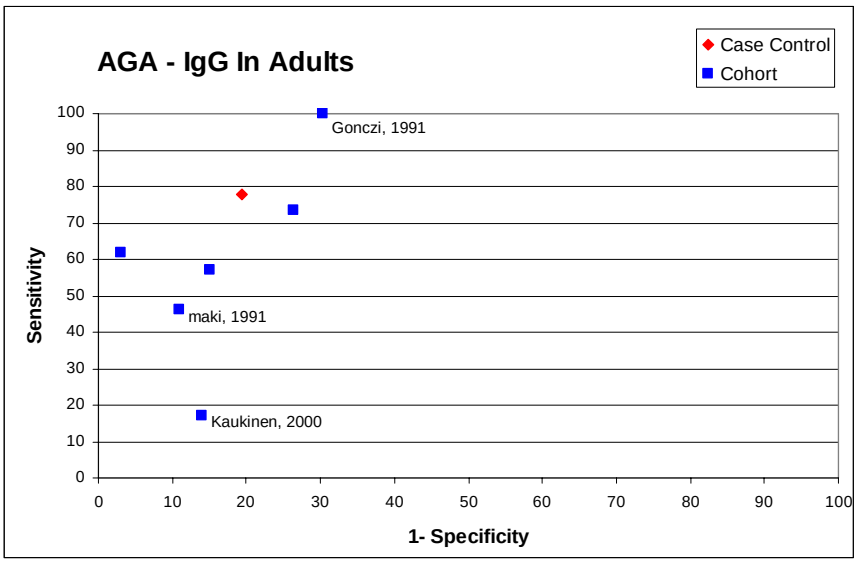


Figure 6: IgG-AGA in children with CD

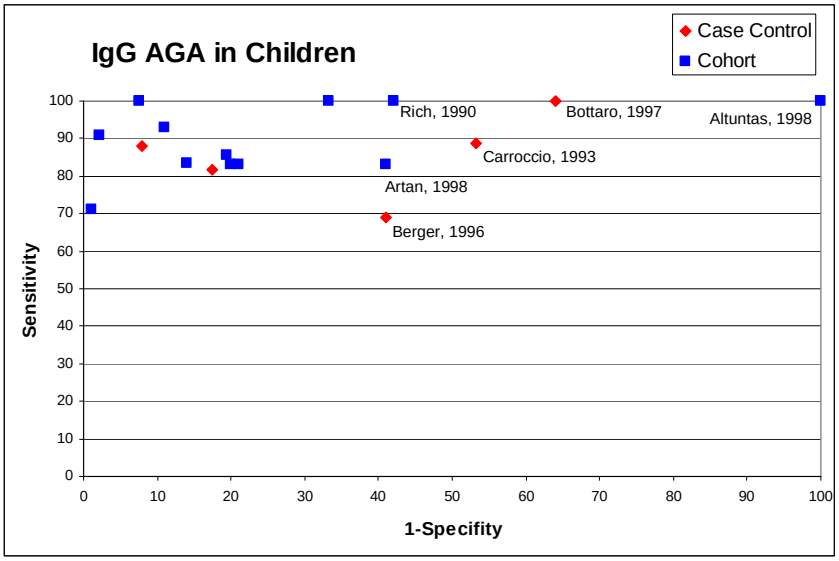
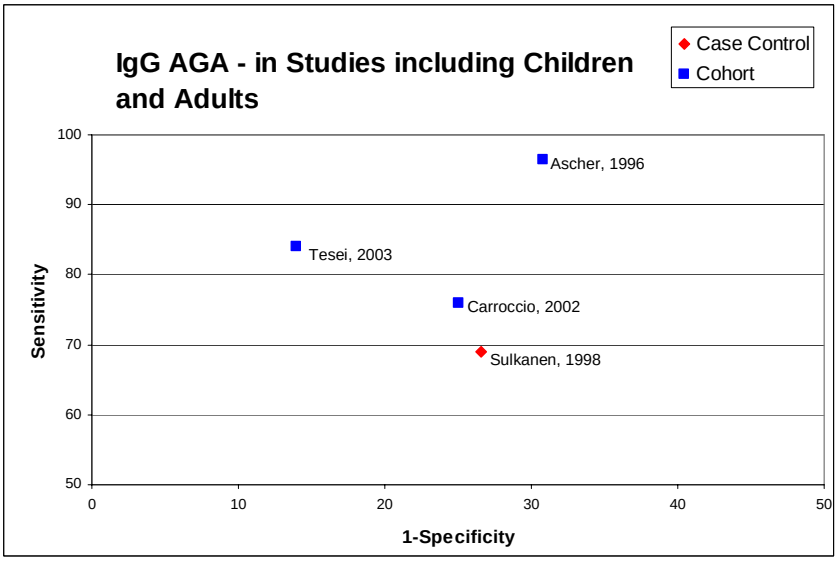


Figure 7: IgG-AGA in children and adults with CD



EMA

EMA—ME. The diagnostic characteristics of IgA-EMA-ME were assessed in 35 studies, and the diagnostic characteristics of IgG-EMA-ME were assessed in three studies. Of these included studies, 11 IgA-EMA-ME studies were conducted in adults,[30,32,39,51,57,63,71,77,78,80,81] 17 in children,[27,35,36,38,41,44,46,51,52,55,56,58,60,69,79,82,83] and five in a mixed population.[27,37,40,47,75] Some studies provided data for more than one age group. One study in children provided data on two different populations (including different control groups).[55] IgG-EMA-ME was assesed in one adult population,[63] one child population,[66] but not in any of the mixed-population studies.

One study was conducted in a population of known CD patients who had previously tested negative for EMA. In this study, the sensitivity and specificity of IgG EMA-ME were both 100%;[66] the performance of IgA-EMA was not reported. Another study that included CD patients with less than a Marsh IIIa grade,[37] demonstrated a sensitivity of 88%. Some studies only provided summary statistics without the raw two-by-two table results,[46,58,69] however, the raw data was abstracted based on the reported sensitivity and specificity, and the group sizes.

IgA-EMA-ME. Among the 11 studies of IgA-EMA-ME conducted in adults,[30,32,39,51,57,63,71,77,78,80,81] the specificity of the test was 100% in all except one, which showed a specificity of 97.2% (Table 8; Figure 8). The sensitivity of the test showed some slight variation among the studies. One outlier study demonstrated a sensitivity of only 74%;[77] however, the authors found that in the remaining five of 19 CD patients who tested negative for EMA, three were IgA deficient. If these patients were excluded, the sensitivity rose to 88%. The authors also go on to say that they seem to have a high proportion of IgA-deficient subjects in their referral base. The remaining ten studies showed sensitivities of 89% or greater. In fact, five studies showed a sensitivity of 100%, one a sensitivity of 99%, and another a sensitivity of 97%. In all, eight out of the 11 showed a sensitivity of 95% or greater, matching the very high specificity of this test. There was no statistical heterogeneity for this analysis. The pooled estimates for the sensitivity and specificity along with their 95% CI values were 97% (95% CI: 95.7-98.5) and 99.6% (95% CI: 98.8-99.9), respectively.

Among the 18 studies that assessed IgA-EMA-ME in children,[27,35,36,38,41,44,46,51,52,55,56,58,60,69,79,82,83] all but one outlier[69] were grouped together, and the sensitivities and specificities were both greater than 89% (Table 9; Figure 9). The outlier study demonstrated a sensitivity of only 74%, and also demonstrated low sensitivity for IgA-EMA-HU (see below).[69] The authors comment on the difficulties of interpretating immunofluorescence data as a likely explanation. Ten studies showed sensitivities greater than 95%, and except for one study with a sensitivity of 89%, the remaining seven studies had sensitivities between 90% and 95%. All these studies demonstrated specificities of 89% or greater, 16 had specificities greater than 90%, and 14 had specificities greater than 96%. There was no evidence of statistical heterogeneity in this analysis. The pooled sensitivity and specificity was 96.1% (95% CI: 94.4-97.3) and 97.4% (95% CI: 96.3-98.2), respectively.

Among the four studies in a mixed-age population that assessed IgA-EMA-ME,[27,37,47,75] all showed specificities of greater than 98% (Table 10; Figure 10). However, these studies showed some variation in the reported sensitivities. One study reported a very low sensitivity of 75%.[47] Two other studies showed a sensitivity of 86% and 88%, respectively, whereas the last showed a sensitivity of 98%.

Table 8: Included studies for IgA-EMA-ME in adults

Author, year; country	Study type	Biopsy criteria	Sens	Spec	PPV	NPV	Prev (%)
Hallstrom, 1989; Finland	Case-control	Flat mucosa	90.6	100	100	88.9	51.8
Biagi, 2001; Italy	Case-control	Partial villous atrophy or greater	94.6	100	100	94.5	49.1
Ladinser, 1994; Italy	Case-control	Revised ESPGAN	100	100.0	100	100	21.1
Sategana-Guidetti, 1995; Italy	Case-control	Roy-Choudhury criteria; partial or total villous atrophy	100	100	100	100	63.7
Valentini, 1994; Italy	Case-control	Partial villous atrophy or greater	99	100	100	96.7	76.2
Volta, 1995; Italy	Case-control	Roy-Choudhury criteria	95	100	100	97.1	35.6
Carroccio, 2002; Italy	Relevant clinical population	Ferguson and Murray; partial or total villous atrophy	100	100	100	100	11.6
McMillan, 1991; Ireland	Relevant clinical population	Revised ESPGAN	89.2	100	100	95.3	28.1
Bardella, 2001; Italy	Relevant clinical population	Marsh	100	97.2	93	100	28.7
Valdimarsson, 1996; Sweden	Relevant clinical population+ a few dypeptic controls	Alexander's classification; partial or subtotal villous atrophy	74	100	100	96	9.7
Vogelsang, 1995; Austria	Relevant study population	Modified ESPGAN; flat mucosa; crypt hyperplasia raised IELs	100	100	100	100	48.0

Table 9: Included studies for IgA-EMA-ME in children

Author, year; country	Study type	Biopsy criteria	Sens	Spec	PPV	NPV	Prev
Chirdo, 2000; Argentina	Case-control	ESPGAN	92.4	100	100	85.2	0.7
Kolho, 1997; Finland	Case-control	Revised ESPGAN	95	100	100	97	0.3
Kolho, 1997; Finland	Case-control	Revised ESPGAN	100	100	100	100	0.5
Whelan, 1996; Ireland	Case-control	Subtotal villous atrophy	100	100	100	100	0.4
Bonamico, 2001; Italy	Case-control	ESPGAN	95.1	98.2	90	44.3	0.5
Gaetano, 1997; Italy	Case-control	ESPGAN	96	96	97.9	92.3	0.7
Carroccio, 1993; Italy	Case-control	Biopsies confirmed at diagnosis, on GFD, and rechallenge (severity grade - not reported)	100	96.7	95.7	100	0.4
Di Leo, 2003; Italy	Case-control	ESPGAN	100	96.5	93.5	100	0.4
Vitoria, 2001; Italy	Case-control	Subtotal villous atrophy	100	100	100	100	0.6
Hansson, 2000; Sweden	Case-control	ESPGAN	95.5	100	100	95.8	0.5
Lerner, 1994; USA, Israel	Case-control	Criteria of Townley modified by Ingkaran	97	98	97	98	0.5
Hallstrom, 1989; Finland	Case-control	Flat mucosa	100	100	100	100	0.4
Chan, 2001; Canada	Relevant clinical population	Villous atrophy, crypt hyperplasia, increased lymphocytes	89	97	80	98	0.1
Russo, 1999; Canada	Relevant clinical population	ESPGAN	75	88.7	69.2	91.3	0.3
Ascher, 1996; Sweden	Relevant clinical population	ESPGAN	95.4	100	100	94.7	0.6
Wolters, 2002; Netherlands	Relevant clinical population (identified retrospectively)	Subtotal villous atrophy with crypt hyperplasia	92	90	90.5	92	0.5
Lindquist,1993; Sweden	Relevant clinical population (suspected CD)	ESPGAN; subtotal or partial villous atrophy	98.1	92.7	94.4	97.5	0.6
Kumar, 1989; USA, Israel	Relevant clinical population and control cases	ESPGAN + Townley	96.0	89.0	87.0	96.7	0.2

Table 10: Included studies for IgA-EMA-ME in studies including both children and adults

Author, year; country	Study type	Biopsy criteria	Notes	Sens	Spec	PPV	NPV	Prev
Cataldo, 2000; Italy	Case-control	Original & revised criteria?	20 IgA-deficient CD vs healthy IgA-deficient non-CD	0	100	0	33.3	0.7
Dickey, 2001; Northern Ireland	Case-control	Villous atrophy		75.3	98.3	98.2	76	0.6
Ascher, 1996; Sweden	Relevant clinical population	ESPGAN		98.2	100	100	98.5	0.5
Carroccio 2002; Italy	Relevant clinical population	Marsh - broke down by criteria; CD was diagnosed as enlarged crypts and/or villous atrophy - with normalization on a GFD		88	99	98.7	90	0.5
Tesei, 2003; Argentina	Relevant clinical population	Marsh II to IV - with confirmation		86	100	100	83	0.6

Figure 8: IgA-EMA-ME in adults with CD

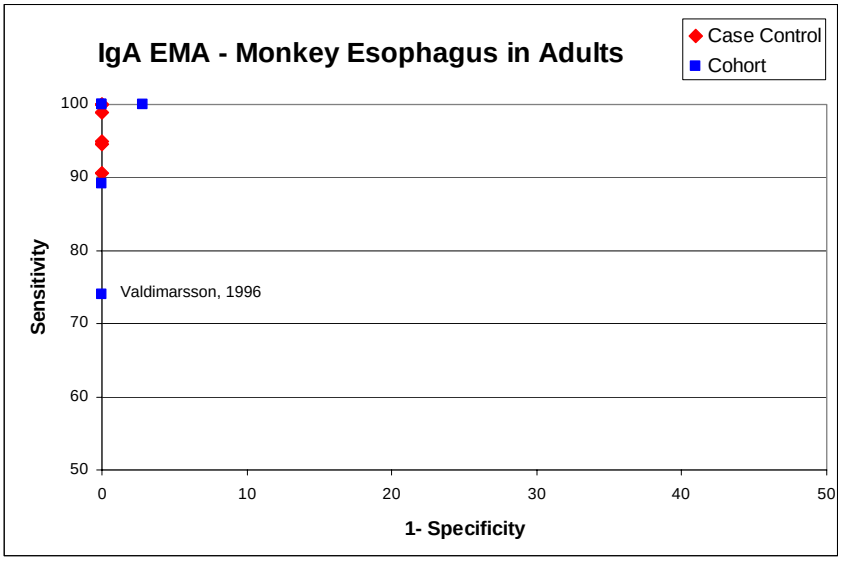


Figure 9: IgA-EMA-ME in children with CD

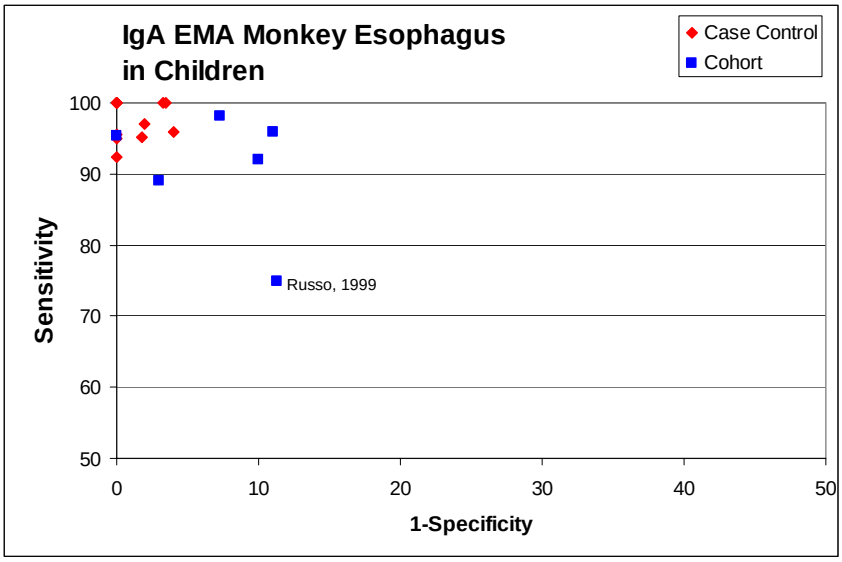
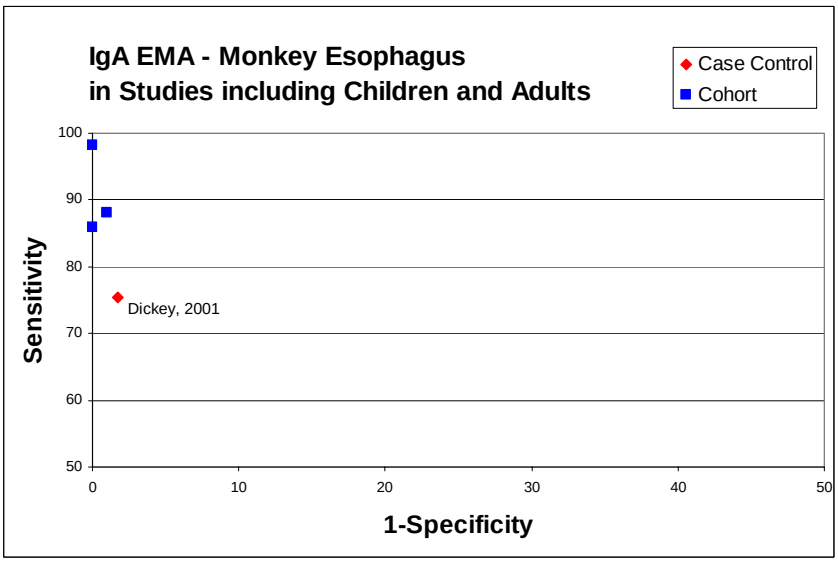


Figure 10: IgA-EMA-ME in adults and children with CD



IgG-EMA-ME. Only two studies meeting our inclusion criteria assessed IgG-EMA-ME, one in adults (Table 11),[63] and one in children (Table 12).[66] In the single adult study,[63] the sensitivity of the test was found to be 39%, whereas, the specificity was 98%. In a case-control study design, Picarelli et al. studied 30 IgA-EMA-negative children suspected of having CD.[66] Of these 30 children, 18 were subsequently found to have CD by duodenal biopsy and nine of the 18 were

found to be IgA deficient. In this highly selected population, the reported sensitivity and specificity of IgG-EMA-ME were both 100%.

Table 11: Included studies for IgG-EMA-ME in adults

Author, year; country	Study type	Biopsy criteria	Sens	Spec	PPV	NPV	Prev (%)
McMillan, 1991; Ireland	Relevant clinical population	Revised ESPGAN	39	98.3	92	78	13.5

Table 12: Included studies for IgG-EMA-ME in children

Author, year; country	Study type	Biopsy criteria	Notes	Sens	Spec	PPV	NPV	Prev
Picarelli, 2000; Italy	Case-control	ESPGAN	30 IgA-EMA neg. pts suspected of CD; 9/18 CD patients IgA deficient	100	100	100	100	0.1

EMA—HU. IgA-EMA-HU was assessed in 13 studies. Six of these studies were conducted in adults,[45,49,54,57,61,70,89] five in children,[36,53,55,69,70] and two in a mixed population.[72,74] One study provided summary statistics without the raw two-by-two table results,[69] however the raw data was calculated from the reported sensitivity and specificity and the group numbers. One study provided data on two different populations (including different control groups).[55]

IgG-EMA-HU was not assessed in any of the studies meeting our inclusion criteria.

Two studies included CD patients (both adult and children) with less than a Marsh IIIa grade, and reported IgA-EMA-HU sensitivities of 87% and 100%.[45]
[70]

IgA-EMA-HU. Six studies in adults assessed IgA-EMA-HU (Table 13; Figure 11).[45,49,54,57,61,70,89] In all six, the specificity was reported to be 100%. There was, however, variability in the reported sensitivities, which ranged from 87% to 100%. Three studies demonstrated sensitivities between 87% and 89%, two between 90% and 95% and one showing a sensitivity of 100%. There was no observed statistical heterogeneity for this analysis. The pooled sensitivity and specificity was found to be 90.2% (95% CI: 85.9-93.4) and 100% (95% CI: 99.1-100), respectively.

Five studies with six separate child populations assessed IgA-EMA-HU (Table 14; Figure 12).[36,53,55,69,70] Four of the six studies were grouped together and revealed sensitivities between 94% and 100%, and specificities of 100%. Of the two outliers,[90] one showed a sensitivity of 100% and a specificity of 77%. The other study,[69] was an outlier in other analyses, and demonstrated a sensitivity of 46% and a specificity of 96%. The authors comment on difficulties of interpretation of the immunofluorescence as a likely explanation. After accounting for this study, there was no statistical heterogeneity documented for sensitivity. The pooled sensitivity for this analysis was 96.9% (95% CI: 93.5-98.6). A pooled specificity for this analysis was not calculated, but is likely close to 100% given that four of the five grouped studies demonstrated a specificity of 100%.

Two studies assessed IgA-EMA-HU in a mixed-age population (Table 15; Figure 13).[72,74] In both these studies, the specificity was 100% (95% CI: 97.5-100) and the sensitivity 93% (95% CI: 88.1-95.4).

Table 13: Included studies for IgA-EMA-HU in adults

Author, year; country	Study type	Biopsy criteria	Sens	Spec	PPV	NPV	Prev (%)
Gillbert, 2000; Canada	Case-control	Mild, moderate, severe villous atrophy	100	100	100	100	33.3
Ladinser, 1994; Italy	Case-control	Revised ESPGAN	90	100	100	98	18.9
Salmaso, 2001; Italy	Case-control	Grades I-IV Marsh with response to a GFD	87	100	100	95.1	24.7
Volta, 1995; Italy	Case-control	Roy-Choudhury criteria	95	100	100	97.1	35.6
Dahele, 2001; Scotland	Case-control	Included 6 with IEL, rest partial villous atrophy or greater	87	100	100	81.3	55.3
Kaukinen, 2000; Finland	Relevant clinical population	Villous height to crypt ration <2.0; IEL and HLA also tested	88.9	100	100	98.9	7.6

Table 14: Included studies for IgA-EMA-HU in children

Author, year; country	Study type	Biopsy criteria	Sens	Spec	PPV	NPV	Prev
Kolho, 1997; Finland	Case-control	Revised ESPGAN	95	100	100	97	0.3
Kolho, 1997; Finland	Case-control	Revised ESPGAN	100	100	100	100	0.5
Gaetano, 1997; Italy	Case-control	ESPGAN	94	100	100	89.2	0.7
Salmaso, 2001; Italy	Case-control	Grades I-IV Marsh with response to GFD	100	100	100	100	0.6
Russo, 1999; Canada	Relevant clinical population	ESPGAN	45.8	95.8	78.6	84	0.3
Iltanen, 1999 Finland	Relevant clinical population	ESPGAN - CD confirmed at follow-up	100	77.1	60.1	100	0.3

Table 15: Included studies for IgA-EMA-HU in studies including both children and adults

Author, year; country	Study type	Biopsy criteria	Sens	Spec	PPV	NPV	Prev
Sblaterro, 2000; Italy	Case-control	ESPGAN	93	100	100	80	0.8
Sulkanen, 1998; Finland	Case-control	ESPGAN	92.6	99.5	99.2	94.9	0.4

Figure 11: IgA-EMA-HU in adults with CD

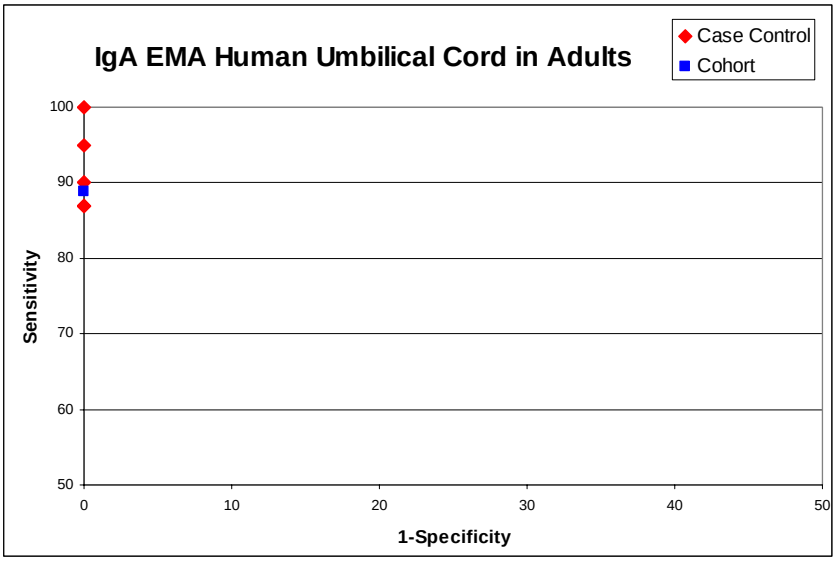


Figure 12: IgA-EMA-HU in children with CD

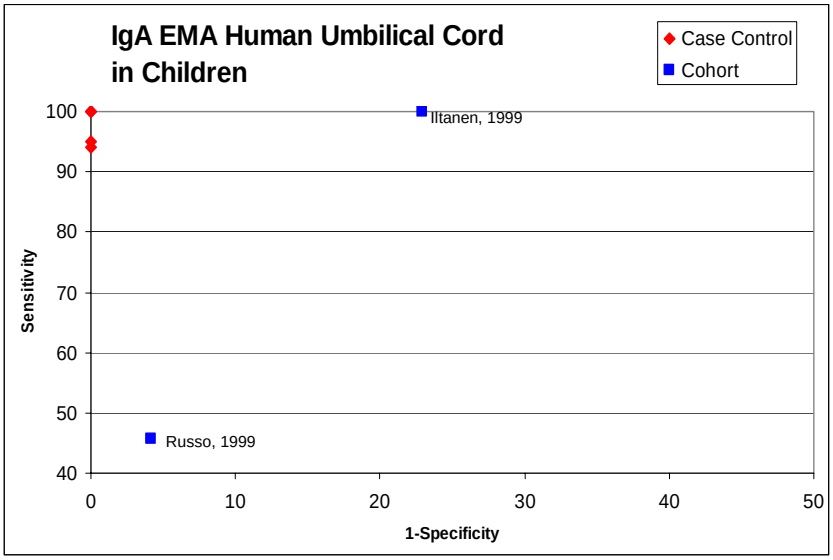
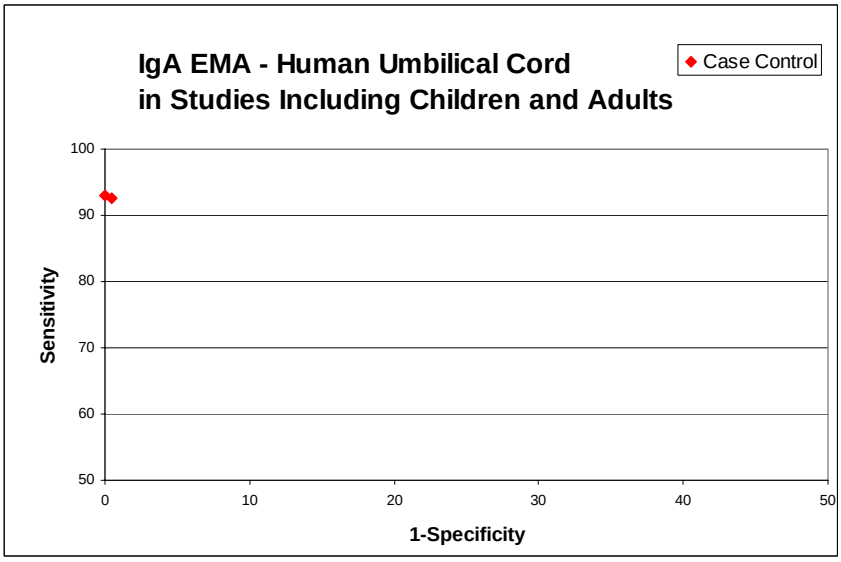


Figure 13: IgA-EMA-HU in adults and children with CD



tTG antibodies

tTG—GP liver. The diagnostic characteristics of IgA-tTG-GP were assessed by ELISA in nine studies**,** and the diagnostic characteristics IgG-tTG-GP assessed by ELISA in three studies. Of the IgA-tTG–GP studies, five were conducted in adults,[30,32,39,45,70] five in children,[35,41,52,70,83] and four in a mixed population.[47,72,74,76] One study provided separate data for more than one age group.[70]

Of the IgG-tTG-GP studies that met the inclusion criteria, none were in adults or children, although two studies were in a mixed population.[72,76]

Two studies included CD patients with less than a Marsh IIIa grade.[45,70] These studies demonstrated sensitivities of 81% and 95% for IgA-tTG-GP.

IgA-tTG-GP. In the analysis of IgA-tTG-GP in adults, five studies grouped themselves by study design.[30,32,39,45,70] The two cohort studies (relevant clinical population)[30,39] both showed sensitivities of 100%, and specificities of 92% and 98%, respectively. On the other hand, the three case-control studies[32,45,70] demonstrated high specificities (97% to 98%), but sensitivities of only 81% to 88% (Table 16; Figure 14). This analysis did not show statistical heterogeneity, but the differences by study design were striking, so a pooled estimate for sensitivity was not performed. The pooled specificity was 95.3% (95% CI: 92.5-98.1).

The analysis of IgA-tTG-GP in children showed very little variability in either the sensitivity, or specificity (Table 17; Figure 15). Among these five studies,[35,41,52,70,83] the sensitivities ranged from 89% to 96%. The specificities were all greater than 92%, with three studies showing specificities greater than 96%,[41,52,83] and two studies having a sensitivity of 100%.[35,70] The pooled estimates of the sensitivity and specificity were 93.1% (95% CI: 88.8-95.9) and 96.3% (95% CI: 93.1-98.0), respectively (Table 17; Figure 15)

Among the studies of mixed-age groups,[47,72,74,76] there was one outlier study with a sensitivity of only 84% but a specificity of 100% (Table 18).[72] The specificities of the remaining studies were all greater than 94% (Table 18; Figure 16), and the sensitivities were between 92% and 95%. Heterogeneity was detected in the estimates of sensitivity, but not for specificity. The pooled specificity was 95.4% (95% CI: 92.7-97.2).

Table 16: Included studies for IgA-tTG-GP in adults

Author, year; country	Study type	Biopsy criteria	Sens	Spec	PPV	NPV	Prev (%)
Biagi, 2001; Italy	Case-control	Partial villous atrophy or greater	87.5	98.1	98	87.1	46.3
Salmaso, 2001; Italy	Case-control	Grades I-IV Marsh with response to a GFD	87	97	90.9	94.9	27.2
Dahele, 2001; Scotland	Case-control	Included 6 with IEL, rest partial villous atrophy or greater	81	97	97.9	74.1	52.5
Carroccio, 2002; Italy	Relevant clinical population	Ferguson and Murray; partial or total villous atrophy	100	92	60	100	18.8
Bardella, 2001; Italy	Relevant clinical population	Marsh	100	98.2	83.3	100	10.0

Table 17: Included studies for IgA-tTG-GP in children

Author, country; year	Study type	Biopsy criteria	Sens	Spec	PPV	NPV	Prev
Bonamico, 2001; Italy	Case-control	ESPGAN	90.3	100	100	30.3	0.5
Salmaso, 2001; Italy	Case-control	Grades I-IV Marsh with response to a GFD	95	100	100	94.1	0.6
Hansson, 2000; Sweden	Case-control	ESPGAN	90.9	95.7	95.2	91.7	0.5
Chan, 2001; Canada	Relevant clinical population	Villous atrophy, crypt hyperplasia, increase lymphocytes	89	94	67	98	0.1
Wolters, 2002; Netherlands	Relevant clinical population (identified retrospectively)	Subtotal villous atrophy with crypt hyperplasia	96	92	92.6	95.7	0.5

Table 18: Included studies for IgA-tTG-GP in studies including both adults and children

Author, year, country	Study type	Biopsy criteria	Sens	Spec	PPV	NPV	Prev
Dickey, 2001; Northern Ireland	Case-control	Villous atrophy	93.2	96.6	97.1	91.8	0.6
Sblaterro, 2000; Italy	Case-control	ESPGAN	84	100	100	62.5	0.8
Sulkanen, 1998; Finland	Case-control	ESPGAN	95	93.7	90.8	96.5	0.4
Troncone, 1999; Italy	Relevant clinical population	ESPGAN	91.7	98	98	94	0.4

Figure 14: IgA-tTG-GP in adults with CD

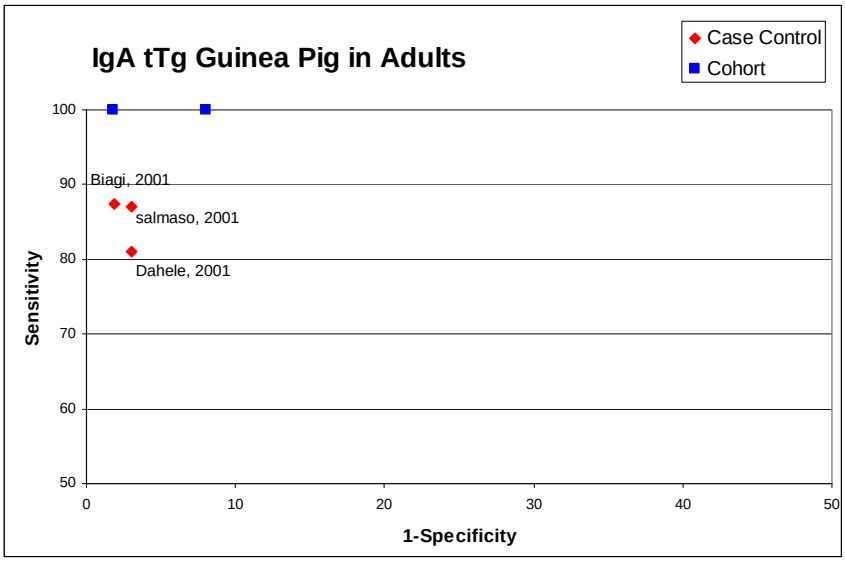


Figure 15: IgA-tTG-GP in children with CD

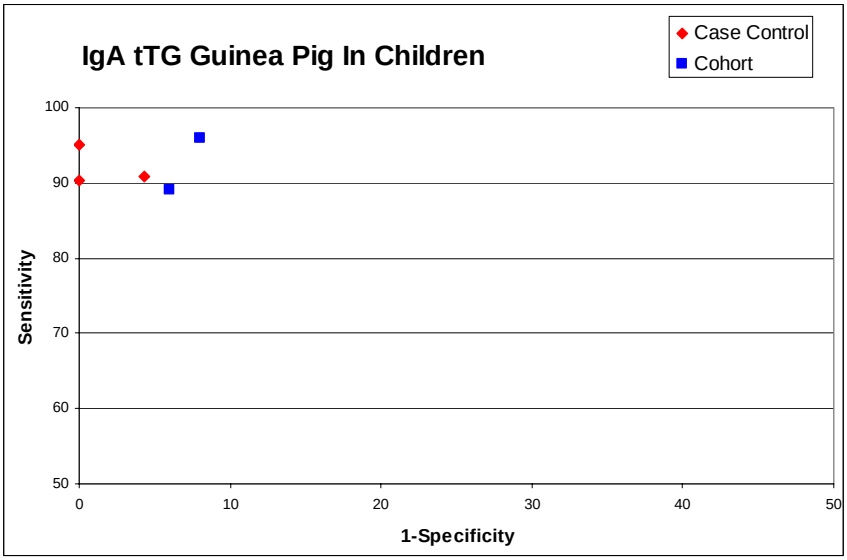
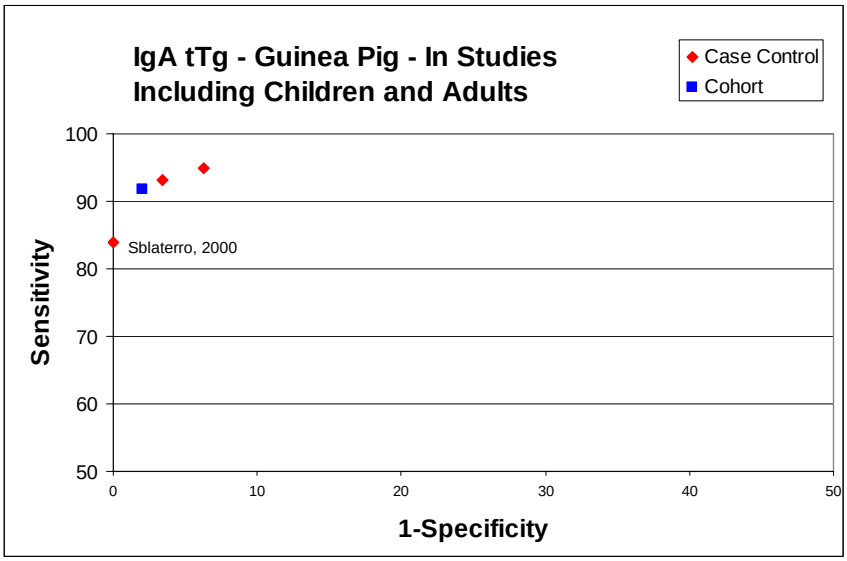


Figure 16: IgA-tTG-GP in adults and children with CD

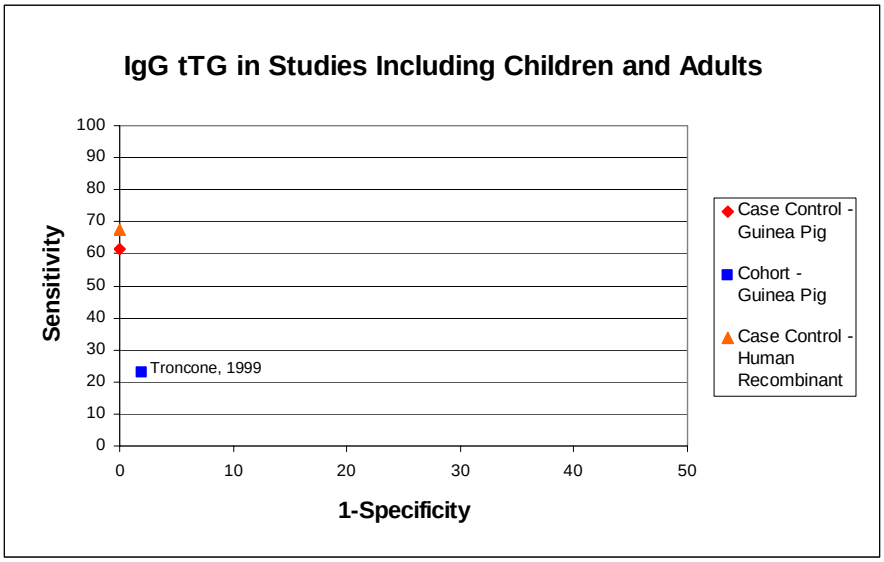


IgG-tTG-GP. Two studies in a mixed-age population assessed IgG-tTG- GP (Table 19; Figure 17).[72,76] The specificities in both studies were greater than 98%, but the sensitivities were 23% and 62%, respectively.

Table 19: Included studies for IgG-tTG-GP in studies including both children and adults

Author, year; country	Study type	Biopsy criteria	Sens	Spec	PPV	NPV	Prev
Sblaterro, 2000; Italy	Case-control	ESPGAN	61.5	100	100	44.4	0.8
Troncone, 1999; Italy	Relevant clinical population	ESPGAN	23	98	92	63	0.4

Figure 17: IgG-tTG-GP in adults with CD



tTG – human recombinant (HR)

IgG-tTG-HR. The diagnostic characteristics of IgA-tTG-HR were assessed by ELISA in ten studies, and the diagnostic characteristics IgG-tTG-HR were assessed by ELISA in two studies. Of the IgA-tTG-HR studies, three were conducted in adults,[39,49,54] three in children,[52,79,83] and three in a mixed population.[40,72,75]

Of the IgG-tTG-HR studies, two were conducted in a mixed population (Table 20),[40,72] but none were conducted in adults or children. One study was conducted in IgA-deficient patients and is described below.[40]

Two studies included CD patients with less than a Marsh IIIa grade.[45,70] These studies demonstrated sensitivities of 81% and 95% for IgA-tTG-GP.

One study was conducted in a mixed-age population of patients with known IgA deficiency.[40] In this study, the sensitivity of IgA-tTG–HR was 0%, wheras, the sensitivities and specificities of IgG-tTG-HR were 100% and 80%, respectively.

Table 20: Included studies for IgG-tTG-HR in studies including both children and adults

Author, year; country	Study type	Biopsy criteria	Notes	Sens	Spec	PPV	NPV	Prev
Cataldo, 2000; Italy	Case-control	Original & revised criteria?	20 IgA-deficient CD vs healthy IgA-deficient non-CD	100	80	90.1	100	0.7
Sblaterro, 2000; Italy	Case-control	ESPGAN		67.6	100	100	48.7	0.8

IgA-tTG-HR. Three studies assessed IgA-tTG-HR in an adult population (Table 21; Figure 18).[39,49,54] There was very little variability in the reported values for the sensitivities and specificities. The sensitivities were 100% in two studies, and 95% in the other. The specificities were 100% in two studies, and 97% in another. The pooled estimates of the sensitivity and specificity were 98.1% (95% CI: 90.1%-99.7%) and 98.0% (95% CI: 95.8-99.1), respectively.

Among the three studies in children (Table 22; Figure 19),[52,79,83] the sensitivities were 96% in two studies and 95% in one. The specificities were 100% in two studies, and 96% in one. The pooled estimates of the sensitivity and specificity were 95.7% (95% CI: 90.3-98.1) and 99.0% (95% CI: 94.6-99.8), respectively.

Only two studies assessed the IgA-tTG-HR in a mixed-age population without IgA deficiency (Table 23; Figure 20).[72,75] The sensitivities and specificities were 92% and 100%, respectively, for the first study, and 91% and 96%, respectively, for the second. The pooled estimates of the sensitivity and specificity were 90.2% (95% CI: 86.4-93.0) and 95.4% (95% CI: 91.5- 97.6), respectively.

Overall, these studies demonstrated a specificity of close to 100% and sensitivity in the range of 90% to 96%.

Table 21: Included studies for IgA-tTG-HR in adults

Author, year; country	Study type	Biopsy criteria	Sens	Spec	PPV	NPV	Prev (%)
Carroccio, 2002; Italy	Relevant clinical population	Ferguson and Murray; partial or total villous atrophy	100	97	80	100	14.5
Gillbert, 2000; Italy	Case-control	Mild, moderate, severe villous atrophy	95.2	100	95.2	100	31.7
Kaukinen, 2000; Finland	Relevant clinical population	Villous height to crypt ration <2.0; IEL and HLA also tested	100	100	100	100	8.7

Table 22: Included studies for IgA-tTG-HR in children

Author, year; country	Study type	Biopsy criteria	Sens	Spec	PPV	NPV	Prev
Vitoria, 2001; Italy	Case-control	Subtotal villous atrophy	95	100	100	93	0.6
Hansson, 2000; Sweden	Case-control	ESPGAN	95.5	95.7	95.5	95.7	0.5
Wolters, 2002; Netherlands	Relevant clinical population (identified retrospectively)	Subtotal villous atrophy with crypt hyperplasia	96	100	100	96	0.5

Table 23: Included studies for IgA-tTG-HR in studies including both children and adults

Author, year; country	Study type	Biopsy criteria	Notes	Sens	Spec	PPV	NPV	Prev
Cataldo, 2000; Italy	Case-control	Original & revised criteria?	20 IgA deficient CD vs healthy IgA-deficient non-CD	0	100	0	33.3	0.7
Sblaterro, 2000; Italy	Case-control	ESPGAN		91.5	100	100	76.9	0.8
Tesei, 2003; Argentina	Relevant clinical population	Marsh II to IV - with confirmation		91	96	97	87	0.6

Figure 18: IgA-tTG-HR in adults with CD

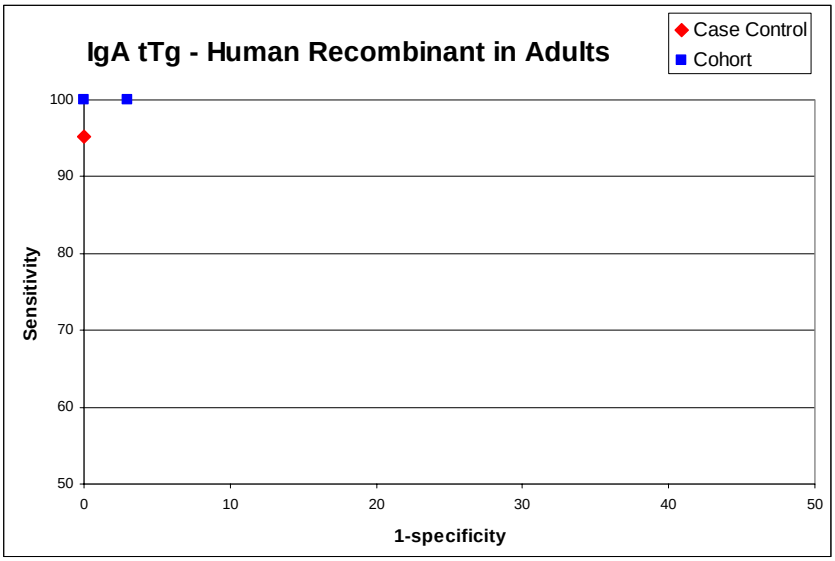


Figure 19: IgA-tTG-HR in children with CD

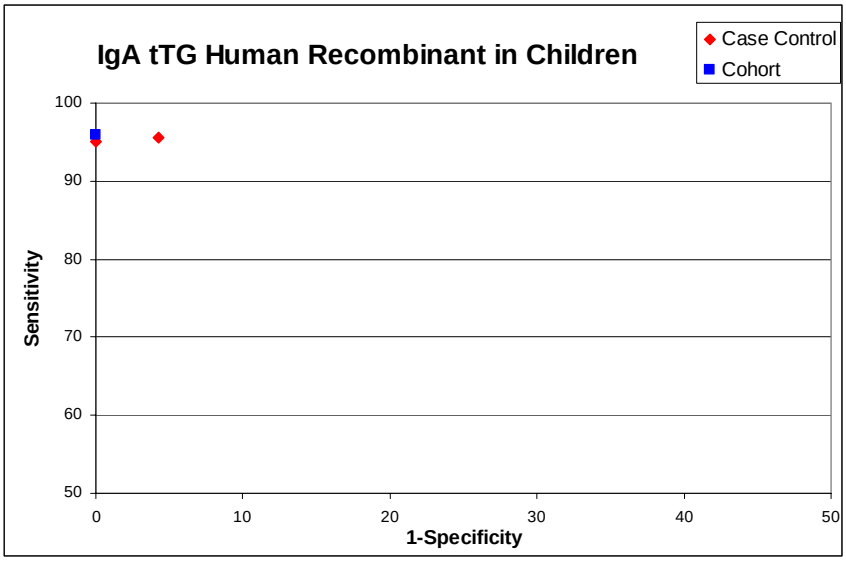
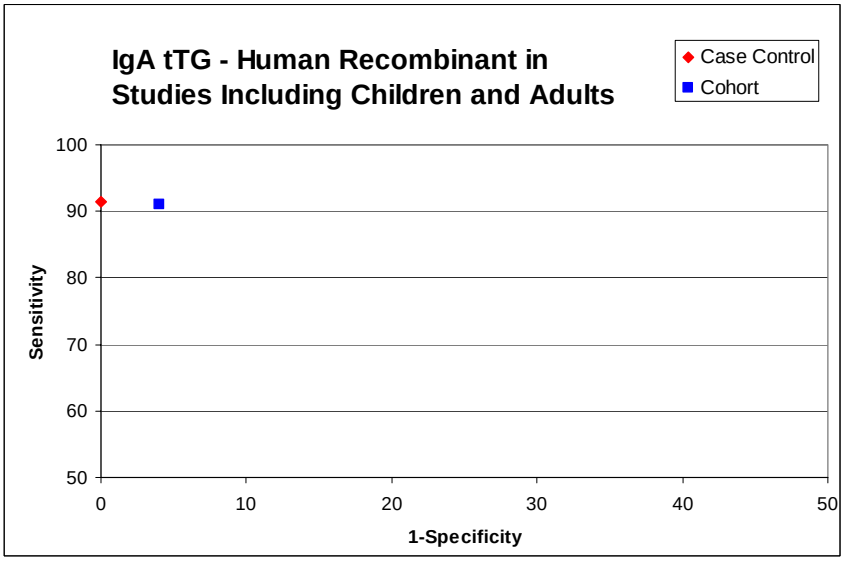


Figure 20: IgA-tTG-HR in adults and children with CD



IgG-tTG-HR, IgA deficient. Only one study of IgG-tTG-HR, conducted in an IgA-deficient population, was identified.[72] In this study, the sensitivity and specificity of IgG-tTG-HR was 68% and 100%, respectively.

Mixed-antibody combinations. Several studies were identified that tested different antibodies in combination. Six studies in children assessed the use of IgA- and IgG-AGA (Table 24).[34,42,48,50,59,85] When either of these tests were positive, the resulting sensitivities ranged from 83% to 100%, and the specificities ranged from 71% to 99%. One study, that apparently used similar methodologies, had the lowest sensitivity (83%) and specificity (36%) of the group.[85] When the same authors tested the antibodies under the requirement of both tests being concordant, the sensitivity fell, as would be expected, to 50%, and the specificity rose to 67%.[85] Three adult studies were identified that used IgA- and IgG-AGA in an either/or protocol (Table 24).[33,50,78] As was observed in the studies of children, significant between-study differences existed, making pooled estimates inappropriate. Nonetheless, in these studies the sensitivity ranged from 77% to 100%, while the specificity ranged from 90% to 97%.

One study in a mixed-age population assessed the use of a combination of IgA- and IgG-tTG-HR antibodies (Table 25).[72] In this study, the sensitivity when either test was positive was 98.5%, while the specificity remained high at 100%. Another study in children assessed the combination of IgA-AGA and IgA-EMA-HU when either test was positive, and found a sensitivity of 100% and a specificity of 73% (Table 26).[69] This same study assessed the same antibodies under the situation where both tests needed to be concordant. In this circumstance, the sensitivity remained 100% and the specificity rose to 93%.

In general, combining tests when either test is positive tended to improve sensitivity at the cost of specificity, while a requirement for the tests to be concordant tended to improve specificity.

Table 24: Included studies for combination IgA and IgG AGA, when either test is positive

Author, year; country	Study type	Biopsy criteria	Notes	Sens	Spec	PPV	NPV	Prev
Valentini, 1994; Italy	Case-control	Partial villous atrophy or greater	Adults	92	90	96.8	77.1	0.76
Bode, 1994; Denmark	Relevant clinical population	Crypt hyperplasia, villous atrophy and increase inflammatory cells	Adults	77	95	71	97	0.41
Gonczi, 1991; Australia	Relevant clinical population (184 children with suspected celiac)	ESPGAN no details on biopsy findings	Adults	100	97.1	96.2	100	0.44
Bode, 1993; Denmark	Relevant clinical population	ESPGAN	Children	86	99	92	99	0.1
Falth-Magnusson, 1994; Sweden	Relevant clinical population	ESPGAN + Alexander grading IV, grade III to IV challenge	Children	88.5	93.7	88.8	93.5	0.4
Lindberg, 1985; Sweden	Relevant clinical population	ESPGAN, Alexander grading	Children	97	83	41.8	98.2	0.3
Artan, 1998; Turkey	Relevant clinical population	ESPGAN	Children: IgA AGA or IgG AGA	83	36	44	77.8	0.3
Gonczi, 1991; Australia	Relevant clinical population (184 children with suspected CD)	ESPGAN no details on biopsy findings	Children	100	98.7	95.2	98.7	0.2
Chartrand, 1997; Canada	Relevant clinical population	ESPGAN – with flat mucosal biopsy	Children	93	71	43	98	0.2

Table 25: Included studies for combination IgA and IgG tTG-HR, when either test is positive

Author, year; country	Study type	Biopsy criteria	Notes	Sens	Spec	PPV	NPV	Prev
Sblaterro, 2000; Italy	Case-control	ESPGAN	Adults and children	98.5	100	100	95.2	0.8

Table 26: Included studies for combination IgA-AGA and IgG-EMA-HU, when either test is positive

Author, year; country	Study type	Biopsy criteria	Notes	Sens	Spec	PPV	NPV	Prev
Russo, 1999; Canada	Relevant clinical population	ESPGAN	Children	100	73	57	82	0.3

Prevalence of CD and the positive predictive value (PPV) and negative predictive value (NPV) of serology. The prevalence of CD in the tested populations is presented in Tables 2 to 26 for the individual studies, and in Table 27 for the pooled estimate for the analysis groups.

The minimum prevalence of CD in individual study populations was greater than 25% in most of the studied analysis groups (i.e., IgA-AGA, IgG-AGA, etc), except for ten analysis groups where the minimum prevalence was between 9% and 12%. In all the analysis groups, the maximum prevalence ranged from 30% to as high as 70%. The pooled prevalence for the analysis groups was predominantly between 30% and 45%.

In assessing the IgA-EMA and IgA-tTG analysis groups, the pooled prevalence ranged from 33% to 46% except for the analysis of IgA-tTG-HR in adults, which showed a pooled prevalence of 16%. Figure 21 is a plot of the individual study prevalence versus the study's PPV, and suggests that below a CD prevalence of about 35% to 40%, the PPV of these IgA-based tests tends to drop from about 90% to 100%, to about 80% or less. As expected, Figure 22 demonstrates the reverse relationship, with the NPV being between 95% and 100% up to a CD prevalence of about 45%, and then dropping off.

Table 27: Weighted pooled estimates with 95% CIs and heterogeneity identified

Analysis	Sens	L 95% CI:	U 95% CI:	Spec	L 95% CI:	U 95% CI:	Prev	L 95% CI:	U 95% CI:	PPV	L 95% CI:	U 95% CI:	NPV	L 95% CI:	U 95% CI:
IgA-AGA–ADULT	H	H	H	H	H	H	0.358	0.332	0.385	H	H	H	H	H	H
IgG-AGA–ADULT	H	H	H	H	H	H	0.367	0.335	0.401	H	H	H	H	H	H
IgA-EMA-ME–ADULT	0.974	0.957	0.985	0.996	0.988	0.999	0.398	0.371	0.425	0.974	0.957	0.985	0.996	0.988	0.999
IgG-EMA-ME–ADULT (one study)	0.393	0.236	0.576	0.984	0.913	0.997	0.135	0.079	0.221	0.393	0.236	0.576	0.984	0.913	0.997
IgA-EMA-HU–ADULT	0.902	0.859	0.934	1.000	0.991	1.000	0.331	0.297	0.368	0.902	0.859	0.934	1.000	0.991	1.000
IgA-tTG-GP—ADULT	0.859	0.808	0.898	0.953	0.930	0.969	0.312	0.279	0.348	0.859	0.808	0.898	0.953	0.930	0.969
IgA-tTG-HR–ADULT	0.981	0.901	0.997	0.981	0.958	0.991	0.160	0.126	0.202	0.981	0.901	0.997	0.981	0.958	0.991
IgA-AGA–CHILD	H	H	H	H	H	H	0.363	0.341	0.385	H	H	H	H	H	H
IgG-AGA–CHILD	H	H	H	H	H	H	0.437	0.413	0.462	H	H	H	H	H	H
IgA-EMA-ME–CHILD	0.961	0.945	0.973	0.974	0.963	0.982	0.400	0.378	0.423	0.961	0.945	0.973	0.974	0.963	0.982
IgA-EMA-HU–CHILD	0.969	0.935	0.986	H	H	H	0.447	0.402	0.493	0.969	0.935	0.986	0.949	0.915	0.970
IgA-tTG-GP–CHILD	0.931	0.888	0.959	0.963	0.931	0.980	0.446	0.401	0.493	0.931	0.888	0.959	0.963	0.931	0.980
IgA-tTG-HR–CHILD	0.957	0.903	0.981	0.990	0.946	0.998	0.519	0.452	0.584	0.957	0.903	0.981	0.990	0.946	0.998
IgA-AGA–MIXED	H	H	H	H	H	H	0.415	0.386	0.444	H	H	H	H	H	H
H = significant heterogeneity by Pearson's Chi square															

Table 27 (cont'd): Weighted pooled estimates with 95% CIs and heterogeneity identified

Analysis	Sens	L 95% CI:	U 95% CI:	Spec	L 95% CI:	U 95% CI:	Prev	L 95% CI:	U 95% CI:	PPV	L 95% CI:	U 95% CI:	NPV	L 95% CI:	U 95% CI:
IgG-AGA–MIXED	H	H	H	H	H	H	0.510	0.480	0.540	H	H	H	H	H	H
IgA-EMA-ME–MIXED	H	H	H	0.995	0.982	0.999	0.467	0.434	0.500	0.859	0.825	0.888	0.995	0.982	0.999
IgA-EMA-HU–MIXED	0.925	0.881	0.954	0.996	0.975	0.999	0.437	0.391	0.484	0.925	0.881	0.954	0.996	0.975	0.999
IgA-tTG-GP–MIXED	H	H	H	0.954	0.927	0.972	0.463	0.425	0.501	0.913	0.877	0.939	0.954	0.927	0.972
IgG-tTG-GP–MIXED	0.451	0.363	0.543	0.988	0.935	0.998	0.265	0.208	0.331	0.451	0.363	0.543	0.988	0.935	0.998
IgA-tTG-HR–MIXED	0.902	0.864	0.930	0.954	0.915	0.976	0.573	0.530	0.616	0.902	0.864	0.930	0.954	0.915	0.976
IgG-tTG-HR–MIXED (one study)	0.677	0.556	0.778	1.000	0.839	1.000	0.518	0.413	0.621	0.677	0.556	0.778	1.000	0.839	1.000

H = significant heterogeneity by Pearson's Chi square
Note: see Appendix G for raw pooled data by antibody test

Figure 21: PPV and prevalence from individual studies

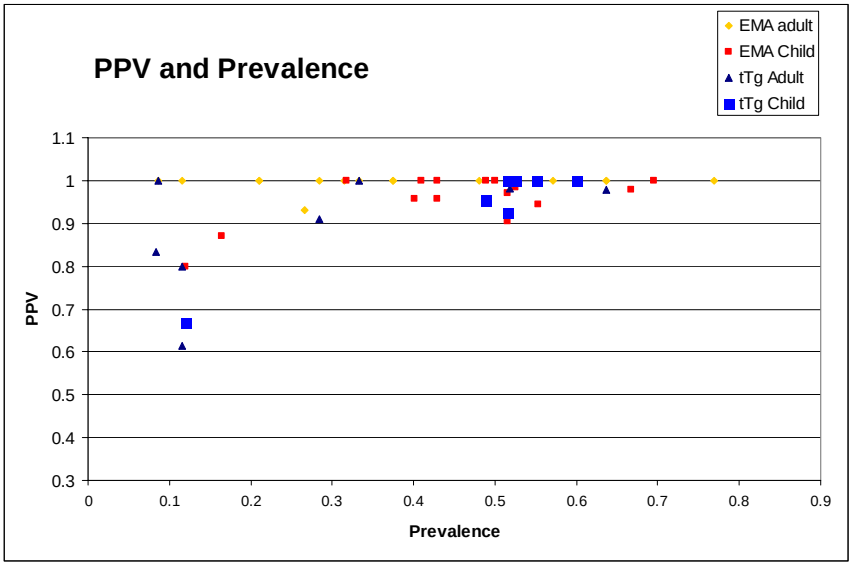


Figure 22: NPV and prevalence from individual studies

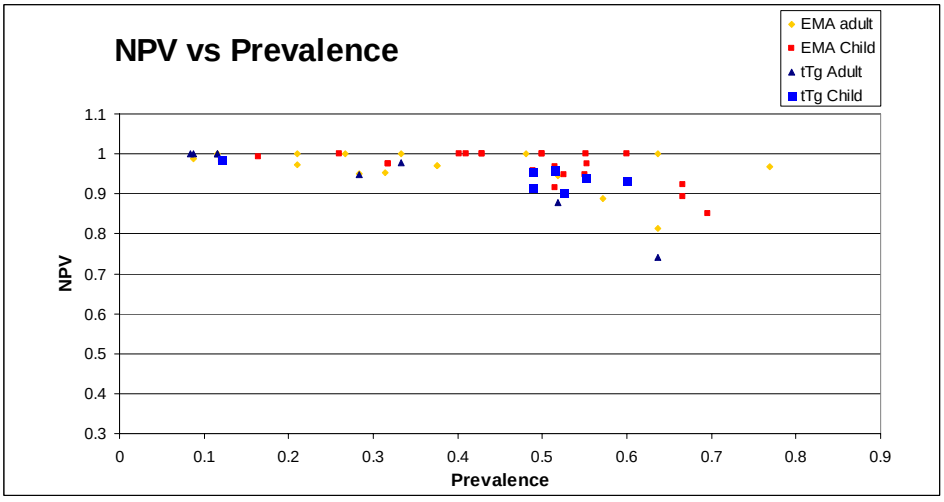
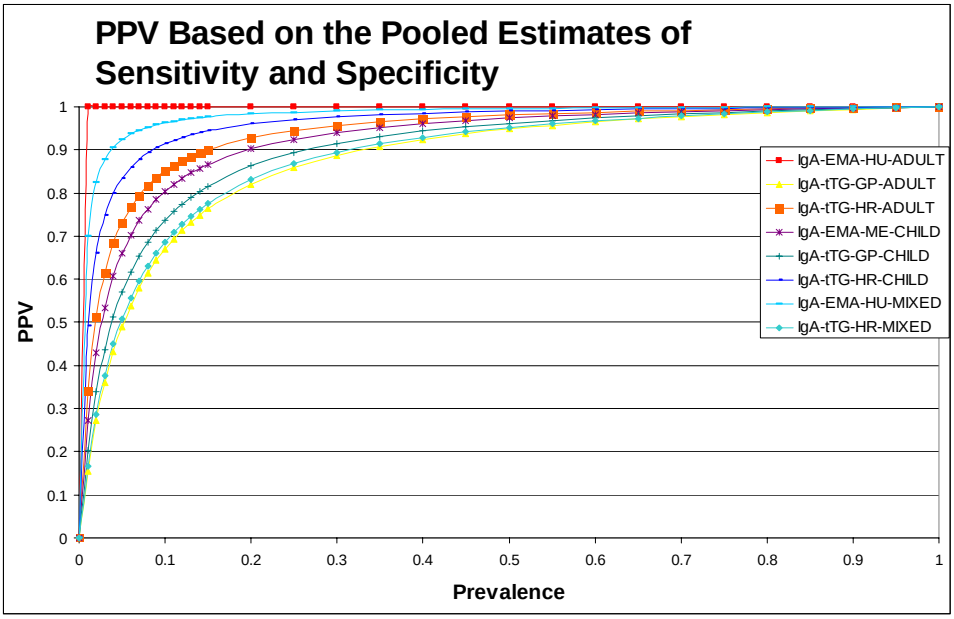


Figure 23: PPV based on the pooled estimates of sensitivity and specificity



HLA DQ2/DQ8

We identified 99 potentially relevant HLA articles that appeared to address HLA DQ2/DQ8 in a CD population (Appendix F).[8-11,15,53,54,62,91-100,100-177] These studies were not designed to determine the diagnostic utility of DQ2 or DQ8 per se.

Of the identified studies, 54 allowed estimation of the prevalence, sensitivity or specificity of HLA DQ2/DQ8 in the studied population.[8-10,15,53,54,93,100,109,120,134-177] In one study, DQ2 data could not be reliably extracted.[169] The authors of one study[9] explicitly stated that the patients used were the same as in two of their other publications.[8,93] In two other publications by the same authors,[9,10] the patients appear to be different and the authors do not indicate that they used patients from a previous study. However, the possibility that these two studies[9,10] share a subset of patients cannot be excluded. Another two studies addressing different topics but with extractable HLA data, also appeared to have used the same patients.[53,136] In cases of duplicate publications, the studies with the greater number of patients were used.[9,136]

The study designs and strictness of CD diagnosis in these articles varied, as did the inclusion of a control group. Most of the CD cases were diagnosed based on the ESPGAN criteria, although in some studies CD was diagnosed based on serology and then in most cases later confirmed by biopsy.[15,109,120,160,161,164,168,170,172,177] Nine of the studies were classified as cross-sectional studies,[169-177], 32 were case-control studies,[8-10,53,100,120,134-159] and 12 were mixed cross-sectional/case-control studies or could be considered as diagnostic cohort studies.[15,54,109,160-168] Four of the mixed design studies[109,164,166,178] used screen-negative patients as the control group, whereas the rest used a control group that was separate from the screened population. The study populations were also variable. The case-control studies used known CD cases compared with variously defined CD negative controls.

Seven studies used relatives of CD patients,[158,161,164,166,169,172,177] four used a population with Down Syndrome,[109,134,160,170] two used a population with type I diabetes,[165,173] and one used a mixed group of patients with CD including some with Down's and others with diabetes.[173] The mixed-design/cohort studies used patients suspected of CD on clinical grounds or subjects who belonged to a high-risk group, such as type 1 diabetics or first-degree relatives of patients with CD. The remaining articles used a screened healthy population or another specific group.

The articles with extractable data stated the frequency of HLA DQ2, and to a lesser extent the frequency of HLA DQ8, in their CD group. The cross-sectional studies did not include a control group. Only the frequency as a surrogate of sensitivity was available. None of the case-control or mixed-design studies calculated the sensitivity or specificity of HLA DQ2 or DQ8. However, these studies allowed us to derive estimates of these statistics from their results or tables. The considerable degree of clinical and methodological heterogeneity between the identified studies did no allow for statistical pooling of the results.

Two studies fulfilled our inclusion requirement of both cases and control groups undergoing intestinal biopsy (Evidence Table 2, Appendix I; Table 28).[136,152] The remaining studies had various control group types: unbiopsied, healthy controls, disease controls, or serology-negative controls. **These studies provide useful information and are presented at the end of the HLA results section for reference**.

The study by Iltanen et al.,[136] was conducted in a group of Finnish children to assess the density of gamma delta positive intraepithelial lymphocytes (γδ+ IELs) in: patients with CD by biopsy (ESPGAN); patients with suspected CD where the diagnosis was excluded by biopsy; and, in a group of biopsy-negative patients who underwent endoscopy for dyspepsia. The biopsy

aspect of this study is presented in its respective section. In this study, HLA DQ2 was found in 19 of 21 (90.5%) of patients with CD as apposed to 29 out of 67 (29.9%) of the control patients. Elevated γδ+ IEL density was significantly associated with DQ2 positivity. The calculated diagnostic measures for this study are presented in Table 28. In this population, DQ2 demonstrated a high sensitivity of 90.5% but a relatively modest specificity of only 70%, which is understandable given that the control population had a fairly high frequency of DQ2 positivity. The prevalence of CD in the study population was 1:4.2 (or 24%). The PPV was 49% and the NPV was 96%, suggesting that a negative DQ2 test result provides the greatest diagnostic information.

Sacchetti et al.[152] studied a group of Italian children suspected of having CD. Patients fulfilling the ESPGAN criteria were classified as having CD (n = 48 of 80), whereas, the remainder (n=32) were considered disease controls. The authors also used a second retrospectively defined group of known CD patients by ESPGAN criteria (n = 74), and a second group control of 180 unbiopsied healthy subjects. HLA DQ2 was determined in the CD group as a whole and in the two control groups, with the results presented in Table 28. In this study, the sensitivity of HLA DQ2 was 88.9% and the specificity was 81% for the comparison with the biopsied controls; the sensitivity of HLA DQ2 was 88.9% and the specificity was 73% for the comparison with the unbiopsied controls. Interestingly, in this study only 18.8% of the biopsy-negative controls were positive for HLA DQ2, whereas, 26.7% of the unbiopsied controls were HLA DQ2 positive. This difference accounts for the higher specificity seen for HLA DQ2 in the comparison with the biopsy-negative control group as compared with the comparison with the healthy controls. The prevalence of CD in the studied population was also quite high in both portions of this study (79% for comparison with biopsied controls and 51% for the comparison with unbiopsied controls). As such the PPV and the NPV of HLA DQ2 in this study were 95% and 62%, respectively. The difference in prevalence between this and the Iltanen study accounts for the differences seen in the PPVs and NPVs.

Table 28: HLA studies with biopsied cases and controls

Author, year; country	Prev of CD	DQ2 in CD	DQ2 in controls	Sensitivity	Specificity	PPV	NPV	CD population
Iltanen, 1999; Finland	0.24	90.48	29.85	90%	70%	49%	96%	Known CD versus biopsied controls
Sacchetti, 1998; Italy	0.79	86.89	18.75	87%	81%	95%	62%	Known CD versus biopsied controls
	0.51	86.89	26.72	87%	73%	77%	84%	Versus unbiopsied healthy controls

HLA all study data. The following section presents the data of the HLA studies that failed to be included on the basis that the control groups were not assessed with the gold standard test for CD (biopsy). These studies collectively provide useful information on the diagnostic value of HLA testing, but have to be interpreted with caution.

The prevalence of DQ2 and DQ8 in these studies is presented in Table 29, while the results of the diagnostic value of HLA DQ2 and HLA DQ8 are presented in Tables 30 and 31. Unfortunately, none of these studies were actual studies of the diagnostic value of HLA DQ2 or HLA DQ8 for the diagnosis or screening of CD. However, as presented in the Tables, the crude data was abstracted and the diagnostic characteristics were calculated. Significant clinical and statistical heterogeneity existed between these studies, making arithmetic pooling of the studies unjustified. Figure 24 and Figure 25 represent the plotting of each study's sensitivity (true positives) versus 1-specificity (false positives) to create a ROC presentation. The value of these figures lies in the global picture they represent regarding the results of each of the studies. Figure 24 demonstrates that the vast majority of the studies cluster together in a region where the sensitivity of HLA DQ2 is greater than 80%, with most studies lying above the 90% sensitivity mark. In contrast, these same studies have specificities in the range of 55% to 80%. Outlier studies are identified by author name. The best sensitivities and specificities were seen in two studies. The first, by Kaur et al.,[163] was a study from India where only 4.6% of the control population was positive for HLA DQ2. The second study, by Tighe et al.,[149] was conducted in a group of patients with CD and ethnically-matched control subjects from Rome, Italy. The prevalence of CD was quite high in the studied group (51%), and the frequency of HLA DQ2 in the control population of 12.2% was much lower than that observed in other Italian studies.

The remaining outlier studies were divided into a low-sensitivity/high-specificity group (Group 1), and a high-sensitivity/low-specificity group (Group 2). In the first case, all the studies were conducted in a non-Western European population. In particular, the worst performance of HLA DQ2 occurred in a study from Chile,[137] where the frequency of HLA DQ2 was very low in both the patients with CD and the control subjects. It is important to note, however, that not all non-Western populations deviated from the main cluster of studies. For example, Catassi et al.[120] found that 91% of Saharawi Arabs (Algeria) with CD carried HLA DQ2 compared with 38.9% of Saharawi controls. These values are similar to those seen in most Western populations. The second group all showed relatively poor specificity, although the sensitivity was preserved. As would be expected, the control groups of these studies were at high risk of having CD (relatives of CD[158,164,166]), or were a population with a known higher frequency of HLA DQ2 (individuals with diabetes[161,165]). As such, the high frequency of HLA DQ2 in these control populations makes the specificity of HLA DQ2 rather poor.

The frequency of HLA DQ8 in Western European populations with CD varies from approximately 2.7% to 6% (Table 29). The frequency is slightly higher in studies from Italy, the UK, and France (5.6% to 8% of CD patients). The frequency of HLA DQ8 in a subset of patients who had HLA testing in a large American serology screening study for CD was 22%,[15] which is quite a bit higher than that reported in the European studies.

A small group of studies allowed the estimation of the sensitivity and specificity of having either HLA DQ2 or DQ8. The results of these studies are presented in Table 33 and Figure 25. As can be seen in the figure, these studies confer a wide variation. Clearly, the sensitivity of using this strategy is quite high and is likely close to 100% in Western populations. The study by Balas et al.,[155] likely represents the closest to the truth, as this was a typical case-control design in patients with know CD compared with healthy controls. The Fasano et al. study[15]

represents the largest study and gives similar results to those obtained by Balas et al., however, the higher frequency of HLA DQ8 in their control group compared with other studies is of concern. Once again, the remaining studies can be grouped into high-specificity/low-sensitivity (Group 1) and high-sensitivity/low-specificity (Group 2). As was the case for HLA DQ2, Group 1 consists of two studies of non-Western populations, whereas, Group 2 represents studies with first-degree relatives and a study that used patients with diabetes as their control group.

Table 29: Prevalence/frequency of HLA DQ2 and HLA DQ8 in prevalence and mixed-design studies, and in case-control studies with HLA DQ8 data

Author	Year	Country	# of CD	% DQ2	% DQ8	% DQ2/8	Population with CD
Lewis	2000	USA	101	90.10	n/a	n/a	Confirmed cases among CD relatives
Book	2001	USA	8	87.50	12.50	100	Down Syndrome
Book	2003	USA	34	n/a	n/a	97.06	Affected 1st-degree relatives of CD sib. pairs
Csizmadia	2000	Netherlands	10	100	20	n/a	Down Syndrome
Fasano	2003	USA	98	83.67	22.45	100	Screened large population only subset tested for HLA
Iltamen	1999	Finland	5	100	n/a	n/a	Sjogren's syndrome
Kaukinen	2000	Finland	6	100	n/a	n/a	Known CD
Maki	2003	Finland	56	85.71	n/a	n/a	Screen of school-age children
Mustalahti	2002	Finland	29	100	n/a	n/a	Relatives of CD or DH
Catassi	2001	Algeria	79	91.3	n/a	95.6	Saharawi Arabs
Lui	2002	Finland	260	96.92	2.69	99.62	Family members of celiacs
Polvi	1996	Finland	45	100	n/a	n/a	Known CD
Ploski / Sollid	1996	Sweden	135	91.85	4.44	96.30	Known CD
Popat	2002	Sweden	62	93.55	n/a	n/a	Known CD
Larizza	2001	Italy	7	100	n/a	n/a	Children with autoimmune thyroid disease, EMA+biopsy
Failla	1996	Italy	7	14.29	n/a	n/a	Down Syndrome (only 7 CD cases)
Farre	1999	Spain	60	93.33	n/a	n/a	1st-degree relatives of celiacs
Balas	1997	Spain	212	94.81	4.25	99.06	Known CD
Zubillaga	2002	Spain	135	92.59	3.70	96.0 (calc)	Mostly CDs, some CD in subjects with Down Syndrome and subjects with diabetes
Karell	2003	France	92	86.96	6.52	93.48	Known CD
		Italy	302	93.71	5.63	89.40	
		Finland	100	91	5.00	96.00	
		Norway/ Sweden	326	91.41	5.21	96.63	
		Uk	188	87.77	7.98	95.74	
		Total	1008	93.71	5.95	93.95	
Kaur	2002	India	35	97.14	n/a	n/a	Known CD
Neuhausen	2002	Israel	23	82.61	56.52	100	Bedouin Arabs
Tuysuz	2001	Turkey	55	83.64	16.36	90.91	Children with known CD
Bouguerra	1996	Tunisia	94	84.04	n/a	n/a	Known CD
Sumnik	2000	Czech	15	80	66.67	100	Diabetics
Perez-Bravo	1999	Chile	62	11.29	25.81	37.10	Chileans

DH = dermatitis herpetiformis

Table 30: Sensitivity/specificity (calculated) for HLA DQ2 in case-control studies

Author, year; country	Prev of CD	% DQ2 in CD	% DQ2 in Controls	Sens	Spec	PPV	NPV	CD population
Fine, 2000; USA	0.06	88 (22/25)	31.24 (134/429)	0.88	0.69	0.14	0.99	Known CD
Howell, 1995; UK	0.38	91.21 (83/91)	23.18 (35/151)	0.91	0.77	0.7	0.94	Known CD
Michalski, 1995; Ireland	0.62	96.67 (87/90)	39.29 (22/56)	0.97	0.61	0.8	0.92	Known CD
Colonna, 1990; Italy	0.36	94.59 (140/148)	40.82 (109/267)	0.95	0.59	0.56	0.95	Known CD
Catassi, 2001; Algeria	0.37	91.1 (72/79)	38.9 (53/136)	0.91	0.61	0.58	0.92	Saharawi Arabs
Congia, 1991; Italy	0.2	96 (24/25)	34 (34/100)	0.96	0.66	0.41	0.99	Known CD
Ferrante, 1992; Italy	0.48	88 (44/50)	16.36 (9/55)	0.88	0.84	0.83	0.88	Known CD
Mazzilli, 1992; Italy	0.5	92 (46/50)	18 (9/50)	0.92	0.82	0.84	0.91	Known CD
Tighe, 1992; Italy	0.49	70.59 (39/43)	8.33 (5/41)	0.91	0.88	0.89	0.9	Known CD
Castro, 1993; Italy	0.38	80 (4/5)	37.5 (3/8)	0.8	0.63	0.57	0.83	Down Syndrome
Lio, 1997; Italy	0.45	100 (18/18)	63.64 (14/22)	1	0.36	0.56	1	Known CD
Sacchetti, 1998; Italy	0.79	86.89 (106/122)	18.75 (6/32)	0.87	0.81	0.95	0.62	Known CD and biopsied controls
Sacchetti, 1998; Italy	0.51	86.89 (106/122)	26.72 (31/116)	0.87	0.73	0.77	0.84	Healthy controls
Iltamen, 1999; Finland	0.24	90.48 (19/21)	29.85 (20/67)	0.9	0.7	0.49	0.96	Known CD
Ploski/Sollid, 1993; Sweden	0.34	94.68 (89/94)	25.97 (47/181)	0.95	0.74	0.65	0.96	Known CD
Pattersson, 1933; Sweden	0.4	92.31 (60/65)	43.75 (42/96)	0.92	0.56	0.59	0.92	Known CD
Ploski/Sollid, 1996; Sweden	0.43	91.85 (124/135)	22.35 (40/179)	0.92	0.78	0.76	0.93	CD vs blood donors
Fernandez-Arquero, 1995; Spain	0.36	92 (92/100)	25.56 (46/180)	0.92	0.74	0.67	0.94	Known CD
Arranz, 1997; Spain	0.5	92 (46/50)	24 (12/50)	0.92	0.76	0.79	0.9	Known CD
Balas, 1997; Spain	0.22	94.81 (201/212)	29.25 (217/742)	0.95	0.71	0.48	0.98	Known CD

Table 30 (cont'd): Sensitivity/specificity (calculated) for HLA DQ2 in case-control studies

Author, year; country	Prev of CD	% DQ2 in CD	% DQ2 in Controls	Sens	Spec	PPV	NPV	CD population
Ruiz Del Prado, 2001; Spain	0.04	94.74 (36/38)	39.22 (351/895)	0.95	0.61	0.09	1	Known CD
Dijilali-Saiah, 1994; France	0.27	88.75 (71/80)	21.13 (45/213)	0.89	0.79	0.61	0.95	Known CD
Dijilali-Saiah, 1998; France	0.44	83.17 (84/101)	20 (26/130)	0.83	0.8	0.76	0.86	Known CD
Tighe, 1993; Israel	0.51	90.7 (24/34)	12.2 (3/36)	0.71	0.92	0.89	0.77	Ashkenazi Jews, known CD
Arnason, 1994; Iceland	0.13	84 (21/25)	36.36 (60/165)	0.84	0.64	0.26	0.96	Known CD
Boy, 1994; Sardinia	0.5	96 (48/50)	32 (16/50)	0.96	0.68	0.75	0.94	Known CD
Congia, 1994; Sardinia	0.42	90.77 (59/65)	39.33 (35/89)	0.91	0.61	0.63	0.9	Known CD
Erkan, 1999; Turkey	0.5	40 (12/30)	6.67 (2/30)	0.4	0.93	0.86	0.61	Known CD
Tumer, 2000; Turkey	0.3	51.52 (17/33)	25.97 (20/77)	0.52	0.74	0.46	0.78	Turkish, known CD
Tuysuz, 2001; Turkey	0.52	83.64 (46/55)	24 (12/50)	0.84	0.76	0.79	0.81	Turkish, known CD
Perez-Bravo, 1999; Chile	0.33	11.29 (7/62)	2.42 (3/124)	0.11	0.98	0.7	0.69	Chilean

Table 31: Sensitivity/specificity (calculated) for HLA DQ2 in mixed-design studies

Author, year; country	Prev of CD	% DQ2 in CD	% DQ2 in controls	Sens	Spec	PPV	NPV	CD population
Book, 2001; USA	0.09	87.50 (7/8)	15.58 (12/77)	0.88	0.84	0.37	0.98	Down Syndrome
Csizmadia, 2000; Netherlands	0.11	100 (10/10)	28 (25/90)	1.00	0.72	0.29	1.00	Down Syndrome
Fasano, 2003; USA	0.52	83.67 (82/98)	42.39 (39/92)	0.84	0.58	0.68	0.77	9019 at risk, 4126 not at risk
Larizza, 2001; Italy	0.08	100 (7/7)	34.62 (27/78)	1	0.65	0.21	1	Children with autoimmune thyroid disease, EMA+biopsy
Polvi, 1996; Finland	0.58	100 (45/45)	28.13 (9/32)	1	0.72	0.83	1	CD vs various controls
Iltamen, 1999; Finland	0.15	100 (5/5)	n/a	1	n/a	n/a	n/a	Sjogren's syndrome
Kaukinen, 2000; Finland	0.17	100 (6/6)	n/a	1	n/a	n/a	n/a	CD vs disease controls
Lui, 2002; Finland	0.52	96.92 (252/260)	57.38 (136/237)	0.97	0.43	0.65	0.93	Family members of celiacs (controls=unaffected family members)
Farre, 1999; Spain	0.55	93.33 (56/60)	18 (9/50)	0.93	0.82	0.86	0.91	CD vs healthy controls
	0.26	93.33(56/60)	63.91(108/169)	0.93	0.36	0.34	0.94	CD vs relatives of CD
Sumnik, 2000; Czech	0.07	80 (12/15)	49.46 (92/186)	0.8	0.51	0.12	0.97	Diabetes (control=EMA neg.)
Kaur, 2002; India	0.11	97.14 (34/35)	4.64 (13/280)	0.97	0.95	0.72	1	CD vs healthy controls
Neuhausen, 2002; Israel	0.31	82.61 (19/23)	61.54 (32/52)	0.83	0.38	0.37	0.83	Bedouin Arabs (some cases and controls not biopsied)

Table 32: Sensitivity/specificity (calculated) for HLA DQ8

Author, year; country	Prev of CD	DQ8 in CD	DQ8 in controls	Sens	Spec	PPV	NPV	CD population
Csizmadia, 2000; Netherlands	0.11	20 (2/10)	20 (18/90)	0.20	0.80	0.10	0.90	Down Syndrome
Fasano, 2003; USA	0.52	22.45 (22/98)	20.65 (19/92)	0.22	0.79	0.54	0.49	Screened at-risk and not-at-risk populations
Lui, 2002; Finland	0.52	2.69 (7/260)	10.55 (25/237)	0.03	0.89	0.22	0.46	Family members of CD patients (controls=unaffected family members)
Ploski/Sollid 1996; Sweden	0.43	4.44 (6/135)	25.14 (45/179)	0.04	0.75	0.12	0.51	Known CD
Balas, 1997; Spain	0.22	4.25 (9/212)	16.85 (125/742)	0.04	0.83	0.07	0.75	Known CD
Sumnik, 2000; Czech	0.07	66.67 (10/15)	65.59 (122/186)	0.67	0.34	0.08	0.93	Diabetes
Neuhausen, 2002; Israel	0.31	56.52 (13/23)	25 (13/52)	0.57	0.75	0.5	0.8	Bedouin Arabs
Tuysuz, 2001; Turkey	0.52	16.36 (9/55)	8 (4/50)	0.16	0.92	0.69	0.5	Turkish known CD
Perez-Bravo, 1999; Chile	0.33	25.81 (16/62)	12.9 (16/124)	0.26	0.87	0.5	0.7	Chileans

Table 33: Sensitivity/specificity (calculated) for HLA DQ2 or DQ8

Author; year; country	Prev of CD	DQ2 or DQ8 in CD	DQ2 or DQ8 in controls	Sens	Spec	PPV	NPV	Notes
Fasano, 2003; USA	0.52	100 (98/98)	59.78 (55/92)	1	0.4	0.64	1	Screened at-risk and not-at-risk populations
Catassi, 2001; Algeria	0.37	96.2 (76/79)	41.9 (57/136)	0.96	0.58	0.57	0.96	Saharawi Arabs
Lui, 2002; Finland	0.52	99.62 (259/260)	67.93 (161/237)	1	0.32	0.62	0.99	Family members of CD (controls=unaffected family members)
Balas, 1997; Spain	0.22	99.06 (210/212)	46.09 (342/742)	0.99	0.54	0.38	1	Known CD
Sumnik, 2000; Czech	0.07	100 (15/15)	87.63 (163/186)	1	0.12	0.08	1	Diabetes
Tuysuz, 2001; Turkey	0.52	90.91 (50/55)	32 (16/50)	0.91	0.68	0.76	0.87	Turkish Known CD
Neuhausen, 2002; Israel	0.31	100 (23/23)	86.54 (45/52)	1	0.13	0.34	1	Bedouin Arabs
Perez-Bravo, 1999; Chile	0.33	37.1 (23/62)	15.32 (19/124)	0.37	0.85	0.55	0.73	Chileans

Figure 24: HLA DQ2

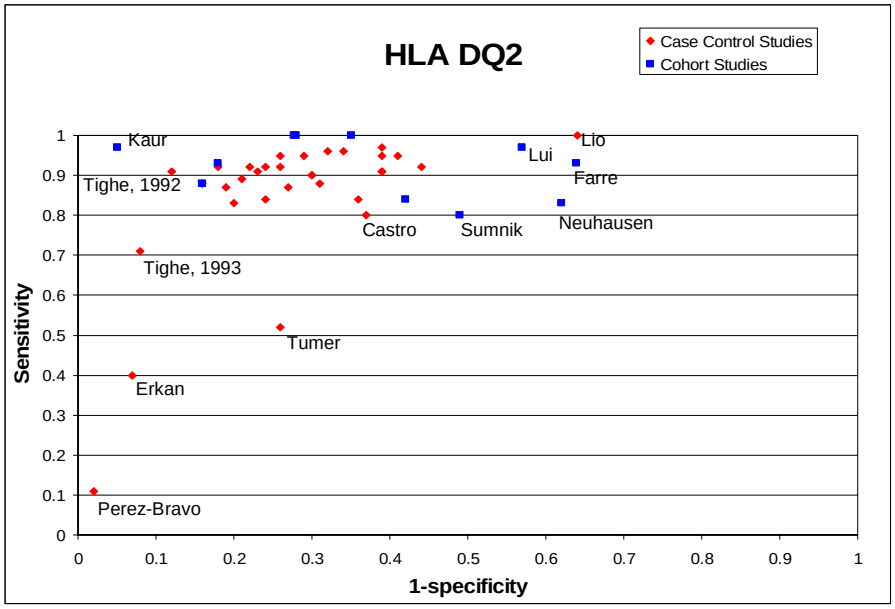
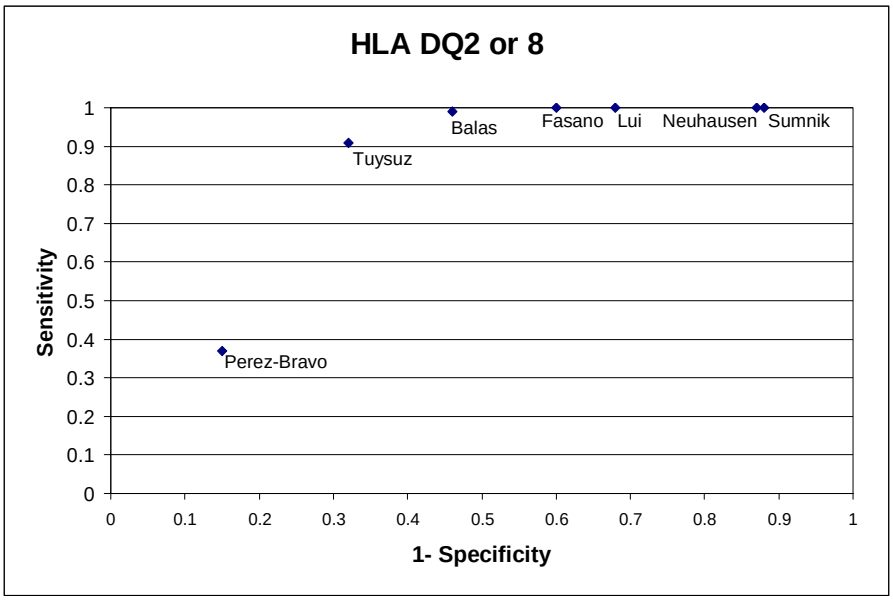


Figure 25: HLA DQ2 and DQ8



Biopsy

Using epidemiologically appropriate eligibility criteria, our comprehensive literature search did not identify any studies that specifically addressed the question of the sensitivity or specificity of biopsy for the diagnosis of CD.

However we sought to obtain indirect evidence regarding the diagnostic performance of biopsy as a test for CD. Some data was available from those studies identified for other review objectives, such as the cross-sectional screening studies, the HLA DQ2/8 studies, and studies of IELs. We also sought studies of follow-up of biopsy negative patients suspected of CD, and studies of silent and latent CD. The findings from these studies are presented in the Discussion and in Appendix H.

Quality Assessment

Overall, the quality of the diagnostic studies assessed in the Celiac 1 objective was quite good (Appendix J, Table 1). However, 59% of the studies reported using a selected patient population that may not be representative of a clinically relevant population. This is likely related to study design. Only 11% of the studies reported on whether the reference test was reported without knowledge of the index test. We felt that this was not a major threat to the validity of the studies.

Celiac 2: Incidence and Prevalence of CD

The literature search yielded 2,116 references (Appendix F). A first-level screen of the titles, abstracts and keywords, for articles that related to the incidence or prevalence of CD, excluded 1,506 references. Full-text versions of each of the 610 retained references were obtained and used for a second-level screen for articles, with a focus on the incidence and/or prevalence of CD. Review articles were also identified and kept for reference (n = 71). Three hundred and forty-eight out of the 610 references were excluded. The remaining 262 references were screened at a third level (Appendix F). Studies were included if they reported the prevalence and/or incidence of CD in the following groups: (1) general populations from North America or Western Europe; (2) first-degree relatives of patients with CD; (3) patients with type 1 diabetes; (4) patients being investigated for anemia; (5) patients with osteoporosis or osteopenia; (6) patients with suspected CD on the basis of their clinical presentations. We did not use any geographic restriction for the studies of populations at risk (first-degree relatives and type 1 diabetics) or of associated clinical presentations (suspected CD, anemia, or metabolic bone disease). Studies of prevalence or incidence that used AGA tests conducted prior to 1990 were excluded after discussion with the AHRQ because of potential problems with the reliability of older AGA assays. Reports which were not sufficiently explicit for data extraction also had to be excluded.[179-181]

We defined incidence studies as those studies that reported the total number of new cases of CD for a given territory and period, over a unit of population density. Therefore, studies of incidence where there was no population denominator were excluded. When multiple studies of incidence of CD were available for a similar country or geographic area, the most recent and/or most encompassing was selected. In general, we excluded the studies whose observation periods pertained exclusively to a period prior to 1990.

A total of 133 publications were selected. Of these, 14 publications were identified as duplicates on the basis that the same study population was reported on elsewhere, or as part of a larger cohort.[122,182-194] The remaining 119 original studies on prevalence and/or incidence of CD in the populations of interest were included and their data abstracted. Of these included studies, 42 assessed the prevalence and/or incidence of CD in a general population. Twelve of the 42 reported on the incidence of CD,[128,195-205] and 30 reported on the prevalence, either in the US (three studies[206-208]), Scandinavia (11 studies[209-219]), Italy and San Marino (seven studies[126,220-225]), UK (four studies[226-229]), or other countries (Spain[230], the Netherlands,[231,232] Switzerland,[233] and Germany[234]).

Studies of the prevalence of CD in populations at risk were divided as follows: 18 studies of the first-degree relatives of CD patients,[129,167,206,235-249] and 34 studies in patients with type 1 diabetes.[234,250-282]

Studies of the prevalence of CD in patients with associated clinical presentations were divided as follows: 12 studies in anemia and/or iron deficiency,[283-294] four studies in metabolic bone disease,[295-298] and 13 studies of patients with suspected CD on the basis of their clinical presentation.[206,238,299-309] The clinical manifestations that were included in the "suspected CD category" were: chronic diarrhea, weight loss, malabsorption or abdominal pain in adults and failure to thrive, short stature, malabsorption, chronic diarrhea, and abdominal pain in children. Four studies included groups at multiple-risk levels.[206,234,238,272]

Incidence of CD in the General Population

The incidence of CD in North America and Western Europe was derived from studies from the following countries: US,[128,205] England,[201] Italy,[202], Sicily,[203] Spain,[204] Netherlands,[200] Sweden,[195] Denmark,[196,197] and Finland (Evidence Table 3, Appendix I; Table 34).[198,199] In the report, crude incidence is defined as the number of new cases per 100,000 population-at-risk per year and cumulative incidence as the number of new cases per 1,000 live births; cumulative incidence is age-specific and its denominator reflects the total number of individuals from the same year of birth (i.e., birth cohort).

Table 34: Included studies of incidence of CD in the general population

Study	Country, period	Group at risk	Period related to results	Incidence	
				Crude incidence (# cases/100,000 patient year)	**Cumulative incidence** (# cases/1,000 births)
Ivarsson, 2003 Duplicate Ivarsson, 2000[193]	Sweden, 1973-97	Children	1997 (0-2 y)	51 (95% CI: 36-70)	Age 2 (1995): 1.7 (95% CI: 1.3-2.1)
			1996 (2-5 y)	33 (95% CI: 24-44)	
			1996 (5-15 y)	10 (95% CI: 7-13)	
Weile, 1993 Duplicate Weile, 1993[196]	Denmark, 1960-88	Children	1960-88		Age 5 (1988): 0.118
Maki, 1990 Duplicate ref[194]	Finland, 1960-84	Children	1974-83	3.46 (95% CI: n/r)	
Hawkes, 2000	England, 1981-95	Children	1991-95	2.15 (95% CI: n/r)	
Magazzu, 1994	Sicily 1975-89	Children	1989 birth cohort		Age 5 (1989): 1.16 95% CI: 0.92-1.42
Lopez-Rodriguez, 2003	Spain, 1981-99	Children 0-14 y	1981-90	6.87 (95% CI: 5.26-8.83)	
			1991-99	16.04 (95% CI: 12.99-19.59)	
		Children 0-4 y	1991-99	42.04 (95% CI: n/r)	
Hoffenberg, 2003	US (Denver, Colorado), 1993-99	Children	1993-99		Age 5 (1999): 9 (95% CI: 4-20)
Jansen, 1993	Netherlands 1990-92	All ages	1991-92	1.0 (95% CI: n/r)	
Corrao, 1995	Italy 1990-91	All ages	1990-91	2.13 (95% CI: n/r)	Age 5 (1991): 0.81
Talley, 1994	US 1960-90 Olmstead County	All ages	1960-90	1.2 (95% CI: 0.7-1.6)	
			1980-90	1.7 (95% CI: n/r)	
Bodé, 1996	Denmark, 1976-91	Adults	1976-91	1.27 (95% CI: n/r)	
Collin, 1997	Finland, 1975-94	Adults	1990-94	17.2 (95% CI: n/r)	
Hawkes, 2000	England, 1981-95	Adults	1991-95	3.08 (95% CI: n/r)	

Incidence in children: The crude incidence of CD in children age 0 to 15 years varied from 2.15 to 51 cases per 100,000 patient years.[193-195,198,201,204] When reported, the relative risk (RR) of CD was greatest for the 0- to 2-year age group, as well as for women, and varied from 32.26 to

42.4[193,195,204] and from 1.9 to 3.34,[128,193,195] respectively. The cumulative incidence at age 5, when reported, varied between 0.089 and 9 cases per 1,000 live births.[128,196,202,203] (see Table 34).

The incidence of CD has been most studied in the Scandinavian countries, particularly Sweden,[193,195,310-313] Denmark,[196,197,313,314] and Finland,[194,198,199] where important disparities have been observed over time and between countries. Reports from these countries have the advantage of being derived from comprehensive prospective databases and from populations which are genetically fairly stable, shedding light on potential environmental causal exposures,[195,196] or on variations in practice patterns.

In Scandinavia, the highest incidences of CD in children were found in Sweden for the 0- to 2-year age group from 1987 to 1997, where an average of 198 new cases per 100,000 patient years (95% CI: 186-210) were observed.[193,195] This peak in incidence was followed by a rapid decline, observed during 1995-97, where incidences dropped to an average of 51/100,000 patient years (95% CI: 36-70). In contrast, the incidence of CD in children aged 2 to 4.9 years and 5 to 15 years was only slightly increased over the 1973-97 period, with a peak in 1996 of 33 cases (95% CI: 24-44) per 100,000 patient years and 10 cases (95% CI: 7-13) per 100,000 patient years for these respective age groups. A cohort effect was noted in that the cumulative incidences at 2 years of age for the children belonging to birth cohorts from 1984 to 1994 were on the gradual rise (up to 4.4 cases/1,000 births [95% CI: 3.8-5.0] for the 1993 cohort), while a progressive decline was observed for birth cohorts from 1994 to 1996 (down to 1.7 cases [95% CI: 1.3-2.1] per 1,000 births for the 1995 cohort). Most of these cases were symptomatic, so that these observations are unlikely to be due to changes in screening practices. Interestingly, these changes mirrored changes in the composition of infant formulas, with the highest values of a wheat/rye/barley exposure index during the years 1982-1994.

In contrast, the incidence of CD in Denmark, a neighbouring country, has been significantly lower and very stable from 1960 to 1988,[196] with an average incidence of 0.089/1,000 live births for that period.[313] A comparison of dietary exposures between Swedish and Danish children diagnosed with CD between 1972 and 1989 showed that by the age of 8 months, the Swedish diet contained more than 40 times more gliadin than the Danish diet.[313] In Finland, incidences have also been fairly stable, and have in fact decreased among infants but increased among older children.[198] However, these observations date back to 1984 and can therefore not be compared with the Swedish epidemics.

Spain has also seen an increased incidence of CD over the past 25 years, from 6.87 (95% CI: 5.26-8.83) cases/100,000/year in 1981-90 to 16.04 cases/100,000/year (95% CI: 12.99-19.59) in 1991-99,[204] an observation that was correlated with an increased proportion of silent or atypical presentations at diagnosis (i.e., inferring a role for changes in clinical practice). The age at diagnosis also correlated positively with the age at which gluten was introduced in the diet.

The role of dietary exposure during infancy is also highlighted in studies from the UK, where recommendations on infant feeding, promoting breastfeeding and later introduction of starches, were published in 1974. Subsequent to these recommendations, there was a fall in the incidence of childhood CD;[315,316] however, this data is not presented in detail because we focused on reports from the past 15 years.

As opposed to the incidences derived from reported cases, the incidence observed from a prospective screening protocol are not subject to variations related to practice patterns and are obviously more comprehensive and accurate. Hoffenberg et al., from the US, conducted the only prospective CD screening study available to date.[128] Between December 1993 and September 1999, a total of 22,346 newborns in Denver, Colorado were screened for HLA genotypes

associated with CD and type 1 diabetes. A representative sample of at risk HLA DRB1*03 positive infants were prospectively followed (n=987), for as long as the first seven years of life. Serological screening was performed at nine, 15 and 24 months of age, then yearly. Small bowel biopsies were recommended if the serology (tTG in most cases) was positive on two separate occasions, or in the presence of clinical suspicion. Between 1993 and 1999, 19 children were found to have evidence of CD, ten children had biopsy-confirmed CD, whereas, nine children had a positive tTG result at least twice. The mean age at presentation of evidence of CD was 4.6 years (range 2.6-6.5). Compared with HLA-DR3-negative children, the RR for evidence of CD was 5.6 (1.5-21, p=0.009) and 9.1 (1.7-48, p=0.003), for those expressing one and two HLA-DR3 alleles, respectively. The RR of CD in females was 3.34 (1-10.9, p=0.048) times that of males. Cognisant of the prevalence of HLA-DR mono- and heterozygotes among the same birth cohort, the authors calculated that by the age of 5, the estimated cumulative incidence of CD in the general population (defined as either biopsy-proven CD or persistently elevated tTG) was 9/1000 births (95% CI: 4-20), or 1:104 (1:49 to 1:221). This remarkably high cumulative incidence (i.e., twice that of the highest value among Swedish children at 4 years of age – 5.0 [95% CI: 4.4-5.7][193]) has to be interpreted in light of the fact that only ten out of the 19 cases had been biopsied; the remaining nine cases were diagnosed on the basis of a persistently elevated tTG titre, the PPV of which the same authors reported to be only 70% to 83%.[317] However, as mentioned above, these results are derived from an actual prospective and systematic screening intervention for CD, where asymptomatic cases would be detected. In all likelihood, there is therefore an important proportion of CD cases who remain undiagnosed during early childhood.

Incidence in adults: The crude incidence of CD in adults varied from lows of 1.27 in Denmark[197] and 3.08 in England, [201] to a high of 17.2 cases per 100,000 patient years in Finland,[199] where specific efforts had been untaken to encourage screening for CD (see Table 34).

As has been observed for children, the incidence of CD in adults seems to have increased over the past 20 years.[199,201] This is largely explained by a change in practice patterns: physicians are more aware of the condition, its atypical manifestations and associated condition, while at the same time, serological testing has become widely available. There are therefore more diagnoses made on the basis of case-finding. This is reflected by the fact that the proportion of patients being diagnosed with CD in the absence of symptoms, or as a result of serological testing, has also increased.[199,201,318-320]

In Finland over the period 1975-94, Collin et al.[199] have observed a ten-fold rise in the incidence of CD. The authors attributed this to the use of serologic screening (physicians were actively told to screen patients with type I insulin-dependent diabetes (IDDM), autoimmune thyroid disease, connective tissue diseases, women with infertility, patients with neurologic symptoms and first-degree relatives of CD patients), the routine performance of intestinal biopsies on all patients undergoing gastroscopy, and to the opening of open-access endoscopy clinics, creating the ability of all general practitioners to refer patients for gastroscopy.

In Italy, a gradual increase in the number of annual new CD diagnoses was observed between 1968 and 1992;[318,320] this increase correlated with an increased proportion of patients with subclinical presentations being identified.[318,320] Interestingly, despite the changing clinical presentation, there was no statistical difference between the histological grades at diagnosis.[320]

The incidence of CD in individuals of all ages varies from 1.0 in the Netherlands[200] to 2.13 in Italy.[202] In Italy, the RR of CD in adults ranged from 0.11 in the >60 year group to 0.33 in the 16-39 year group, compared with children.[202] The RR of CD for females was 1.90 (95% CI: 1.48-2.45).[202]

In the US, the 30-year incidence (1960-90) for Olmstead County was 1.2 (95% CI: 0.7-1.6), and the incidence for 1980-90 was slightly higher at 1.7 (95% CI: not reported).[205] This observation contrasts with the cumulative incidence of 9/1000 by age 5 reported by Hoffenberg from Denver, Colorado;[128] clearly, further knowledge of the epidemiology of CD in the US is required.

The point prevalence of CD can be calculated from registers of CD cases and the size of the population at risk; we found reports of such an observation in three of the included incidence studies.[199,199,205] The point prevalence of CD was 21.8/100,000 in Olmstead County in 1991,[205] 2.7/100,000 (95% CI: 11.0-14.5) in the Netherlands in 1992,[200] and 204/100,000 (95% CI: 181-231) in Finland in 1994.[199] Of note, the later prevalence from Finland was observed in a community where intense efforts had been carried to screen the population at risk for CD.

Prevalence of CD in the General Population—Different Geographic and Racial/Ethnic Populations

Thirty-seven studies reported on the prevalence of CD in a general population (Evidence Table 4, Appendix I; Table 35). Three of these were conducted in the US,[206-208] 16 in the Scandinavian countries,[184-187,209-219,232]) eight in Italy,[126,182,183,220,221,223-225] five in the UK,[188,226-229] and five in other countries (Spain,[230] Republic of San Marino,[222] the Netherlands,[231] Switzerland[233] and Germany[234]). Several pairs of duplicate publications were identified including two triplets,[182-188,211,213,218,220,321] which brought the total number of included unique articles down to 30. The articles with the most complete data were used for the report.[126,206-234] Only seven studies were conducted in a child population,[206,209,215,220,221,223,232,233] but one large American study included separate data for both adults and children.[206] All the included studies were conducted between 1992 and 2003. A summary of the included study characteristics is presented in Table 35. A breakdown of the included studies by screening test and age group is provided in Table 36.

The prevalence of CD by serology in the general unselected populations of North America and Western Europe, ranged widely from 152 per 100,000 (0.152% or 1:658) to 2,670 per 100,000 (2.67% or 1:37). The prevalence by biopsy ranged from 152 per 100,000 (0.152% or 1:658) to 1,870 per 100,000 (1.87% or 1:53). In four of the studies, a large proportion of the serology-positive subjects did not undergo biopsy.[206,216,224,232]

Among the included studies, there was no clear pattern relating prevalence to study age group, or in a consistent way to country, with large numbers of studies clustering around a prevalence range of 0.0025 to 0.014 by serology and 0.0025 to 0.010 by biopsy (Table 35; Figure 26, 27). In fact for prevalence by serology, the 50th, 75th, and 80th percentiles occurred at a prevalence of 0.00637 (0.64%), 0.0117 (1.2%), and 0.0125 (1.3%), respectively, while by biopsy the 80th percentile was at a prevalence of 0.0074 (0.74%) (Table 37; Figure 26, 27). Categorizing the studies by screening test and age group reduced the variability somewhat, but significant between study variation persisted. There were not enough studies to divide an analysis by screening test, age group, and country, simultaneously.

Among the studies conducted in the US,[206-208] the prevalence ranged from 0.00312 (0.312% or 1:320—only child population in this group) to 0.00949 (0.949% or 1:105). The largest of these, by Fasano et al.,[322] found a prevalence of CD in "not at risk" populations to be 0.95% in adults, 0.31% in children, and 0.75% overall (0.0075 or 1:133). This study included a predominately Caucasian population, although other ethnic groups were included (94% white; 3% black; 1.5% hispanic; 1% asian; 0.5% other). Not et al.[208] found the prevalence by EMA confirmation of initial AGA testing to be 0.004 (0.4% or 1:250) in another predominately Caucasian population that also included other ethnic backgrounds (Caucasian [87%], African-American [11.5%], and Asian [1.5%]). Finally, Green et al.[207] found a prevalence of 0.005 (0.5% or 1:200) in 1,749 patients undergoing upper endoscopy. The reason for the initial endoscopy in this study was not clearly described, and only those patients with endoscopic features suggestive of CD were biopsied, which may have underestimated the true prevalence of CD. The prevalence of CD among the six Italian studies was similar to that seen in the American studies, showing a range from 0.2% to 0.86%.[126,221,223-225,230] The prevalence of CD in other countries is presented in Table 35.

Only four studies demonstrated a prevalence of CD of greater than 0.015 (1.5%) (UK,[323] Sweden,[209,219] Germany[234]), and an additional six showed a prevalence of between 0.010 (1.0%) and 0.015 (1.5%) (UK,[228] Sweden,[216] Netherlands,[232] Ireland,[229] Finland[214,215]). These studies would suggest a potentially higher prevalence of CD in these countries, though it should be kept in mind that other studies from these same countries showed a prevalence of less than 1.0%, including four studies from Sweden[211,213,216,217] (Figure 28). Only three of the eight studies conducted in a child population demonstrated a prevalence of CD of greater than 1.0% (Finland,[215] Sweden,[209] Netherlands[232]).

Among the 30 included studies, there was a considerable amount of variation in the point estimates for the prevalence of CD both by serology and by biopsy due to differences in serological test strategies, biopsy definitions and patient sampling, making pooled estimates unreliable. To further explore the potential sources of variability in the observed prevalence of CD, we plotted the studies' prevalence versus its sample size (Figure 29). This scatter diagram visually illustrates the distribution of the prevalence of CD among the included studies. The study with the highest reported prevalence of CD (2.67%), was also the one with the smallest sample size of 150 healthy patients, and also included several other at-risk groups, which were the primary focus of that study.[234] Overall, studies with the smallest sample sizes tended to produce both the highest and lowest prevalence of CD. Using an arbitrary cut-off of 1,600 patients to divide "small" and "large" sample size studies, the prevalence by serology ranged fairly evenly from 0.17% to 2.67% for the 13 small studies, while 12 of the 18 large studies were located within a range of 0.5% to 1.26% (one study did not provide prevalence by serology).

Table 35: Prevalence of CD by country

Author, year	Country	Age group	Test	Total patients	Prevalence by serology	Prevalence by biopsy	Notes
Fasano, 2003	USA	Adults	EMA - ME; all positive EMA tested with tTG-HU	2,845	0.00949		116/350 biopsied
Green, 2000	USA	Adults	EGD/biopsy	1,749		0.00515	Not all sytematically biopsied; only those with suggestive endoscopic features
Not, 1998	USA	Adults	IgG- and IgA-AGA - ELISA; confirmed with IgA-EMA ME or HU	2,000	0.00400		
Fasano, 2003	USA	Children		1,281	0.00312		
Johnston, 1998	UK	Adults	IgA-AGA, IgA-EMA	1,823	0.00823		
Sanders, 2003	UK	Adults	IgG- and IgA - ELISA; EMA-ME	1,200	0.01917	0.01000	22/23 biopsied
West, 2003	U.K.	Adults	IgA EMA-ME, IgA-tTGA	7,527	0.01156		
Rutz, 2002	Switzerland	Children	IgA-EMA-ME, IgA-tTG, IgG-AGA and IgA-AGA	1,450	0.00759	0.00690	10/11 biopsied
Borch, 2001	Sweden	Adults	Biopsy, IgA- and IgG-AGA; IgA-EMA-ME	482	0.01452	0.01867	
Grodzinsky, 1996	Sweden	Adults	IgA-AGA; IgA-EMA	1,866	0.00589	0.00375	Prevalence by IgA-EMA not reported
Ivarsson, 1999	Sweden	Adults	IgA- and IgG-AGA - ELISA, cut-off not recorded; IgA-EMA - ME; serum IgA level	1,894	0.00475	0.00475	
Sjoberg, 1994	Sweden	Adults	IgG- and IgA-AGA	1,537	0.01431	0.00065	13/22 biopsied
Sjoberg, 1999	Sweden	Adults	IgA-AGA, IgA confirmed with EMA-ME	1970	0.00152	0.00152	

EGD=esophagogastroduodenoscopy; IF=immunofluorescence; prevalence expressed as proportion (multiply by 100 for percent, or 100,000 for per 100,000 value)

Table 35 (cont'd): Prevalence of CD by country

Author, year	Country	Age group	Test	Total patients	Prevalence by serology	Prevalence by biopsy	Notes
Carlsson, 2001	Sweden	Children	AGA, EMA, biopsy using Watson capsule	690	0.01884	0.01594	
Riestra, 2000	Spain	Adults	IgG/IgA-AGA, IgA-EMA; the study was conducted as a 1) two-step protocol (determination of IgA/IgG-AGA, if positive measuring IgA-EMA); and a 2) one-step protocol (measuring IgA-EMA)	1,170	0.00171	0.00256	1 CD picked up when AGA and EMA was neg.
Corazza, 1997	Republic of San Marino	Adults	IgA-EMA; biopsy	559	0.00179	0.00179	
Hovdenak, 1999	Norway	Adults	IgA- and IgG-AGA; IgA-EMA	2,069	0.00387	0.00338	
Rostami, 1999	Netherlands	Adults	IgA-EMA	1,000	0.00300	0.00300	
Csizmadia, 1999	Netherlands	Children	IgA-EMA	6,127	0.01224	0.00506	57/75 biopsied
Pittschieler, 1996	Italy	Adults	IgA- and IgG-AGA; IgA-EMA; biopsy	4,615	0.00195	0.00195	38 of 140 biopsied
Trevisiol, 1999	Italy	Adults	IgA-EMA; biopsy	4,000	0.00250	0.00250	
Volta, 2001	Italy	Adults (mostly)	IgA-EMA-HU; biopsy	3,483	0.00574	0.00488	Prevalence of 0.57% (20/3483) if included 3 patients with normal villous but with increased IELs
Catassi, 2000	Italy	Children	IgG-AGA (7 AU); IgA-AGA (15 AU); IgA-EMA indirect IF (1:5 dilution); biopsy	2,096	0.00859		
Catassi, 1996	Italy	Children	IgA- or IgG-AGA; confirmed with EMA and biopsy	17,201	0.00645	0.00477	

EGD=esophagogastroduodenoscopy; IF=immunofluorescence; prevalence expressed as proportion (multiply by 100 for percent, or 100,000 for per 100,000 value)

Table 35 (cont'd): Prevalence of CD by country

Author, year	Country	Age group	Test	Total patients	Prevalence by serology	Prevalence by biopsy	Notes
Di Pietralata, 1992	Italy	Children	IgA-AGA; biopsy	3,022	0.00629	0.00596	
Dickey, 1992	Ireland	Adults	IgA AGA	443	0.01129		
Jager, 2001	Germany	Mixed - mostly adults	IgA-AGA, IgG-AGA, IgA-tTG -	150	0.02667		Mixed group of at-risk populations, healthy group used
Kolho, 1998	Finland	Adults	EMA -HU	1,070	0.01028	0.00748	
Maki, 2004	Finland	Children	IgA and IgG tTG; IgA and IgG EMA - IF; total serum IgA; HLA DR, DQ2 and DQ8	3,654	0.01259	0.00739	
Collin, 2002	Finland	Mixed - mostly adults	Biopsy	2,974	0.00605		
Weile, 2001	Denmark and Sweden	Adults	Serum IgA: IgG-AGA; IgA-AGA, cut-off >40 units; EMA; in cases of IgA <0.07g/L, IgG-AGA was analyzed	1,573	0.00254		
EGD=esophagogastroduodenoscopy; IF=immunofluorescence; prevalence expressed as proportion (multiply by 100 for percent, or 100,000 for per 100,000 value)							

Table 36: Prevalence of CD by serological screening test

Screening test	Age group	Number of studies	Total patients	Prevalence range
Primary biopsy	Adults	2[207,210]	4,723	0.00515 - 0.00605
IgA AGA	Overall	2[223,229]	3,465	0.00629 - 0.01129
	Adults	1[229]	443	0.01129
	Children	1[223]	3,022	0.00629
IgA / IgG AGA	Adults	1[216]	1,537	0.01431
IgA AGA - IGA EMA	Overall	6[208,209,211,217,219,226]	8,831	0.00152 - 0.01884
	Adults	5[208,209,211,217,219]	6,999	0.00152 - 0.01884
	Children	1[321]	1,823	0.00823
IgA/IgG AGA – IgA EMA	Overall	7[212,213,218,220,221,224,227]	30,648	0.00195 - 0.01917
	Adults	5[212,213,218,224,227]	11,351	0.00195 - 0.01917
	Children (Italy)	2[220,221]	19,297	0.00645 - 0.00859
IgA/IgG AGA – IgA tTG	Mostly adults (Germany)	1[234]	150	0.02667
IgA EMA	Overall	7[126,214,222,225,230-232]	17,409	0.00171 – 0.01224
	Adults	7[126,214,222,225,230,231]		0.00171 – 0.01028
	Children (Netherlands)	1[232]	6,127	0.01224
IgA EMA – IgG tTG	Overall	4[206,215,228,233]	16,757	0.00312 - 0.01259
	Adults (USA, UK)	2[206,228]	10,372	0.00949 - 0.01156
	Children	3 (includes Fasano Child Group)[206,215,233]	6,385	0.00312 - 0.01259
Note: Country of study was indicated when possible; prevalence expressed as proportion (multiply by 100 for percent, or 100,000 for per 100,000 value)				

Table 37: Prevalence of CD by statistical percentiles

Percentiles	Serology	Biopsy
5	.0016255	.0007378
10	.0018050	.0015761
25	.0030919	.0025321
50	.0063702	.0047672
60	.0084439	.0050768
75	.0117290	.0071429
80	.0125193	.0074416
90	.0184088	.0147536
95	.0225417	.0183992
100	.0266667	.0186722
Minimum	.00152	.00065
Maximum	.02667	.01867

Prevalence expressed as proportion (multiply by 100 for percent, or 100,000 for per 100,000 value)

Figure 26: Frequency distribution of prevalence of CD by serology among included studies

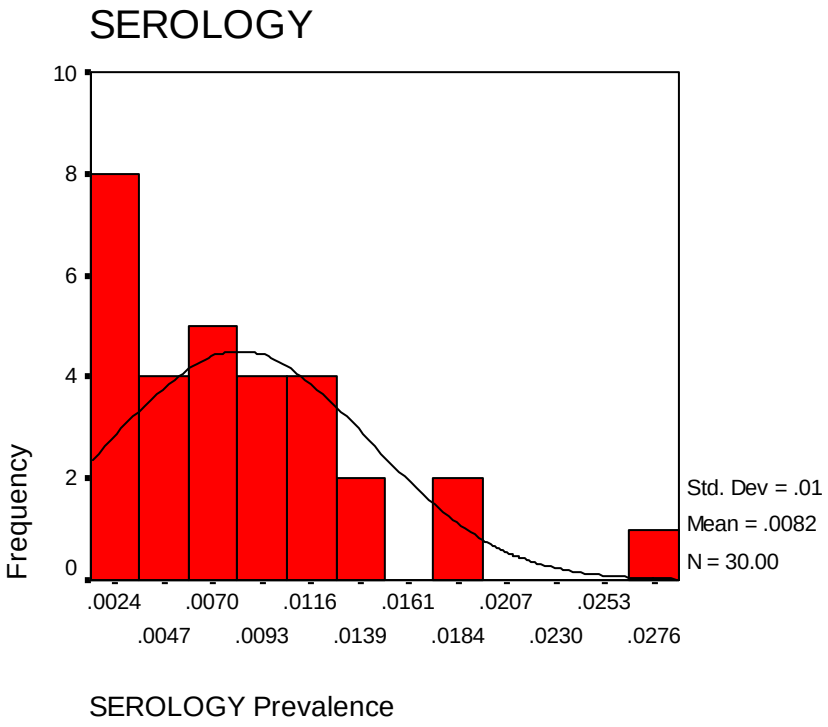


Figure 27: Frequency distribution of prevalence of CD by biopsy among included studies

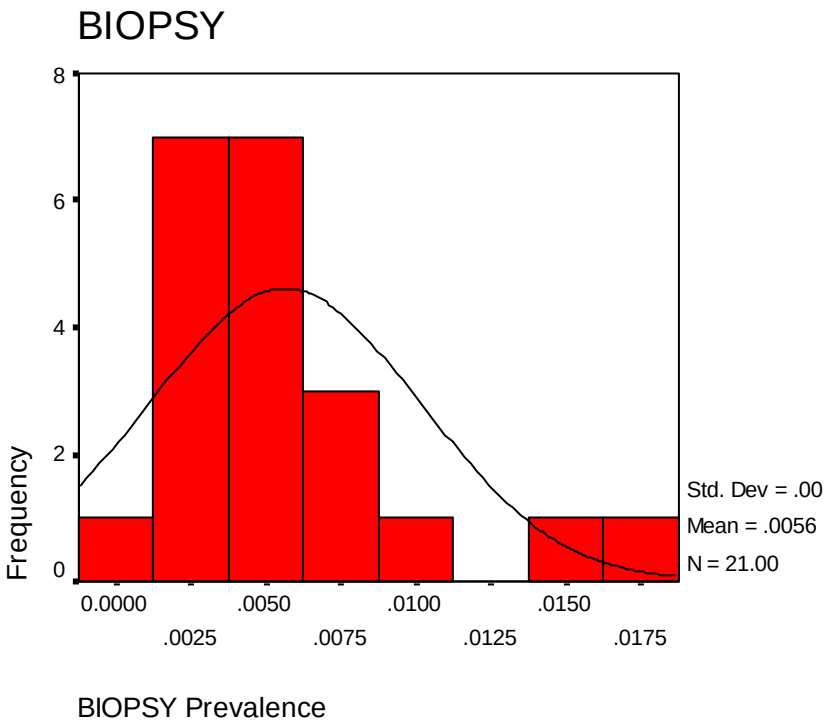


Figure 28: Prevalence of CD by country

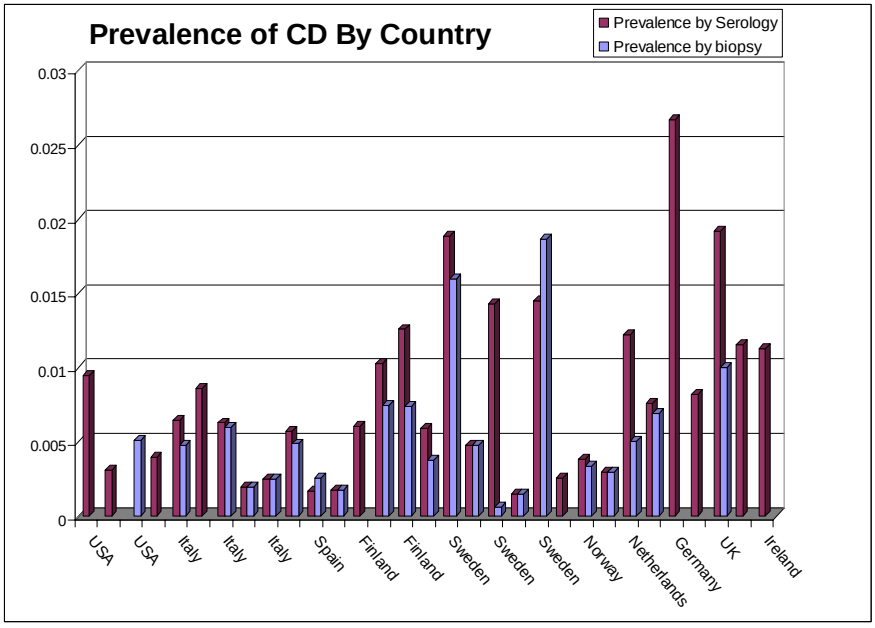
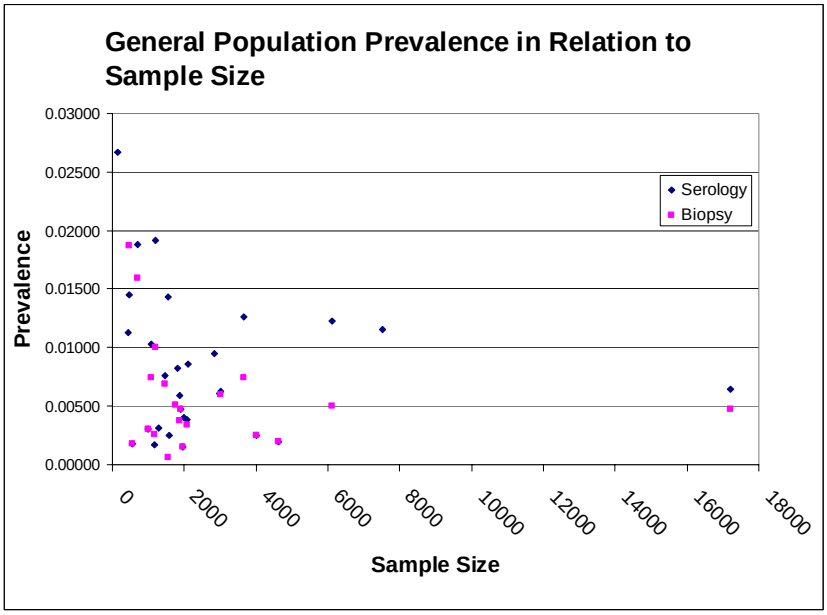


Figure 29: General population prevalence in relation to sample size



Prevalence of CD in Patients with Suspected CD

Adults: The prevalence of CD in adults suspected of the diagnosis was reported in four studies (Evidence Table 5, Appendix I; Table 38); three from Italy,[300,301,303] and one from the US.[206] The following reasons for suspecting a diagnosis of CD were documented: anemia, persistent iron deficiency, bowel disturbances, chronic intermittent diarrhea, abdominal pain, constipation, dyspepsia, severe malabsorption, tiredness and weight loss, mineral metabolism deficiencies, osteoporosis, arthralgias, arthritis, dermatitis, hypertransaminasemia, type I diabetes mellitus, infertility, and gluten intolerance in childhood not further investigated.

All three Italian studies were from referral centers, and intestinal biopsies were performed on all suspected cases, which cumulated to 347. The prevalence of CD was very high in these series, i.e., 43%,[300] 50%,[301], and 12%.[303]

In a large study of prevalence of CD in at-risk and not-at-risk individuals in the US, a total of 1,910 adults with CD-associated symptoms or disorders underwent serological testing with EMA. Fifteen of the 28 EMA-positive subjects (53.6%) consented to a biopsy, which was confirmatory in all cases.[206] The source of these patients and their mode of recruitment/referral were not reported. Based on the EMA result, the prevalence of CD in these adults with suspected CD was 1.5%.

Children: The prevalence of CD in children suspected of the diagnosis was reported in nine studies (Table 38); three from Canada,[304,305,307] two from the US,[206,324] and one each from Denmark,[302] England,[308] Italy,[309] and New Zealand.[306] The following reasons for suspecting a diagnosis of CD were documented: abdominal pain,[238,304,305,307,309] diarrhea,[238,304,305,308] failure to thrive/short stature,[206,238,304-306,309] weight loss,[305] vomiting,[304,305] abdominal distension,[304,305]

chronic GI symptoms,[306] inflammatory bowel disease,[304] family history of CD,[238,304,306,309] type I diabetes mellitus,[206,238,306] iron deficiency anemia (IDA),[309] thyroid disease,[304] trisomy 21,[206,304,309] as well as enamel hypoplasia, recurrent aphtous stomatitis, autoimmune diseases, IgA deficiency, and occult hypertransaminasemia.[309]

Five of the eight studies came out of referral centers where all suspected cases (cumulating to 978) were biopsied.[302,304-306,308] The prevalence of CD in these children ranged from 4.6%[306] to 17%.[305]

In a case-finding study among 26 family pediatricians in Italy, 240 children were screened with EMA based on the presence of risk factors, and 18 diagnoses of biopsy-proven CD were made, resulting in a prevalence of 7.5%.[309]

Three studies, two American[206,238] and one Canadian,[307] reported the prevalence of CD in children with related symptoms or conditions based on EMA testing. The cumulative number of children was 2,426, and the prevalence ranged from 1.1% in the Canadian study of children with chronic abdominal pain,[307] to 4.0% in the large American study of CD prevalence in at-risk and not-at-risk populations.[206]

All ages: Hin et al., performed a case-finding study through nine primary care clinics of central England that served a total population of 70,000 (Table 38).[299] A thousand patients were enrolled for serological screening, satisfying the following entry criteria: irritable bowel syndrome, anemia, family history of CD, malabsorption symptoms, diarrhea, fatigue, thyroid disease, diabetes mellitus, weight loss, short stature, failure to thrive, epilepsy, infertility, arthralgia, or eczema. The mean age of the screened subjects was 42.8 years; 5.3% were aged under 10, and 3.1% were aged 80 to 90 years. Thirty patients were EMA-positive, all of whom were confirmed by biopsy to have some enteropathy (90% had subtotal or total villous atrophy), and only one out of 30 patients had only IELs in the absence of villous atrohpy. The mean age of the 30 cases with CD was 42.8 years, and there was only one child diagnosed with CD. The prevalence of CD was 3.0%.

Table 38: Included studies for prevalence of CD in patients with suspected CD

Study, year; country	Clinical setting	Age group	Dx criteria	N tested	Prevalence (%)
Bardella, 1991; Italy	Referral center	Adults	Biopsy	60	43.3
Bardella, 2001; Italy	Referral center	Adults	Biopsy	80	50.0
Carrocio, 2002; Italy	Referral center	Adults	Biopsy	207	11.6
Fasano, 2003; USA	Not reported	Adults	EMA	1,910	1.5
Bode, 1993; Denmark	Referral center	Children	Biopsy	191	7.3
Day, 2000; New Zealand	Referral center	Children	Biopsy	153	4.6
Thomas, 1992; England	Referral center	Children	Biopsy	381	7.9
Chan, 2001; Canada	Referral center	Children	Biopsy	77	13.0
Chartrand, 1997; Canada	Referral center	Children	Biopsy	176	17.0
Ventura, 2001; Italy	Community pediatricians	Children	Biopsy	240	7.5
Fitzpatrick, 2001; Canada	Community pediatricians	Children	EMA	92	1.1
Fasano, 2003; USA	Not reported	Children	EMA	1,326	4.0
Hill, 2000; USA	Referral center	Children	EMA	1,008	2.5
Hin, 1999; England	Community practice	All ages	Biopsy	1,000	3.0

Prevalence of CD in with Type I Diabetes

The literature search identified 36 studies that assessed the prevalence of CD in patients with type I diabetes (insulin-dependent diabetes mellitus [IDDM]).[191,192,234,250-282] Two sets of duplicate publications were identified.[191,192,277,282] The publications with the most complete data sets were used.[277,282] Of the 34 unique studies (Evidence Table 6, Appendix I; Table 39), seven were conducted in an adult population,[257,263,266,270,273,277,279] 21 in a child population,[250-252,254-256,260-262,264,265,267,271,272,274-276,278,280-282] and six were conducted in a mixed population of adults and children.[234,253,258,259,268,269]

All the included studies initially screened the study population with one or more antibodies. Three studies did not confirm positive serology with biopsy,[265-267] whereas in nine studies confirmatory biopsies were performed in less than 75% of the screened-positive patients.[253,259,264,269,272,274,277-279] These studies were not included in the pooled estimates of the prevalence of CD by biopsy. All the studies that reported biopsy criteria used partial villous atrophy or greater to define CD.

For all the included studies, the minimum prevalence of CD in IDDM by serology was 1% and the maximum was 12%. By biopsy, the minimum and maximum prevalence was 1% and 11%, respectively. Within a given study, the prevalence by serology was almost uniformly greater than the prevalence by biopsy, as would be expected. Table 39 (individual studies) and Table 40 (pooled summaries) list the study details, the individual study estimates of CD prevalence and the pooled estimates of prevalence when appropriate.

The prevalence of CD in adults was assessed in seven studies.[257,263,266,270,273,277,279] Six of these studies used IgA EMA as the screening test,[257,263,266,270,273,279] whereas the largest study used IgA- and IgG-AGA, followed by EMA for confirmation.[277] In this last study, EMA confirmation was positive in 22 of the initially screened sample of 848 patients (2.6%), but biopsy confirmation was only performed in 14 of these patients, making the estimate of 0.83% prevalence by biopsy unreliable. The second largest study (n=509) did not confirm the EMA-positive patients with biopsy, and demonstrated the lowest prevalence of CD by EMA (1.4%) of all of the studies.[266] In another study of 185 patients,[279] the prevalence of CD by EMA was 4.9%, but only five of nine screen-positive patients were biopsied, making the prevalence of 2.2% (4/185) by biopsy a likely underestimation since four of the five biopsied EMA-positive patients were diagnosed with CD. A small study of 62 patients used biopsy as the screening test and found the prevalence of CD to be 11.3%, which is the highest prevalence of the group.[263] The remaining studies had uniform biopsy confirmation.[257,270,273] In these studies the prevalence of CD by EMA ranged from 3.1% to 7.9%, and the prevalence of CD by biopsy ranged from 2.6% to 6.4%.

Twenty-one studies assessed the prevalence of CD in children with IDDM.[250-252,254-256,260-262,264,265,267,271,272,274-276,278,280-282] Six of these studies used IgA-AGA or -AGA in combination with either IgG-AGA or other antibody tests.[254,256,267,271,274,276] The largest study tested 776 children with AGA and ARA (reticulin antibodies), and found a prevalence of CD by serology of 9.8%.[274] However, only 35 of 76 serology-positive patients were biopsied, making the reported prevalence by biopsy of 2.5% a likely underestimation. A single study of 459 patients that used IgA-AGA as the screening test found the prevalence of CD by serology to be 4.1%, and the prevalence of CD by uniform biopsy confirmation to be 4.6%.[276] The second largest study (n=498) used a combination of IgA- and IgG-AGA, and found a prevalence of CD by serology of 6.0% and a prevalence of CD by biopsy of 3.2%.[254] Two other studies that used IgA and IgG-AGA[271] or paired IgA-AGA measurements,[256] found a very similar prevalence by serology of 10.7% and 8.5%, respectively, and a prevalence by biopsy of 3.95% and 2.8%, respectively. The last study in this group did not perform biopsy confirmation of the IgA- and IgG-AGA derived prevalence of 3.76%.[267]

Seven studies used IgA-EMA to screen for CD in children with IDDM.[251,252,255,260,272,275,281] One Hungarian study of 205 children demonstrated a relatively high prevalence by serology and biopsy of 11.7% and 8.3%, respectively,[252] whereas an Austrian study of 403 children demonstrated a relatively low prevalence by serology and biopsy of 3.0% and 1.5%, respectively.[275] A study by Rossi et al.[272] from the US demonstrated a prevalence of CD of 4.7%. The remaining studies demonstrated fairly consistent results, with the prevalence of CD by serology ranging from 5.5% to 7.8%, and the prevalence by biopsy ranging from 3.3% to 6.5%.[251,255,260,281]

Three studies used IgA-tTG either alone[264] or in combination with IgG-tTG.[265,278] IgA-tTG was used alone in a study of 503 children which demonstrated a prevalence by serology of 4.4%. Ten of the 23 serology-positive patients did not undergo biopsy confirmation, making the

reported prevalence of 1.7% a likely underestimation. Of the two studies that used IgA- and IgG-tTG, the first did not perform biopsy confirmation and reported a prevalence of CD by serology of 8.4%,[265] whereas, the other found a prevalence of CD by serology of 6.3%, and by biopsy of 2.9%, although only eight of 13 serology-positive patients underwent biopsy.[278]

Five studies used a combination of IgA-EMA and one or more other antibodies, to assess the prevalence of CD in children with IDDM.[250,261,262,280,282] In three studies, EMA was combined with AGA,[250,261,280] in one it was combined with tTG,[262] and in the one it was combined with AGA and tTG.[282] In one study, only the confirmed biopsy prevalence of 8.3% was reported.[280] Overall, this group reported prevalences by serology ranging from 5.0% to 9.6%, and by biopsy ranging from 3.7% to 8.6%.

The remaining six studies assessed the prevalence of CD in a mixed-age population of patients with IDDM.[234,253,258,259,268,269] One study of 1,785 patients found the prevalence of CD by IgA AGA to be 4.1%. In this study, only 49 of 73 screen-positive patients underwent biopsy confirmation, making the reported prevalence by biopsy of 0.73% an underestimation.[269] Another large study of 1,114 patients used IgA and IgG AGA as an initial screen of screen-positive patients, and then performed a second level screen with IgA EMA before moving on to biopsy.[259] The EMA confirmed prevalence of CD was 4.9%, whereas, the reported biopsy confirmed prevalence was a relatively high 5.7%. In this study, 78 of 121 initial AGA-positive patients underwent biopsy, suggesting that most of the EMA-positive patients were biopsied.

Among the two studies that used IgA EMA as the screening test in a mixed-age population, the prevalence of CD by serology was 2.3%[258] and 5.7%.[268] It was unclear in the first study how the final confirmed prevalence of CD of 0.75% was arrived at,[258] whereas, in the other study the uniformly confirmed biopsy prevalence was 5.7%.[268]

The final two studies assessed the prevalence of CD in a mixed-age population of diabetics using IgA-tTG.[234,253] The prevalence of CD by serology was fairly high in both these studies: 9.6%[234] and 11.5%.[253] The first study did not perform biopsy confirmation, whereas, in the last study only 20 of 98 screen-positive patients were biopsied, making the reported prevalence of CD by biopsy of 1.8% a likely underestimation.

Clinical heterogeneity existed for some subgroups of this analysis making an overall pooled estimate of the prevalence of CD in children and adults with IDDM not entirely possible. However, a summary table (Table 40) is provided which presents the data grouped by age group and screening test, and Figure 30 presents the prevalence of CD in diabetes by study size. For similar studies a weighted pooled prevalence is provided, and individual study data with annotation is presented for studies that could not be pooled.

Table 39: Included studies of prevalence of CD in type I diabetes

Author, year; country	Total patients	Age group	Screening test(s)	First serology	Confirmatory serology	Biopsy proven	Biopsy criteria & description	Prevalence by serology	Prevalence by biopsy	Notes
Li Voon Chong, 2002; UK	509	Adults	EMA	7	None	n/a	None done	0.0138	n/a	
Talal, 1997; USA	185	Adults	EMA	9	None	4	ESPGAN	0.0486	0.0216	Only 5/9 biopsied
Rossi, 1993	211	Children, some adults	EMA	10	None	3	ESPGAN	0.0474	0.0142	Only 3/10 biopsied
Kaukinen, 1999; Finland	62	Adults	EMA		None	7	ESPGAN	0.0000	0.1129	
Sjoberg, 1998; Germany	848	Adults	AGA - IgG or IgA; EMA	258	22	7	Marsh	0.0259	0.0083	Only 14/22 biopsied
Sategna-Guidetti, 1994; Italy	383	Adults	EMA	12	None	10	Roy-Choudhury	0.0313	0.0261	10/12 biopsied
Rensch, 1996; USA	47	Adults	EMA	3	None	3	Loss of villous architecture, crypt hyperplasia, and increased IELs	0.0638	0.0638	
Frazer-Reynolds, 1998; Canada	263	Children	EMA	17	None	12	Carey capsule; Marsh criteria;	0.0646	0.0456	17/19 biopsied
Gillett, 2001; Canada	233	Children	EMA or AGA	19	None	14	Not reported	0.0815	0.0601	18/19 biopsied
Hansen, 2001; Denmark	104	Children	EMA or tTG	10	None	9	Partial or total villous atrophy, crypt hyperplasia and IEL infiltration	0.0962	0.0865	9/10 biopsied
Saukkonen, 1996; Finland	776	Children	AGA or ARA	76	None	19	Not reported	0.0979	0.0245	Only 35/76 biopsied
Spiekerkoetter, 2002; Germany	205	Children	tTG IgA or IgG	13	None	6	Marsh	0.0634	0.0293	Only 8/13 biopsied

Table 39 (cont'd): Included studies of prevalence of CD in type I diabetes

Author, year; country	Total patients	Age group	Screening test(s)	First serology	Confirmatory serology	Biopsy proven	Biopsy criteria & description	Prevalence by serology	Prevalence by biopsy	Notes
Arato, 2003; Hungary	205	Children	EMA	24	None	17	n/r	0.1171	0.0829	
Barera,1991; Italy	498	Children	AGA IgA then if neg IgG AGA	30	None	16	Subtotal villous atrophy	0.0602	0.0321	22/30 biopsied
Barera, 2002; Italy	273	Children	EMA, second EMA	15	10	9	Marsh; type II or III lesion	0.0549	0.0330	
Valerio, 2002; Italy	383	Children	EMA or IgG AGA	n/r	None	32	ESPGAN	n/r	0.0836	
Carelo, 1996; Spain	141	Children	IgA AGA if positive on two occaions	12	None	4	Subtotal villous atrophy	0.0851	0.0284	
Roldan, 1998; Spain	177	Children	IgA, IgG AGA, (and known cases, and some tested with EMA)	19	None	7	ESPGAN	0.1073	0.0395	Mixed group diagnosed by different means
Juan, 1998; Spain	93	Children	EMA	7	None	6	ESPGAN	0.0753	0.0645	
Sigurs, 1993; Sweden	459	Children	AGA	19	None	21	Watson Capsule	0.0414	0.0458	18/19 biopsied included known CD
Agardh, 2001; Sweden	162	Children	AGA, EMA, or tTG IgG or IgA	8	8	6	As described by Carlsson et al. 1999, Pediatrics 103:1248	0.0494	0.0370	Only 6 of 8 biopsied
Acerini, 1998; UK	167	Children	EMA or AGA	11	None	8	ESPGAN	0.0659	0.0479	9/11 biopsied
De Block, 2001; Belgium	399	Mixed	EMA	9	None	3	No biopsy performed	0.0226	0.0075	Unclear how the 3 cases confirmed

Table 39 (cont'd): Included studies of prevalence of CD in type I diabetes

Author, year; country	Total patients	Age group	Screening test(s)	First serology	Confirmatory serology	Biopsy proven	Biopsy criteria & description	Prevalence by serology	Prevalence by biopsy	Notes
Jager, 2001	197	Mixed	tTG	19	None		n/r	0.0964		
De Vitis, 1996; Italy	1114	Mixed	IgA, IgG then IgA EMA	121	55.00	63	Marsh - "villous atrophy"	0.1086	0.0566	78/121 biopsied
Not, 2001; Italy	491	Mixed	EMA	28	None	28	Intestinal biopsy; Marsh's modified classification	0.0570	0.0570	
Bao, 1999; USA	847	Mixed	tTG	98	None	15	n/r	0.1157	0.0177	Only 20/98 biopsied
Kordonouri, 2000; Germany	520	Mixed - mostly children	tTG	23	None	9	Marsh criteria	0.0442	0.0173	10/23 not biopsied
Aktay, 2001; USA	218	Mixed - mostly children	EMA	17	None	10	Partial or total villous atrophy, inflammation in lamina propria with increased IELs, and hyperplasia of crypts; classified as partial or total villous atrophy	0.0780	0.0459	14/17 biopsied
Cronin, 1997; Ireland	101	Mixed - mostly adults	EMA	8	None	5	n/r	0.0792	0.0495	
Schober, 2000; Austria	403	Mixed - mostly children	EMA	12	None	6	Modified Marsh and Crowe; Watson-type capsule	0.0298	0.0149	11/12 biopsied
Lampasona, 1999; Italy	287	Mixed - mostly children	tTG IgA or IgG	24	None	n/a	No biopsy	0.0836	n/a	

Table 39 (cont'd): Included studies of prevalence of CD in type I diabetes

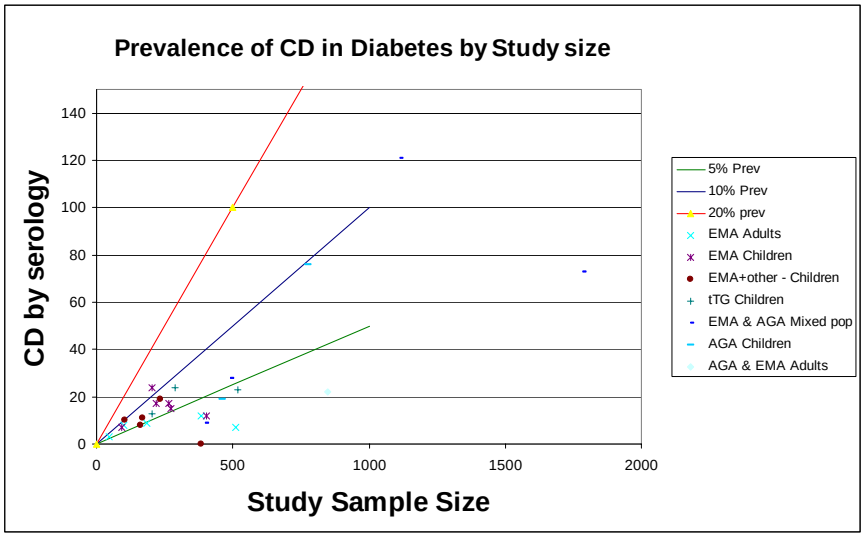
Author, year; country	Total patients	Age group	Screening test(s)	First serology	Confirmatory serology	Biopsy proven	Biopsy criteria & description	Prevalence by serology	Prevalence by biopsy	Notes
Lorini, 1996; Italy	133	Mixed - mostly children	AGA IgA or IgG	5	None	n/a	No biopsy	0.0376	n/a	
Page, 1994; Mixed	1785	n/a	AGA	73	None	13	n/a	0.0409	0.0073	Only 49/73 biopsied

Table 40: Summary of prevalence of CD in type I diabetes by age groups and screening test

Number of studies	Total patients	Age group	Screening test(s)	Prevalence by serology	Prevalence by biopsy
1[277]	848	Adults	AGA - IgG or IgA; then EMA	0.0259	0.0083*
1[266]	509	Adults	EMA	0.0138	n/a
1[279]	185	Adults	EMA	0.0486	0.0216*
1[263]	62	Adults	EMA	n/a	0.1129
3[257,270,273]	531	Adults	EMA	0.0433	0.0339
1[274]	776	Children	AGA or ARA	0.0979	0.0245*
1[276]	459	Children	AGA	0.0414	0.0458
4[254,256,267,271]	949	Children	AGA – various combinations	0.0695	0.0331
1[252]	205	Children	EMA	0.1171	0.0829
1[275]	403	Children	EMA	0.0298	0.0149
5[251,255,260,272,281]	1058	Children	EMA	0.0624	0.0437
4[251,255,260,281]	847	Children	EMA	0.0661	0.0437
5[250,261,262,280,282]	1049	Children	EMA - combinations	0.0721	0.0658
1[265]	287	Children	tTG IgA with IgG	0.0836	n/a
1[278]	205	Children	tTG IgA with IgG	0.0634	0.0293*
1[264]	520	Children	tTG	0.0442	0.0173*
1[269]	1785	Mixed	AGA	0.0409	0.0073*
1[259]	1114	Mixed	IgA, IgG-AGA then IgA-EMA	0.0494	0.0566*
1[268]	491	Mixed	EMA	0.0570	0.0570
1[258]	399	Mixed	EMA	0.0226	0.0075[†]
1[234]	197	Mixed	tTG	0.0964	n/a
1[253]	847	Mixed	tTG	0.1157	0.0177*

*large proportion of serology-positive patients not biopsied,[253,259,264,269,272,274,277-279] these were not included in the pooled analysis of prevalence by biopsy
**no description of how diagnosis made – result not pooled

Figure 30: Prevalence of CD in diabetes by study size



Prevalence of CD in Relatives of Patients with CD

There were 18 studies on the risk of CD in first-degree relatives of patients with biopsy-proven CD,[129,167,206,235-249], four of which also provided data on the risk of CD in second-degree relatives (Evidence Table 7, Appendix I; Table 41).[206,235,238,239]

First-degree relatives: First-degree relatives were directly evaluated with small bowel biopsy in five studies; three were performed in England in the 1970's,[242,243,245] and two in Finland during the 1990's.[129,167] The biopsy criteria for a diagnosis of CD was not reported in one study,[243] and implied at least some degree of villous atrophy in the other four.[129,167,242,325] The percent of all at-risk family members that were studied varied from 34%[245] to 100%.[243] The study size varied between 29[242] and 182,[245] and the cumulative number of patients tested was 494. The prevalence of CD among first-degree relatives undergoing intestinal biopsy varied from 5.5%[243] to 22.5%;[245] the pooled prevalence was 16%.

Serological screening of the first-degree relatives of patients with biopsy-proven CD was performed in 12 studies.[206,235-237,239-241,244,246-249] In seven of those studies, intestinal biopsy was performed on at least 80% of the subjects who tested positive serologically, i.e., in 84 % of subjects in one study,[237] and in 100% of subjects in the other six studies.[236,239,244,247-249] Serological screening was performed with AGA alone in one study,[236] whereas, the other six studies used EMA, either alone[239] or in combination.[237,244,247-249] Six studies used criteria implying some degree of villous atrophy,[236,237,239,244,247,248] whereas, one study included cases with Marsh I changes.[249] The study size varied from 92[248] to 943[239] subjects, for a cumulative number of 2,607 subjects. For the studies that required some degree of villous atrophy for diagnosis, the prevalence varied from 4%[236] to 12%,[248] and the mean prevalence was 7.6%.

However, when Marsh I lesions were also considered diagnostic, the prevalence of CD among first-degree relatives was reported at 44.1%.[249]

In five other studies of first-degree relatives,[206,235,240,241,246] confirmatory biopsy was not routinely performed (available in 9%[246] to 58%[241] of the cases), and the reported prevalence of CD was based on the serology results. EMA was used for serological screening in all of these studies, either alone,[206,240] or in combination with AGA [241,246] or tTG.[235]

Two of these studies were performed in families where at least two index cases prevailed and are, therefore, reviewed separately.[235,241] Ninety percent of the at-risk populations from these two studies were tested, which represents a cumulative number of 629 subjects. The prevalence of CD among these first-degree relatives from families where there are at least two index cases of known CD or dermatitis herpetiformis (DH) was 9.4%[241] and 17.2%.[235]

The study size of the other three studies varied from 115[240] to 4,508,[206] and the cumulative number of first-degree relatives tested was 5,265. The prevalence of CD among these serology-tested first-degree relatives varied between 2.8%[246] and 4.5%[206] (mean prevalence 4.3%).

Other relatives: One study from the US[238] reported an EMA-based prevalence of 4.7% in 192 first- and second-degree relatives; the prevalence from each of the groups of relatives was not reported separately.

An American study by Book et al.[235] studied the prevalence of CD in second-degree relatives and first cousins of CD sibling pairs (i.e., families with two affected index cases). Eighty-two second-degree relatives and 47 first cousins were tested with EMA and tTG, and the diagnosis was biopsy confirmed in 40% of the cases. The serology-based prevalence was 19.5% in second-degree relatives and 17.0% in first cousins.

Two other studies, one large (n=1,275) American study of prevalence of CD in at-risk and not-at-risk subjects,[206] and one Hungarian study,[239] provided data on the prevalence of CD in second-degree relatives. The EMA-based prevalence of CD in those groups was 2.6% and 5.5%, respectively (mean prevalence 2.7% on a cumulative number of 1,329 second-degree relatives).

Table 41: Prevalence of CD in relatives of CD patients

Study, year; country	Relative Type	Index case	Screening	Dx criteria	N tested	Prevalence (%)
Polvi, 1996; Finland	1st degree	CD in family	Biopsy	ESPGAN	90	20
Holm, 1993; Finland	1st degree	CD in family	Biopsy	Some VA	121	10.7
Robinson, 1971; England	1st degree	CD child in family	Biopsy	Some VA	29	10.3
Rolles, 1974; England	1st degree	CD child in family	Biopsy	Not reported	72	5.6
Stokes, 1976; England	1st degree	CD in family	Biopsy	Some VA	182	22.5
Tursi, 2003; Italy	1st degree	CD in family	Biopsy	Marsh I-IV	111	44.1
Corazza, 1992; Italy	1st degree	CD adult in family	AGA	Some VA	328	4.0
Pittschieler, 2003; Italy	1st degree	CD in family	EMA, TTG	Some VA	92	12.0
Rostami, 2000; Netherlands	1st degree	CD in family	AGA, EMA, Hx	ESPGAN	338	10.9
Hogberg, 2003; Sweden	1st degree	CD in family	AGA, EMA, TTG	Some VA	120	8.3
Korponay-Szabo, 1998; Hungary	1st degree	CD in family	EMA	Some VA	943	9.1
Farre, 1999; Spain	1st degree	CD in family	AGA, EMA	Some VA	675	5.6
Kotze, 2001; Brazil	1st degree	CD in family	EMA	+ve serology*	115	3.5
Fasano, 2003; US	1st degree	CD in family	EMA	+ve serology	4,508	4.5
Vitoria, 1994; Spain	1st degree	CD in family	AGA, EMA	+ve serology	642	2.8
Mustalahti, 2002; Finland	1st degree	>1 DH or CD sib	AGA, EMA	+ve serology	466	9.4
Book, 2003; US	1st degree	CD sib pairs	EMA, TTG	+ve serology	163	17.2
Hill, 2000; US	1st & 2nd degree	CD in family	EMA	+ve serology	192	4.7
Fasano, 2003; US	2nd degree	CD in family	EMA	+ve serology	1,275	2.6
Korponay-Szabo, 1998; Hungary	2nd degree	CD in family	EMA	+ve serology	54	5.6
Book, 2003; US	2nd degree	CD sib pairs	EMA, TTG	+ve serology	82	19.5
Book, 2003; US	1st cousins	CD sib pairs	EMA, TTG	+ve serology	47	17.0

*EMA titre $\geq$ = 1/5
VA = villous atrophy; DH = dermatitis herpetiformis

Prevalence of CD in Patients with IDA

Twelve studies were identified that allowed for the extraction of the prevalence of CD among patients who were evaluated for anemia (Evidence Table 8, Appendix I; Table 42).[283-294] In all of these, IDA was the primary focus of the study or made up the cause of anemia in the majority of the study patients. Tables 42 and 43 summarize the characteristics of the included studies.

Three studies assessed the prevalence of CD in IDA patients with GI symptoms.[283,288,290] The prevalence of CD in these studies ranged from 10.3% to 15% of the studied group. One small study assessed the prevalence of CD in a group of patients who had IDA but no identified GI source.[286] In this study, the prevalence of CD by AGA and confirmed by EMA was 30%.

In another study, the authors assessed the prevalence of CD in pre-menopausal women with IDA.[293] The overall prevalence of CD in this population was found to be 12.9% by tTG, and 8.5% after biopsy confirmation. CD was found in 1 of 22 (4.5%) of women with heavy periods, and 4 of 18 (22%) of women with normal menstrual flow.

Four studies assessed the prevalence of CD in asymptomatic IDA patients by serology.[285,287,291,292] Two of these used EMA screening,[291,292] whereas the other two initially screened with AGA and then confirmed with EMA.[285,287] The prevalence of CD in this group ranged from 2.3% to 5.0%. Another three studies assessed the prevalence of CD by biopsy in asymptomatic IDA patients, finding it to be between 2.9% and 6%.[284,289,294]

Table 42: Included studies of CD in adult patients with anemia

Author, year; country	No. of pts	Age group	Population	Anemia type	Screening test	First serology	Confirmatory serology	Biospsy proven	Biopsy criteria	Prevalence by serology	Prevalence by biopsy
Akerman, 1996; Israel	93	Adult - some teens	Out-patients with IDA (50% symptomatic)	IDA	EGD/ biopsy			13	Subtotal or greater villous atrophy	n/a	0.139785
Annibale, 2001; Italy	71	Adults	Asymptomatic	IDA	EGD/ biopsy			4	Marsh	n/a	0.056338
Corazza, 1995; Italy	200	Adults	Referred to hematology	IDA	IgA/IgG-AGA then EMA then biopsy	16	10	10	Not mentioned	0.05	0.05
Dickey, 1997; UK	10	Adults	Asymptomatic, previously investigated no gross GI cause found	IDA	IgA AGA then EMA	4	3		Endoscopic biopsy; criteria n/r; finding of villous atrophy and IELs in duodenal biopsy	0.3	n/a
Howard, 2002; UK	258	Adults	IDA identified through lab	IDA, folate	IgA/IgG-AGA and EMA then biopsy	28		12	Not applicabe	0.10852713	0.046512*
Kepczyk, 1995; USA	39	Adults	Mostly symptomatic out-patients with IDA	IDA	EGD/ biopsy			4	Villous atrophy, crypt hyperplasia, inflammatory infiltrate	n/a	0.102564
McIntyre, 1993; UK	50	Adults	Out-patients with IDA	IDA	EGD/ biopsy			3	Not reported	n/a	0.06

*24/28 biopsied

Table 42 (cont'd): Included studies of CD in adult patients with anemia

Author, year; country	No. of pts	Age group	Population	Anemia type	Screening test	First serology	Confirmatory serology	Biospsy proven	Biopsy criteria	Prevalence by serology	Prevalence by biopsy
Oxentenko, 2002; USA	113	Adults	Undergoing EGD for IDA	IDA	EGD/ biopsy			17	CD was defined as total or partial villous atrophy with IELs	Not applicable	0.150442
Ransford, 2002; UK	484	Adults	Referred to hematology	IDA	EMA then EGD/ biopsy	17		11	Revised ESPGAN; duodenal histologic changes were graded according to Marsh I-III	0.03512397	0.022727[†]
Unsworth, 2000; UK	483	Adults	Blood donors	Anemia unspecified	IgA-EMA then biopsy	32		22	n/r	0.06625259	0.045549[‡]
Annibale, 2003; Italy	59	Adult	Pre-menopausal women with IDA	IDA	IgA tTG then biopsy	7		5	Marsh	0.11864407	0.084746[**]
Van Mook, 2001; The Netherlands	35	Adult	Asymptomatic	IDA	EGD / biopsy			1	Marsh I	Not applicable	0.028571

[†]5 Marsh I identified by CD3
[‡]25/32 biopsied
[**]5/7 biopsied; 30 had heavy periods; CD in 1/22 with heavy periods, and 4/18 with normal periods

Table 43: Summary of prevalence of CD in adult patients with anemia by population and screening test

No. of studies	Total patients	Population	Screening test(s)	Prevalence by serology	Prevalence by biopsy
3[283,288,290]	245	Symptomatic IDA	Biopsy	n/a	0.139
1[286]	10	Asymptomatic, previously no gross GI cause found investigated	IgA-AGA then EMA	0.3	n/a
1[293]	59	Pre-menopausal women with IDA	IgA-tTG then Biopsy	0.119	0.085
4[285,287,291,292]	1,425	Asymptomatic serology screened	IgA-EMA, or-AGA followed by EMA; all biopsy confirmed	0.061	0.039
3[284,289,294]	156	Asymptomatic biopsy screened	Biopsy	n/a	0.051

Prevalence of CD in Patients with Low Bone Mineral Density (BMD)

Four articles were identified that assessed the prevalence of CD in patients with low BMD (Evidence Table 9, Appendix I).[295-297,326] The study characteristics and definitions used to define low BMD, osteopenia, and CD are presented in Table 44. Three of these studies determined BMD using dual energy X-ray absorptiometry (DXA), and defined osteoporosis as a BMD less than 2.5 standard deviations from the peak bone mass of sex-matched control,[295,297,326] whereas, the other used single photon absorptiometry (SPA).[296] One study included patients with non-traumatic fractures,[295] whereas, in the others, idiopathic osteoporosis was sufficient for inclusion. All four studies used serology screening with biopsy confirmation of screen-positive patients. Three studies relied on AGA testing as the initial screen[295,296,326] followed by biopsy,[296] or further confirmatory serology testing with EMA[295] or tTG[326] prior to biopsy. The final study screened with EMA-ME, with positive screens moving on to biopsy.[297] Two studies defined the biopsy criteria for CD and used a fairly standard but rigid requirement of subtotal or greater villous atrophy.[295,297]

In the studies that used this test as the initial screen, AGA was positive in 6% to 21% of the patients with osteoporosis. However, in these studies CD was confirmed by biopsy in only 0.9% to 3% of patients.[295,296,326] The study that used EMA-ME as a screening test identified potential CD cases in 7.3% of patients, but none of these met the authors' biopsy criteria for CD.[297]

Table 44: Prevalence of CD in patients with low BMD

Author, year; country	Population	BMD definition	Test	Prevalence
Lindh, 1992, Sweden	92 consecutive patients with idiopathic osteoporosis screened for CD; 91% F (mean age 66+-12 Y); and 9 M (mean age 50+-12 Y)	Bone mineral content by photon absorptiometry (SPA) of non-dominant forearm; criteria n/r	IgA-AGA ELISA; cut-off was 2 SD above the mean of blood donors; confirmatory biopsy in 6 - criteria n/r	11/92 (12.0%) AGA +ve.; 3% (3/92) biopsy confirmed Mean proximal SPA 0.97 g/cm^2 Mean distal SPA 0.67 g/cm^2
Gonzalez, 2002; Argentina	127 postmenopausal women with osteoporosis; age (Y): mean 68, range 50-82; 747 controls; age (Y): mean 29, range 16-79	History of non-traumatic fractures and lumbar spine and/or femoral neck BMD below T-score -2.5 DXA	IgA and IgG-AGA ELISA; cut-off levels: for IgA - 15 AU/mL; for IgG - 20 AU/mL; positives confirmed with IgA-EMA-ME positive at 1:5 dilution; positives confirmed with biopsy in EMA positives; showing villous atrophy, crypt hyperplasia and IEL >30%	1/127, or 7.9 x 1000 (95% CI: 0.2-43.1); test positivity: AGA found in 8 of 127 (6.3%) pts on level 1; 1 of these 8 pts was EMA positive on the 2nd level and eligible for biopsy which established a diagnosis of CD in 1 (0.9%)
Mather, 2001; Canada	Idiopathic low BMD; mean age 57 Y; range 18-86 Y; 81.3% (78) F; 18.7% M (18) All osteopenic; 45/78 F and 13/18 M osteoporotic	DXA Osteopenia: BMD <1 SD of mean sex-matched peak BMD Osteoporosis: BMD <2.5 SD of mean sex-matched peak BMD	IgA- EMA-ME titers of ≥1:10; and biopsy confirmation based on subtotal or greater villous atrophy	7 (7.3%) of 96 pts were EMA +ve; all biopsies were negative based on subtotal or greater villous atrophy prevalence of 0%
Nuti, 2001; Italy	255 females with osteoporosis; mean age 66.6 Y range 36-65 Y	DXA BMD below T-score -2.5	IgA-AGA ELISA-cut-off level of 10 AU/mL-1; IgA-tTg cut-off >22 AU; confirmatory biopsy criteria n/r	53/255 (20.8%) +ve IgG-AGA; 24/53 +ve for tTG antibody (9.4%); intestinal biopsy in 10/24 resulted in 6 (2.4%) with confirmed CD
F=female; M-male; DXA=dual X-ray absorptiometry; Y=years; n/r=not recorded				

Quality Assessment

Using the cross-sectional checklist, the overall quality of reports of the included studies for the Celiac 2 objective, was marginal to fair (Appendix J, Table 2). For example, most of the studies did not report on whether the patients were consecutively enrolled, which could possibly lead to selection bias.

Celiac 3: Risk of Lymphoma in CD

Literature Search

Out of 379 references resulting from the literature search on CD and lymphoma, 150 were initially excluded because they did not directly address this topic (Appendix F). Of the 229 studies that were screened using full reports of the studies, 211 were excluded for the following reasons: review articles (n=73; 19.3% of level 2 articles); did not address the topic (n=33); assessed the risk of CD in lymphoma (n=28); were uncontrolled studies, including surveys (n=53); or, studied the basic mechanisms and the pathogenesis of lymphoma in CD (n=24).

The following eight exclusions were made from the 18 publications that reached level 3 (i.e., eligibility criteria): duplicate publications (n=7);[127,327-332] (for two of these reports,[328,332] patients originated from the same center [i.e., General Hospital, Birmingham]) and the reports were conducted during the same periods as other reports,[329,330,333] and we could not rule out that they were not similar series); data was not extractable (n=1).[334]

The nine controlled studies selected for data extraction were grouped as follows: eight cohort studies,[333,335-341] and one case-control study[342] (Evidence Table 10, Appendix I; Table 45). Mortality data from one controlled study in refractory CD is presented at the end of this section for reference.[343]

Table 45: Included studies for risk of lymphoma in CD

Study, year; country, period	Study type	Participants	Risk of lymphoma	Mortality	Other observations
Cottone, 1999; Sicily, 1980-97	Retrospective cohort	• 228 CD patients • 76% females • mean age at Dx 34.7 • 98% adult Dx • 100% on strict GFD	• Incidence NHL 3.1% • SIR NHL 3.75, p <0.01	SMR all causes 3.8 (1.9-6.7)	
Holmes, 1989; England, 1941-85	Prospective cohort	• 210 CD patients • 55% females • 51% on strict GFD	• Incidence NHL 4.3% • SIR NHL 42.7 (19.6-81.4)	SMR not reported	SIR NHL vs GFD compliance: • Strict GFD 44.4 • Gluten diet 100
Logan, 1989; Scotland, 1979-1986	Prospective cohort	• 653 CD patients • 60% females		Mortality from NHL 2.6% SMR from lymphoma 31 p<0.001 SMR all causes 1.9 (1.5-2.2)	SMR childhood Dx 1.4 (0.4-3.7) SMR adult dx 1.9 (1.5-2.3)
Askling, 2002; Sweden, 1964-94	Retrospective cohort	• 11,019 CD patients • 59% females • Mean age at Dx 17.4 (range 0->70)	• Incidence NHL 0.34% • SIR NHL 6.3 (4.2-125)	SMR from NHL 11.4 (7.8-16) SMR all causes 2 (1.8-2.1)	SIR NHL childhood Dx 1.9 (0.4-5.5) SIR NHL adult Dx 7.0 (5.0-9.5)
Collin, 1996; Finland, 1970-93	Prospective cohort	• 383 CD patients • 73% females • Mean age at Dx 41.8 (range 16-78) • 75% on strict GFD	• Incidence NHL 0.26% • SIR NHL 2.66 (0.07-14.8)		
Corrao, 2001; Italy, 1962-94	Prospective cohort	• 1,072 CD patients • 76% females • mean age at Dx 35.7 (range 18->50) • 59% on strict GFD		SMR from NHL: 69.3 (40.7-112.6) SMR all causes: 2.0 (1.5-2.7)	SMR age 18-29 at Dx: 2.5 (0.5-7.3) SMR age 30-49 at Dx: 2.4 (1.3-4.0) SMR age >50 at Dx: 1.9 (1.3-2.6) SMR strict GFD: 0.5 (0.2-1.1) SMR unlikely GFD: 6.0 (4.0-8.8)
Green, 2003; USA, 1981-2000	Prospective cohort	• 381 CD patients • 64% females • mean age at Dx 44 +/- 18	• Incidence NHL 1.3% • SIR NHL 6.2 (2.9-14)		
Selby, 1979; Australia, 1959-78	Retrospective cohort	• 93 CD patients • 67% females • mean age at Dx 40 (range 14-70)	• Incidence NHL 4.3% • SIR NHL 4.94, p<.0005		
Delco, 1999; USA, 1986-95	Case-control	• 458 CD patients • 4% females	• OR NHL 4.53 (2.01-10.23)		
Dx=diagnosis; SIR=standardized inidence ratio; NHL=non-Hodgkin's lymphoma; SMR=standardized mortality					

Measures of Risk

Eight out of nine studies were cohort studies, either prospective or retrospective. The standardized incidence ratio (SIR) was the most commonly reported measure of association; it was calculated as the incidence observed in the patient cohort divided by the expected incidence from the control population, along with a measure of precision (i.e., its 95% CI). The results were expressed either as SIRs of lymphoma or as the standardized mortality ratio (SMR) from lymphoma (SMR-NHL). The all-cause mortality was also reported in some studies.

It was not possible to pool these measures of risk, since SIRs by definition incorporate variables inherent to each population. The attributable risk (AR), was calculated whenever the incidence rates of NHL in CD patients and in the age-adjusted general population, were available.

Study Characteristics

There were eight cohort studies (five prospective [333,336,338-340] and three retrospective [335,337,341]) and one case-control study.[342] Two studies were from Italy,[335,339] two from the UK,[333,336] two from Scandinavia,[337,338] two from the US,[340,344] and one from Australia.[341] The observation periods varied from 7 years[336] to 44 years (1941-85;[333]), and the mean duration of patient follow-up varied from 6 years[335,339-341] to 18.6 years.[333] Patients were either selected from a national patient register,[336] from hospital discharge databases,[337,342] or represented all consecutive cases from a single[333,335,338,340,341] or multiple[339] institution(s). The cohort sizes varied from 93[341] to 11019;[337] 55% to 76% of patients with CD were female, except for the study by Delco et al.,[342] which used discharge diagnoses databases from the US Veterans Affairs hospitals (4% female CD patients). The mean age at diagnosis of CD was reported in six studies: in four studies, the diagnosis of CD was made almost exclusively in adulthood.[335,338,339,341] The mode of presentation was reported in four studies.[335,338,339,341] Adherence to a GFD was reported in five studies,[333,335,338,339,341] and could be used in the analysis in three of them.[333,338,339] Control data for the cohort studies was derived from local and national mortality data and cancer registers.

Types of Lymphomas

The total number of lymphomas diagnosed in each study and their histological type was not uniformly reported. Of the 84 lymphomas that were mentioned within these nine studies, 64 were referred to as "non-Hodgkin lymphoma (NHL)" not otherwise specified, one as "lymphoma," nine as " enteropathy-associated T-cell lymphoma (ETCL)," five as "B cell lymphoma", two as "large cell lymphoma," one each as a "T-cell other than ETCL," "lymphosarcoma" (currently classified as small cell lymphoma), and "histiocytic medullary reticulosis" (currently termed hairy-cell leukemia). Logan et al.[336] reported that they found "mostly lymphosarcomas (i.e., small-cell lymphomas) or reticulum-cell sarcomas (i.e., large-cell lymphomas) as well as two Hodgkin's lymphomas," whereas, the remaining authors systematically excluded Hodgkin's lymphomas from their respective analyses.

Incidence of Lymphoma and Related Mortality Data

The case definition of CD differed between the reports of institutional series and those derived from database analysis. The results will therefore be presented differently according to each of these two study designs.

Institutional series: By institutional studies, we mean reports on the evolution of cases consecutively diagnosed with CD and followed in one or several selected institution(s) over a specific period. Six out of the nine controlled studies were performed in that setting; in five out of six studies, the data originated from a single referral center.[333,335,338,340,341] The sixth study is the product of a collaborative effort between nine Italian centers.[339] In these studies, all cases were biopsy-proven CD.

Holmes et al., from Birmingham England, reported on a series of 210 biopsy-proven CD patients diagnosed and followed between 1941 and 1985.[333] This series was originally reported by Harris in 1967,[329] and reviewed in 1976[330] and in 1989[333] by Holmes. By this third publication, the authors had excluded all non biopsy-proven cases of CD, as well as the cases of cancer that arose either prior to or within 12 months of diagnosis of CD. The length of follow-up was of a minimum 13 years, 17.4 patient-years for men and 19.4 patient-years for women. Based on the original publication by Harris, we can assume that a large proportion of these patients (80% in Harris' series) were diagnosed with CD in adulthood. There were nine cases of NHL, compared with an expected 0.21, resulting in a SIR-NHL of 42.7 (95% CI: 19.6-81.4), which was the highest reported degree-of-risk for lymphoma among the controlled studies we identified.

Green et al.[340] prospectively followed 381 patients with biopsy-proven CD from New York City, most of whom were of European descent, and diagnosed between 1981 and 2000. The mean age at CD diagnosis was 44 +/- 18 years, and the duration of CD-related symptoms prior to diagnosis was 5 +/- 8 years. The mean follow-up was 6 +/- 11 years, for a total of 1,977 patient-years following the diagnosis of CD. There were a total of nine cases of NHL, occurring any time before or after the diagnosis of CD, leading to an attributable risk of NHL from CD of 120.2 cases per 100,000 patient years. The SIR-NHL, diagnosed at any time, was 9.1 (95% CI: 4.7-13), and the SIR-NHL for any lymphoma diagnosed at least one month after the diagnosis of CD was 6.2 (95% CI: 2.9-14).

Cottone et al.[335] reported on 228 patients with biopsy-proven CD and followed from 1980 to 1997, from a large referral center in Sicily. Ninety-eight percent of the patients had been diagnosed with CD during adulthood and the mean age at diagnosis was 34.7 years. The mean duration of follow-up was 6 years (range: 1 month to 17 years). No case of refractory CD was mentioned. There were seven cases of NHL, compared with an expected number of 1.824 (SIR-NHL of 3.75 ($p<0.01$)). The cumulative incidence of NHL was 3%, compared with an expected of 0.8%, leading to a risk difference or AR of 2.2%. The mean age at diagnosis of lymphoma was 59.4 years, and the mean time from the diagnosis of CD was 6.5 years. Lymphomas occurring prior to or within 6 months of CD diagnosis were excluded.

A large Italian multicenter study by Corrao et al.,[339] prospectively followed 1,072 patients with CD and spanned from 1962 to 1994, totaling 6,444 patient years. The mean follow-up was 6 years, and all patients were diagnosed with CD during adulthood (mean age at diagnosis of CD 35.7 years). The outcomes were strictly measured in terms of mortality data, i.e., mortality from NHL and from all causes. Events occurring at the time of CD diagnosis were included. There

were 16 instances of death from NHL. The SMR-NHL was 69.3 (95% CI: 40.7-112.6), whereas, the SMR of death from all cause (SMR-all cause) was 2.0 (95% CI: 1.5-2.7), showing that the risk of death from NHL in CD is disproportionately elevated.

Selby et al.[341] reported on a series of 93 patients with CD that were followed at a single institution in Australia between 1959 and 1978, for a mean duration of 6 years. Patients presented either during the teenage or adulthood, all were symptomatic at the time of diagnosis, and there were no refractory cases. There were four patients with NHL (simultaneous CD and lymphoma diagnosis included), compared with an expected of 0.081 (SIR-NHL 4.94, $p<0.0005$).

Collin et al.[338] reported on a prospective cohort of 383 patients with CD, diagnosed and followed at a single institution over the 1970-93 period, for a mean follow-up of 8.1 years (3,107 patient years in total). The mean age at diagnosis was advanced: 41.8 years, with a range of 16 to 78 years. Seventy-five percent of the patients adhered to a strict GFD and 82% of patients were symptomatic at the time of CD diagnosis. Simultaneous lymphoma and CD diagnoses were not excluded. There was a single case of lymphoma, compared with an expected 0.4 (SIR-NHL 2.66 [95% CI: 0.07-14.8]). As well, the 10- and 15-year survival of CD patients did not differ significantly from those of the general population.

Large database and register series: Logan et al.[336] reviewed the death certificates of CD patients belonging to a comprehensive register of CD patients that exists in Scotland since 1979, constituting a cohort of 653 CD patients gathered from 1979 to 1986. There were 17 deaths attributed to lymphoma, instead of an expected 0.55. Both Hodgkin and NHL were included, and so were those lymphomas occurring simultaneously to the diagnosis of CD. The SMR-lymphoma was 31 ($p<0.001$), which was disproportionately increased compared with the SMR-all causes, which was 1.9 (95% CI: 1.5-2.2).

Askling et al.[337] reported on the largest CD patient cohort (n=11,019), gathered from a comprehensive Swedish database of hospital discharge diagnoses over 1964 to 1994. It was not possible to ascertain how the diagnosis of CD was made or confirmed. The mean age at diagnosis of CD was 17.4 (range 0 to >70), and the mean follow-up was 9.8 years (range 0-32), for a total of 97,236 patient years. The ascertainment of outcome was achieved through the Swedish cancer register, as well as the register of causes of death. Lymphomas arising prior to or within 12 months of CD diagnosis were excluded, as for the incident lymphomas found at autopsy. There were 38 cases of NHL, and a SIR-NHL of 6.3 (95% CI: 4.2-125) was calculated. The SMR-NHL was 11.4 (95% CI: 7.8-16), which was disproportionately elevated compared with the SMR-all causes (2.0 [95% CI: 1.8-2.1]).

Delco et al.[342] used the database of discharge diagnoses from all US Veteran Affair hospitals to gather a total of 458 CD patients, hospitalized between 1986 and 1995. The concomitant diagnoses received by those patients were compared with those of five controls per CD patient, randomly selected from the same year's discharge database (total 2,692 controls). The mean age of the CD group was 63.8 +/- 12.4 years and the mean age of the control group was 59.7 +/- 14.8 years ($p<0.001$). Ninety-three percent of the patients with CD were white, compared with 74% of the control subjects ($p<0.0001$). The odds ratio (OR) of NHL (OR-NHL) in CD, was 4.53 (2.01-10.23).

Role of a GFD

The impact of GFD compliance was analyzed and reported in only two of the nine studies. Holmes et al.[333] reported a SIR of NHL in patients on a strict GFD (SIR 44.4), versus those who did not adhere to a GFD (SIR 100). Corrao et al.[339] observed that the mortality from all causes was lower in patients on a strict GFD, as opposed to those who were unlikely GFD-compliant (SMR 0.5 [95% CI: 0.2-1.1] and 6.0 [95% CI: 4.0-8.8], respectively). Although, in the study by Askling[337], compliance could not be directly ascertained, the SIR of lymphoma 1 to 4 years after diagnosis was 9.7 (95% CI:6.3-14), wheres, it dropped to 3.8 (95% CI: 2.2-6) five or more years after diagnosis, suggesting that the risk of lymphoma decreases over time on a GFD.

Risk of Lymphoma Versus Symptoms

The mode of presentation leading to the diagnosis of CD was not commonly reported. The reports from Italy[335,339] were unique in that they both detailed the circumstances by which the diagnosis of CD was diagnosed, portraying their cohorts as largely asymptomatic, since 45%[335] and 70%[339] of their patients had subclinical presentations, i.e., either mild symptoms, anemia, or were detected through screening. Conversely, it is reasonable to suggest that the studies that used hospital discharge diagnoses of CD as entry criteria would be largely made up of symptomatic CD patients. Unfortunately, it is not possible to compare the measured risk of lymphoma in the Italian studies to those of our other reports, because of the great disparities in populations, data collection and analyses amongst them.

The presence or absence of symptom at the time of CD diagnosis was not evaluated as a risk factor for lymphoma per se. Corrao et al.[339] did, however, analyze the impact of the mode of presentation on the mortality from all causes in CD. They observed that patients diagnosed with mild symptoms or by antibody screening did not show any relevant excess mortality, compared with the symptomatic group (SMR 1.2 [95% CI: 0.1-7.0] and 2.5 [95% CI: 1.8-3.4], respectively).[339]

Impact of the Age at Diagnosis of CD

Several studies analyzed the risk of lymphoma with respect to the age at diagnosis of CD. Patients who were diagnosed with CD during adulthood were either 1) asymptomatic during childhood or 2) symptomatic but eluded the diagnosis. For the later circumstance, authors have referred to “diagnostic delay” as a symptomatic period in the absence of diagnosis or treatment. The impact of the diagnostic delay was analyzed in two studies.[336,339] Corrao et al.[339] compared the mortality from all causes in patients who had suffered a diagnostic delay of more than 10 years, one to 10 years, or less than 1 year (no diagnostic delay), and found that the longer the untreated symptomatic period, the greater the mortality from all causes (SMR 3.8 [95% CI: 2.2-6.4], 2.6 [95% CI: 1.6-4.1], and 1.5 [95% CI: 0.9-2.3], respectively). Logan et al.,[336] on the other hand, reported opposite results: while the SMR-all causes was significantly greater than 1 for their entire cohort (1.9 [95% CI: 1.5-2.2]), for those CD patients diagnosed only in adult-life despite an obvious childhood illness typical of CD, all-cause mortality was similar to that of other CD patients diagnosed in adult life. A difference in methodology might explain this discrepancy, since the ascertainment of outcomes was derived from registers in Logan’s study

and was probably not as accurate and reliable for outcomes such as the presence or absence of symptoms during childhood.

Logan et al.[336] also reported that the all-cause mortality was increased in the patients diagnosed as adults, but not those who were diagnosed as children (SMR 1.9 [95% CI: 1.5-2.3] and 1.4 [95% CI: 0.4-3.7], respectively).

The patients from Corrao's cohort were exclusively diagnosed with CD as adults. The SMR-all causes for patients diagnosed between 18 and 29 years was slightly less, and not significantly different from 1.0, compared with those who were diagnosed later on in life, i.e., 2.5 (95% CI: 0.5-7.3) for those diagnosed at age 30 years versus 2.4 (95% CI: 1.3-4.0) for those diagnosed at age 49 years and 1.9 (95% CI: 1.3-2.6) for those diagnosed at age >50 years.

Askling et al.[345] reported on 11,019 patients with CD, diagnosed at all ages, and found that the SIR-NHL was not significantly greater than one in CD patients who were diagnosed during childhood, in contrast with those who were diagnosed as adults (SIR-NHL 1.9 [95% CI: 0.4-5.5] for diagnoses made at ages 0 to 19 years compared with 7.7 [95% CI: 4.9-12] for those diagnosed between 20 and 59 years). Part of the increased risk in adults may be explained by the fact that in some of these cases the diagnosis of lymphoma can be made simultaneously or soon after that of CD. However, cases of lymphoma diagnosed within 12 months of CD diagnosis were excluded from Askling' study, so that the risk of lymphoma in adult CD diagnosis remains elevated independently of cases with simultaneous presentation.

Risk of Lymphoma in Refractory CD

We were unable to identify a single source of controlled data on the risk of lymphoma in refractory CD. There was one indirect source of controlled evidence on the mortality in CD. Nielsen et al.,[343] from Denmark, published the mortality data from 98 patients with CD diagnosed between 1964 and 1982, 24% of which were treated with prednisone because they did not respond to a GFD, i.e., probable refractory CD. The mortality in CD exceeded that of the general population (controlled for age and sex) by a factor of 3.4 ($p<0.025$); in GFD-responders, this factor was 2.2 ($p<0.025$), whereas it was 5.8 ($p<0.005$) in the non-responders. The causes of death were poorly documented, and therefore, will not be described here.

Quality Assessment

The overall quality of the included studies was good (Appendix J, Tables 3-5). For example the assessment of outcomes was complete in the included studies.

Celiac 4: Consequences of Testing for CD

Out of 1,199 citations that were identified by the search strategy for the Celiac 4 objective, 140 met the level 1 screening criteria (excluded 1059) (Appendix E). Of these, 126 met the level 2 screening criteria (excluded 14). At level 3, 35 articles satisfied the screening criteria (Evidence Table 11, Appendix H)[346-380] (excluded 72 articles at level 3). Eleven relevant articles were identified in other celiac objectives: five from Celiac 2;[381-385] four from Celiac 3;[331,335,336,343] and two from Celiac 5.[386,387]

The search strategy did not identify any studies that would allow us to address the specific benefits and harms of testing with different strategies for CD. The consequences such as false-positive results were dealt with in Celiac 1. We address the response to treatment in the sections that follow.

For the consequence of osteoporosis/fracture, an additional search was conducted with the search terms osteoporosis and CD, and five additional relevant studies were identified.[388-392]

The consequences that were included in this review were: 1) costs, 2) patients complying with treatment, 3) response to treatment in terms of symptoms, and 4) clinical outcomes such as reduced risk of complications–osteoporosis, mortality, anemia.

Given the recent recognition that the number of subclinical and silent CD cases may be eight times that of classically symptomatic cases, it is important to determine if the clinical outcomes vary according to type of clinical presentation. Where possible, results of the analysis according to type of clinical presentation are presented.

Part A

Most papers included in the consequences of testing for CD dealt with patients (who were newly diagnosed) after they initiated a GFD. Most studies evaluating the consequences of nutritional status were before/after studies. In total, 15 studies dealing with either nutritional status, weight, body mass index (BMI) and body composition, were identified.[346-350,352,357,359,361,363-365,369-371]

Seven studies were case control,[347-349,352,357,364,369] one a cohort study,[346] and in seven studies, the patients acted as their own control group.[350,359,361,363,365,370,371]

Eight studies were based on children with CD,[347,349,350,352,357,359,369,370], three studies were based on adolescents with CD[363-365] and four studies were based on adults with CD[346,348,361,371].

There were five studies that evaluated costs of screening as a consequence.[360,366,379,380,382]

Type 1 diabetes and CD. Four studies evaluated diabetes and CD in children.[347,357,359,370] Three studies were from Europe (UK,[347] Hungary,[359] and Finland[370]) and one was from Australia.[357] Two were case control studies[347,357] and two studies had patients with CD act as their own controls.[359,370] All the studies assessed the effect of a GFD diet (range 3-12 months) on the diabetic control of type 1 diabetes.

The UK study[347] evaluated 230 children with type 1 diabetes who were screened for CD with serology. Those children with positive serology were biopsied. Eleven children were diagnosed with CD and followed longitudinally. The control subjects were the children diagnosed with type 1 diabetes with negative serology. The controls were matched for age, sex and duration of diabetes in a 2:1 ratio (22 controls:11 cases). At baseline, the weight (standard deviation score;

SDS), BMI SDS and HbA1c of the cases were statistically lower than the controls. No statistical difference was noted for height SDS, C-peptide level and insulin requirements. Also, the cases (type I diabetes with positive CD serology) received significantly less intensive insulin regimens compared with controls. Six type 1 diabetic children with CD participated in the GFD. After 12 months of a GFD, the differences seen in the BMI SDS was reversed between the cases and controls. HgA1c levels did not improve significantly on a GFD. Insulin dose requirements increased for both cases and controls, but still did not significantly differ from each other. Insulin regimens were not statistically different between cases and controls after a GFD.

The Australian study[357] included children and adolescents with coexisting type 1 diabetes and CD, which were identified from a database of the Diabetes Center at the Royal Alexandra Hospital for Children. CD had to be biopsy-proven. Twenty patients (5M:15F) were enrolled out of 36 patients identified on the database. Forty control patients from the same database were matched for age, sex and duration of IDDM. No immediate criteria on screening from the database was given in the study. At baseline, the current height SDS, current weight SDS, BMI SDS and HbA1c were not significantly different from controls. Compliance with a GFD was based on dietary records classifying patients to: no detectable gluten; trace of gluten; and, gluten containing. For compliance, 30% of patients were classified as adhering to a strict GFD, 30% consumed trace amounts of gluten, and 40% had a significant amount of gluten in their diet. No differences were detected in growth parameters or HbA1c according to compliance to a GFD.

The Hungarian study[359] included 205 children with type 1 diabetes that were randomly selected from screening for CD. None of these patients had suspicion for CD. Twenty-four children were positive for EMA and 17 (7 boys and 10 girls) had subtotal villous atrophy. The height of the children with CD and type 1 diabetes were normal compared with children with only type 1 diabetes at baseline. But the BMI of the 17 children was significantly lower (14.2 vs 16.3 kg/m^2) compared to controls. After three months of a GFD, BMI significantly increased (14.2 vs 16.8 kg/m^2). Furthermore, significant increases in insulin requirements (0.64 U/kg vs 0.48 U/kg) occurred after a GFD. The percentage of HbA1c did not change on a GFD compared with baseline (7.82% versus 7.67%).

The study from Finland by Saukkonen et al.,[370] retrospectively screened 776 children with type 1 diabetes over a 2.7 year period with serology and, if positive, jejunal biopsy. Eighteen children (2.3%) had confirmed CD. HbA1c levels did not change after introduction of a GFD. Correlation of height SDS and mean weight for height were not compared post-GFD.

Body composition and anthropometrics. Six studies specifically detailed body composition after a GFD.[348-350,352,369,371] Of these studies, four examined children,[349,350,352,369] and two included adults.[348,371]

Of the studies conducted in adult patients with CD, one was from Italy[348] and the other from Argentina.[371] In the Italian case-control study, 212 treated patients with histologically-confirmed CD were assessed. Of these, 71 (33.4%) (51 women and 20 men) were asymptomatic, had maintained a constant body weight during the previous 6 months, and were on a strict GFD. Forty-three of the patients were diagnosed as children (28 women and 15 men; average age 5.2 years) and 28 were diagnosed as adults (23 women and 5 men; average age 28 years). The average consumption of a GFD was $\geq$ 2 years. For each patient, there were two sex- and age-matched healthy controls (142 controls). Body composition was calculated by means of DEXA. The weight and BMI of female CD patients were lower than the controls (55.5 kg vs 58.7 kg,

p=0.004 and 20.9 kg/m^2 vs 22.4 kg/m^2, p=0.03). The height and BMD were not significantly different, although BMD for those diagnosed as adults was lower than controls. Fat mass (22.9% vs 27.5%, p<0.05) and lean mass (38.8% vs 40.5%, p<0.03) were also significantly lower in cases versus controls. The weight (69.2 kg vs 73.3 kg, p=0.03), height (175 cm vs 178 cm, p=0.05) and BMI (21.9 kg/cm^2 vs 23.5 kg/cm^2, p=0.05) of male patients were significantly lower than in controls. Fat mass (13.9% versus 16.8%, p<0.05) and lean mass (55.5% versus 56.7%, p<0.03) were also significantly lower than in controls.

The study from Argentina by Smecuol et al.,[371] enrolled 47 (41 females, 6 males) unselected, consecutive patients with newly diagnosed CD (diagnosed between Sept 1991 and Oct 1993). Twenty-five patients were re-evaluated in 1995 (24 females and 1 male). The diagnosis of CD was based on clinical features of classic and atypical symptoms, with positive small bowel biopsy and positive serology. Three patients were asymptomatic, the rest had classical features of CD. After 12 months, all patients on an initial GFD, improved. In the study, the patients acted as their own control—15 patients adhered strictly to the GFD, while ten were on a partial GFD. Patients on a strict GFD consumed less calories than patients who were poor compliers (p<0.05). After treatment, fat mass (18.2 kg, p<0.0001) and bone mass (2 kg/m^2, p<0.002) increased significantly. Lean tissue mass did not increase. Body weight (55.7 kg, p<0.0001), BMI (22.2 kg/m2, p<0.001) and triceps skinfold thickness (15.8, p<0.0001) were increased significantly; mid-arm muscle circumference and muscle mass did not change. Patients who more strictly adhered to the GFD tended to demonstrate greater increases, although the trend was not significant.

Of the four studies that evaluated children, two were from Italy[349,352], one was from the Netherlands,[350] and one was from India.[369] Both Italian studies were case-control studies, whereas, in the Netherlands study, the patients acted as their own control. In one of the Italian studies by Barera et al.,[349] 29 consecutive children (14 boys and 15 girls) with a diagnosis of CD were enrolled (mean age 9.54 ± 3.42 yr). Diagnosis was according to ESPGAN criteria. Four patients had classic symptoms, while the rest had atypical CD. The patients were studied over 1.02 ± 0.15 years of GFD. Each patient was age- and sex-matched to a healthy control patient (n=29). At baseline, children with CD weighed less than the controls (28.3 ± 11 kg vs 34.5 ± 14.1 kg, p=0.04), had lower lean mass of limbs (8.4 ± 4.8 kg vs 10.8 ± 4.7 kg, p=0.0013), less fat mass (4.6 ± 3.5 kg vs 7.5 ± 4.9 kg, p=0.006), less percentage of fat mass (17.4 ± 8.3% vs 23.7 ± 8.4%, p=0.002) and lower bone mineral content (1067.2 ± 451.3 g vs 1317 ± 553.8 g, p=0.006). Height, BMI, lean mass, and ratio of lean mass to height, did not differ from controls at baseline. After an average of 1 year on a GFD in 23 children, no significant differences were found in weight, height, BMI, lean mass, lean mass to height, lean mass of limbs, fat mass, percentage of fat mass or bone mineral content (BMC), compared with controls. Compliance was good in all patients as assessed by EMA (only three subjects were still positive).

The second Italian study by Rea et al.,[352]enrolled 23 children (8 boys and 15 girls, mean age 4.7 ± 0.76 yr) from Jan 1992 to Dec 1994, according to ESPGAN criteria. They were sex- and age-matched to healthy controls from the ambulatory clinic. At baseline, the height, BMC, arm muscle area (AMA), triceps skinfold (TSF), subscapular skinfold (SSSF), and fat area index (FAI), were significantly lower than controls. The BMI and weight for height index (WHI) were not different. After GFD, all the parameters improved when compared with patients to before GFD. Height, BMC, AMA, BMI, TSF, SSF, FAI and WHI all significantly improved. If patients post-GFD were compared with controls, the height was still significantly lower (p=0.01) but the rest of the values were not significant. After a GFD, the blood chemistry of these patients

was assessed. The hemoglobin, iron, protein, albumin triglycerides, calcium, and zinc levels were significantly different from the baseline value; however, transferrin, cholesterol, phosphorus and alkaline phosphatase levels were not different.

The study from the Netherlands by Boersma et al.,[350] enrolled 28 children (9 boys and 19 girls) with newly diagnosed CD (between Jan 94 to Jan 95). All children had classic symptoms and had positive small bowel biopsies. After 3 years of a GFD, the BMI SDS and height SDS improved significantly ($p<0.0001$ for both). The initial improvement of BMI SDS was seen in the initial 6 months with subsequent gradual improvement. The height SDS improved continuously over the 3 year period, and the improvement was significant.

In a study from India by Poddar et al.,[369] 104 children evaluated for CD between Sept 1997 to Dec 1998 were included. All children had diarrhea, failure to thrive or pallor as a clinical presentation. Fifty-seven were diagnosed as having CD (by modified ESPGAN score) and the remaining 47 were controls. Seven children who did not respond to a GFD and were excluded, were diagnosed with other diseases. The mean follow-up of patients after starting a GFD was 19.6 ± 8 months (range 4-36 months). The remaining 50 children had a dramatic response to the GFD. Symptoms subsided in 16±9.8 days (range 4-30) and all showed significant weight gain (66% ± 14% vs 86% ± 11% of expected, $p<0.001$). Height gain improved, but was not significant (88 ± 5% vs 94 ± 5% of expected, p=not significant). Seventeen percent of the children had poor compliance to the GFD. No attempt at subdividing patients into poor versus good compliance was made.

Nutritional status. Two studies looked at nutritional status with biochemical markers.

In the study from Finland by Kemppainen[346] nutritional status of newly diagnosed patients with CD before and after GFD was reported. Forty patients with CD diagnosed between Nov 1988 to Dec 1990 were included. All had abdominal symptoms. Diagnosis was made on presence of partial villous atrophy (eight patients), subtotal villous atrophy (17 patients) or total villous atroph (15 patients). On mean histomorphometric index, there was a statistically significant trend ($p=0.004$) comparing partial villous atrophy (0.018 ± 0.003), subtotal villous atrophy (0.0015 ± 0.002) and total villous atrophy (0.013 ± 0.002). When biochemical measurements were examined according to grade of villous atrophy, significant differences were seen for ferritin ($p<0.01$) and transferrin ($p<0.05$). Serum ferritin was still significantly lower in total villous atrophy, as was erythrocyte folate levels if sex was standardized in an analysis of variance. Severity of villous atrophy also correlated with ferritin, erythrocyte folate, and serum vitamin B12. Abnormal values of serum protein, vitamin A, and vitamin B12, were low. There were no abnormal vitamin E levels. Villous atrophy improved in all patients within 12 months of a GFD. Two patients had subtotal villous atrophy, 29 had partial villous atrophy and three had normal villi after a GFD. Six patients withdrew from the study. BMI increased after a GFD, as did most of the biochemical measurements. One patient with subtotal villous atrophy still had a low hemoglobin value. Of the 29 patients with partial villous atrophy, three had low folate levels, seven had low hemoglobin, one had low vitamin B12, one had low protein, five had low vitamin A, five were low in ferritin, five had low iron, and ten patients had low zinc levels. Only one patient (out of three) who had normal villi also had low hemoglobin levels.

In the study from Italy, by Bardella et al.,[361] 26 adults (five male and 21 female, mean age 42.2, range 22-81) with malabsorption and biopsy-confirmed CD were enrolled. They were followed for a mean of 55.4 months (range 13-137 months) on a GFD. Eight patients remained in good health with normal blood tests. The remaining 18 patients had abnormalities despite

GFD. No correlation was noted with severity of symptoms of malabsorption and biochemical abnormalities. Iron deficiency was found in five patients. Abnormal calcium, phosphorus, alkaline phosphatase and/or bone density was found in seven patients. Macrocytic anemia was found in four patients. Clinical symptoms were seen in 11 patients. No correlations between abnormal values and grade of histology on biopsy were found.

Compliance. Three studies were identified that looked at compliance, [363-365]. All studies were conducted in Italy and assessed an adolescent population.

In the first study of adolescents that looked at dietary compliance, Fabiani et al.[363] evaluated 28 biopsy-proven CD patients (17 females and 11 males). These 28 adolescents were selected from a group of 6,315 students, age 11 to 14 years, who had previously been screened for CD. All were advised to start a GFD. Twenty-three of the 28 patients participated in this study. The mean follow-up duration was 23 ± 7 months (range 9-3 months). Fifty-two percent (12/23) were on a strict GFD and 47% (11/23) partially adhered to the diet. Improvement in most patients was seen after starting a GFD. Weight gain was reported in 12 patients (52%)—11 had increased height velocity and appetite, eight had disappearance of symptoms of abdominal pain, six had resolution of diarrhea, five had disappearance of anemia and three had disappearance of recurrent aphthous stomatitis. Three patients did not demonstrate any change.

The second study, also by Fabiani,[364] was a 5-year case-control study that enrolled two groups of patients. The first group (group A) included subjects between the ages of 11 and 14 years, who were diagnosed as a result of a mass screening program. The second group (group B) were patients diagnosed due to typical symptoms of CD between 1985 to 1986. All patients had biopsy-proven CD according to ESPGAN criteria. All patients were followed for 5 years and advised to start a GFD. Twenty-seven patients were in group A and 22 agreed to participate; 24 patients were in group B and 22 agreed to participate. There were no differences between the patients in group A and group B in terms of BMI and height SDS. No difference was found between the two groups in terms of symptoms. Adherence to the treatment was significantly lower in patients from group A compared with group B. There were a significantly greater proportion of patients in group B that demonstrated strict adherence to a GFD (15/22; 68%) compared with patients in group A (5/22; 23%).

The third study to look at compliance looked at 306 teenage patients with CD (mean age 15.9 yr; range 10-27 yr) recruited consecutively from a CD clinic.[365] Of the patients, 186(60%) were female and 120 were male. Diagnosis of CD was biopsy confirmed. Recall questionnaire was used to evaluate diet and compliance. Compliance was recorded in three categories: 1) strict gluten diet (n=223 [73%]); 2) occasional relapse (n=46) 15%; and, 3) gluten-containing diet (n=37) 12%. Eighty percent of the female patients, compared with 64.2% of the male patients, adhered to a strict diet (p=0.012). Compliance also varied with age, with older age associated with less compliance (p=0.05). Growth status was grouped according to compliance to a GFD—the mean standardized height, the relative weight for age, and the relative weight for height, did not differ significantly between the compliance groups. Symptom scores were relatively good among all groups. No statistically significant differences were noted. School performance was not significantly different between good versus poor compliers.

Costs. Five studies included an assessment of costs involved in different screening strategies.[360,366,379,380,382]

Harewood et al.[366] performed a decision analysis to compare costs of serological testing versus small bowel biopsy (AGA vs EMA versus small bowel biopsy) for diagnosis of CD. The analytic technique used was a cost minimization and the viewpoint was third-party payer. A sensitivity analysis was conducted. The authors demonstrated that initial screening with EMA is the least costly strategy for diagnosis in a low to medium risk population.

Gomez et al.[382] evaluated a screening algorithm for CD in 1,000 consecutive subjects who were screened while attending a central laboratory. Gomez and colleagues compared two screening protocols: (1) three-level screen–IgG/IgA-AGA antibodies at the first level, then IgA-EMA, and finally intestinal biopsy versus screening, and (2) tTG-GP and total IgA as first-line screen, and EMA for positive patients followed by intestinal biopsy. The analytic framework and viewpoint were not stated. In this study, a comparative cost analysis was performed. They found that the combination of a highly-sensitive test at the first step with a highly-specific test at the second step appears to be a more reliable screening mechanism.

Zaccari et al.,[379] in an Italian model, proposed a four-level screening protocol for children at least 15 months of age, including: 1) AGA, 2) EMA, 3) intestinal permeability, and 4) small bowel biopsy. In this study, they evaluated only the total costs at each level of screening.

Atkinson et al.,[360] in a Canadian study, evaluated the operating costs of EMA in the diagnosis of CD using a cost-minimization model with a decision analytic approach with three strategies. The analytic perspective used was the societal viewpoint, and costs were discounted at 5% per annum. A one-way sensitivity analysis of all probability and cost estimates was performed. Incremental costs of the GFD were estimated from a survey of 25 patients which resulted in a lifetime incremental cost of $44,000. If a small bowel biopsy was performed initially, the cost was $997; for EMA followed by small bowel biopsy, the cost was $866. The total cost was $3,714, which resulted in an incremental cost savings of $2,177 if small bowel biopsy had been performed first. In the sensitivity analysis, the specificity of EMA would have to be greater than 95% to make EMA least expensive.

Part B

There were 27 studies that examined the response of various endpoints to a GFD.

One Italian study,[354] used a case-control design to evaluate the effect of a GFD on thyroid status. The study by Annibale et al.,[358] evaluated the impact of a GFD on anemia and iron deficiency in newly diagnosed CD cases identified from screening of adults with IDA in Italy. In a case-control study, Ciacci et al.[351] investigated the impact of a GFD on pregnancy outcomes, and Addolorato et al.[374] evaluated the impact of a GFD on anxiety and depression in a population of CD patients in Italy. Mortality was evaluated in seven cohort studies.[331,335,336,343,362,367,368] Seventeen studies assessed either change in BMD or fracture as an endpoint in individuals with CD.

Thyroid study. In the Italian study,[354] 241 consecutive adults with biopsy-confirmed CD were enrolled between Jan 1996 and July 1998 (177 women and 64 men). Forty percent of patients had classical symptoms, 44% had atypical symptoms and 16% had silent CD. Two hundred and twelve patients, matched for age, sex and ethnic origin, were used as controls. All newly-diagnosed CD patients were started on a GFD and patients with hypo- or hyperthyroidism were

started on appropriate medical therapy. Thyroid dysfunction was found in 73 (61 women and 12 men) of 241 patients with CD, and in 24 (19 women and 5 men) of the 212 patients in the control group ($p<0.0005$). The difference was statistically significant for women when divided by sex ($p<0.0005$). Hypothyroidism was diagnosed in 31 patients (12.9%) and nine controls (4.2%) ($p<0.003$); it was subclinical in 29 CD patients and eight controls and overt in the remainding patients. The difference was only significant for women ($p=0.0045$). Twenty-one patients and four controls had non-autoimmune hypothyroidism. Ten patients and five controls had autoimmune hypothyroidism. Hyperthyroidism was diagnosed in three patients and seven controls; it was subclinical in two patients and five controls. Autoimmune thyroid disease with euthyroidism was present in 39 patients and eight controls. The difference was only statistically significant in women ($p<0.0005$). At diagnosis, the BMI, hemoglobin, iron, and albumin levels were similar between patients with thyroid disease and those without. After 1 year of a GFD, 128 patients were reassessed. Ninety-one patients had normal thyroid function, whereas, 37 had some impairment. Compliance to diet was not different between the two groups. Subclinical hypothothyroidism improved in 10/14 patients with non-autoimmune hypothyroidism. Three of five patients with autoimmune hypothyroidism shifted to autoimmune thyroid disease with euthyroidism; four out of five patients with no improvement in thyroid function had poor compliance with diet. Significant improvement in nutritional indices was also seen with BMI in females, HBG in both sexes, and serum albumin and serum iron in both sexes.

Iron deficiency. In this Italian prospective study,[358] 190 consecutive patients (160 women and 30 men) who were referred to the GI department from the hematology for IDA between Jan 1994 to May 1997, were examined. Twenty-six patients were diagnosed with CD (24 women and 2 men); average age 31.3 years (range 20 -72). Seventy-seven percent of patients had total villous atrophy and 23% had subtotal atrophy; repeat endoscopy with biopsy specimens were taken after 6 months. After GFD, 20 patients (18 women and 2 men) were followed for 24 months. After 6 months, 14 of the 18 female patients (77%) recovered from IDA. Only 5/18 reversed from iron deficiency as defined by normal ferritin levels. At 12 months, 17/18 recovered from IDA. Nine patients reversed from iron deficiency. After 24 months, the same patient still did not reverse from IDA. Ten patients (55%) reversed their iron deficiency. Of the two males, at 6 months of a GFD, only one recovered from anemia but not from iron deficiency (low ferritin). At 12 months, both patients reversed their anemia and iron deficiency. At 24 months, further increases in ferritin were observed. In a subgroup of patients that had repeat small bowel biopsies at 6 and 12 months, there was a significant inverse correlation between increases in Hb concentrations and decreases in histological scores of duodenitis. This study demonstrated that recovery from IDA occurs within the first 6 to 12 months, but reversal from iron deficiency occurs in 50% of cases (predominantly premenopausal women). Long-term follow-up of ferritin results and small bowel biopsies in subjects with CD would be helpful to determine if iron deficiency resolves completely.

Pregnancy outcomes. In this case-control study from Italy by Ciacci et al.,[351] 297 women with CD were enrolled. Three types of analyses were used. Analysis A was a case-control study between untreated women (n=94; at least one pregnancy when symptoms of CD were present and lead to eventual diagnosis) and treated CD women (n=31; at least one pregnancy after 1 year of a GFD). At baseline, weight, height and body mass index were the similar between the two groups. However, the treated group was significantly younger than the untreated group (37.3 ±

12 yrs vs 22.4 ± 1.6 yrs, $p<0.01$), which may have biased the results. The number of pregnancies per woman was also lower for the treated group (2.72 ± 0.16 vs 1.6 ± 0.11, $p<0.0001$). The number of abortions per woman (0.489 ± 0.085 vs 0.032 ± 0.032, $p<0.0001$), as well as the abortion to pregnancy ratio, was much lower for the treated group compared with the untreated group(0.153 ± 0.027 vs 0.024 ± 0.024, $p<0.005$). Subgroup analysis taking into account the age at diagnosis, demonstrated that for those women diagnosed at age 30 years or less (n=27), the number of abortions per woman was 0.556 ± 0.156 and the abortion to pregnancy ratio was 0.234 ± 0.066. The prevalence of abortion in pregnancies was 17.8% in untreated CD patients, compared with 2.4% in treated patients ($p<0.001$). The RR of abortion was 8.9. Low-birth-weight baby to pregnancy ratio (0.126 ± 0.037 vs 0.024 ± 0.024, $p<0.03$) was significantly lower in the treated group. The duration of breast feeding was significantly longer for the treated group (2.77 ± 0.52 vs 7.03 ± 1.17, $p<0.0003$). The threatened abortion to pregnancy ratio and premature delivery to pregnancy ratio was not significantly different from untreated to treated CD women. For the subgroup of women <30 years (n=27), birth weight, baby to pregnancy ratio, and duration of breast feeding, did not alter the statistical significance. The prevalence of low birth weight babies in nonabortive pregnancies was 12.7% for untreated patients and 2.4% for treated patients ($p<0.05$). The RR of low birth weight babies was 5.84 times greater in the untreated group compared with the treated group.

In Analysis B, women with CD were all untreated and then analyzed depending on whether diarrhea was present or not. The authors found that the abortion to pregnancy ratio and the premature delivery ratio were found to be lower in CD women without diarrhea compared with those women with diarrhea, although the difference was not statistically significant.

In Analysis C, the effect of a GFD on pregnancy outcome was analyzed. The study examined 12 women with CD after 1 year of a GFD (own control); there was at least one pregnancy without treatment. All outcomes were better in the group of women on the GFD: number of pregnancies 2.5 ± 1.24 versus 1.08 ± 0.29 ($p<0.003$); number of abortions per woman 1.08 ± 1.16 versus 0.08 ± 0.28 ($p<0.02$); abortion to pregnancy ratio 0.405 ± 0.140 versus 0.074 ± 0.280, $p<0.02$); and, low birth weight baby to pregnancy ratio 0.292 ± 0.129 versus 0 ($p=0.05$). The threatened abortion to pregnancy ratio, premature delivery to pregnancy ratio, and duration of breast feeding, were not significantly different between the two groups. The prevalence of abortion was 43.3% for the untreated group, compared with 7.7% for the treated group of CD women ($p<0.01$). The RR of abortion was 9.18. There were no low birthweight babies born to women in the GFD group, whereas, the prevalence of low weight babies was 29.4% in the untreated group (RR=11).

One of the limitations of the Ciacci et al. study was that it did not include an external control group or control for confounders. A historical cohort population-based study of the Danish Medical Birth Registry by Norgard, 1999[393] evaluated birth outcomes in women with CD. This study included 211 newborns born to 127 mothers with CD from 1977-1992 and compared them with 1,260 control deliveries. Women with CD were identified from hospital discharge diagnoses. Discharge records were linked to Medical Birth Registry which contained information on relevant outcomes. Outcomes included birthweight, low birthweight (<2500 g) pre-term birth (<37 wk), intrauterine growth retardation (birthweight <2500 g and gestational age ≥37 wk of pregnancy), and perinatal mortality. Potential confounders including maternal age, infant's gender, parity, and gestational age, were adjusted for in the analyses. The investigators could not control for other confounders such as smoking. Another potential limitation is that the date of diagnosis of CD was the initial time of discharge from hospital with CD. It is possible

that women may have been initially diagnosed in the ambulatory care clinic. Details about the clinical presentation of the women with CD and biopsy findings were not available. The mean age at time of delivery was 27.5 years for women with CD and 26.3 years for control women.

Norgard et al.,[393] found that before women were hospitalized for CD, they were at an increased risk of low birthweight babies (adjusted OR=2.6 [95% CI: 1.3-5.5]), and intrauterine growth retardation (12.3% vs 4.8% of controls; adjusted OR=3.4 [95% CI: 1.6-7.2]). After women with CD were first hospitalized, there was no increased risk of low birthweight babies (6% post diagnosis) or intrauterine growth retardation, when compared with controls. The results of this study have implications for women with undiagnosed (atypical or silent) CD.

Anxiety and depression. The study from Italy by Addolorato et al.,[374] enrolled 43 newly-diagnosed adult patients affected with classic CD, selected from 234 adult CD patients from an outpatient clinic between June 1995 and Oct 1998. No psychiatric disorders other than anxiety and/or depression were allowed. The diagnosis of CD was based on positive serology and biopsy. Of the 43 enrolled patients, eight dropped-out leaving 35 (14 males and 21 females, mean age 29.8 ± 7.4 yr) patients for analysis. After a period of 12 months of GFD treatment, the patients were analyzed. The adherence to a GFD was evaluated based on patient self-report and family member interview. A group of 59 healthy asymptomatic controls (27 males and 32 females, age 31.7 ± 6.9 yr) were matched for gender, age, residence, employment, socioeconomic and marital status. The psychological assessment was performed using a self-rating psychometric test for anxiety (State and Trait Anxiety Inventory test) and another for depression (SDS Zung self rating depression scale). Both tests were administered before and after GFD. Of the 59 controls, 23.7% showed high levels of anxiety, 15.2% showed trait anxiety, and 9.5% were positive for depression. Of the 35 untreated CD patients, 71.4% had high levels of anxiety, 25.7% showed trait anxiety and 57.1% were positive for depression. After 1-year of GFD, 25.7% had high levels of anxiety, 17.1% had trait anxiety, and 45.7% were still depressed. The levels of high anxiety (71.4% vs 23.7%, $p<0.0001$) and levels for depression (57.1% vs 9.6%, $p<0.0001$) were significantly higher in the CD patients than in the controls. The proportion of untreated CD patients with trait anxiety did not differ from controls. After a 1-year GFD, a significant decrease in high-state anxiety (71.4% vs 25.7%, $p<0.001$) was found when treated patients were compared with the untreated group. No significant differences were found for trait anxiety or depression.

Fractures. We identified six controlled studies that addressed the outcome of fractures in a CD population[385,388-390,394]and two reviews.[381,391] The study by Cook et al.[395] was not included since it did not have a comparison or control group. The study characteristics and methods for each study are summarized in Evidence Tables 12 (Appendix H).

All six studies were retrospective and there were two cohort studies[385,388] . Two studies included individuals that had biopsy-confirmed CD. All studies included controls as a comparator, and in three studies the controls appeared to be population-based.[385,388,394] With regards to the ascertainment of the outcome of fracture, data was obtained from self-report data from administrative databases,[394] patient register,[385,388,394] or from interview/case reports.[389,390,392] Only two studies mentioned inclusion of asymptomatic subjects.[389,392] Bone histology was mentioned as an outcome in a subset of patients in one study.[390]

The case-control study by Fickling and colleagues,[390] compared individuals with CD attending a GI outpatient department and/or members of local celiac societies. The authors found a higher prevalence of past history of fractures in the CD patients (21%[16/765]) compared with a control group (3% [2/75]; RR 7.0). There was no difference in BMD T-score results between those with and without a history fracture, although those patients with a fracture history were older ($p<0.02$). Limitations of this study include the fact that they did not identify whether CD was biopsy-confirmed, and a potential for selection bias.

Thomason et al.,[373] in a case-control study, used self-report data for 244 patients with biopsy-proven CD and found that fractures were not significantly increased in those with CD compared with controls (OR 1.05, 95% CI: 0.68-1.02), although there did seem to be a trend to increased wrist fractures (OR 1.21, 95% CI: 0.66-2.25). The mean age of these patients was older (60.2) and the mean BMI was higher (23.9) than that reported in other studies. However, this study may have been limited by potentially not having adequate power to detect fractures. In addition, all the fracture data was self-reported.

Vasquez et al.,[389] in a retrospective case-control study, found that 25% (41/165) of CD patients had one to four fractures, compared with 8% in age- and sex-matched controls. The majority of fractures occurred prior to diagnosis of CD and the most common fracture site was the wrist (OR 3.5, 95% CI: 1.8-7.2). Potential sources of bias for this study include the fact that the cases were from a malabsorption clinic and may therefore represent patients with more severe disease (mean BMI=21.4). The OR for vertebral fractures was 2.8 (95% CI: 0.7-1.15), although there was incomplete ascertainment of X-rays, since not all X-rays were of adequate quality. This was the only study to include an assessment of the proportion of patients on a strict versus a reduced GFD.

Two studies were population-based.[385,388] Vestergaard et al.,[388] evaluated all individuals with CD in Denmark captured from hospital discharge data, and did not find an increase in fractures requiring hospitalization in patients with CD (n=1,021; 7,774 patient years) relative to controls (n=23; 316 patient years) with an independent independent relative risk (IRR) at pre-diagnosis of 0.70 (95% CI: 0.45-1.09) for all fractures. For spine, the IRR pre-diagnosis was 2.14 (95% CI: 0.70-6.57) and 1.07 (95% CI: 0.39-2.95) for rib and pelvis. There are significant limitations to this study since the diagnosis of fractures was hospital-based and therefore, fractures that did not require hospitalization would be missed and could lead to under-reporting. In addition, the diagnosis of CD was only validated in a sample of nine cases (with a validity of 78%), and all cases of CD had to be hospitalized to be included.

West et al.,[385] in the largest analysis of fractures in CD patients identified from the UK GPRD primary care database, found an increase in fractures in CD patients relative to controls. The mean age at diagnosis was 43.5 years, and the ascertainment of fractures was from an administrative database. For any fracture, the hazard ratio was 1.3 (95% CI: 1.16-1.46; 137.9/10,000 patient years vs 105.9/10,000 patient years in controls]). The hazard ratio for hip fracture was 1.9 (95% CI: 1.2-3.02) and the hazard ratio for wrist fracture was 1.77 (95% 1.35-2.34). The absolute difference in the overall fracture rate was 3.2/1,000 person years and 0.97/1,000 for hip fractures in those older than age 45. In contrast to earlier studies, the authors did not find a difference in the risk of fracture after CD diagnosis compared with before diagnosis.

A recent case-control cross-sectional study by Moreno et al.,[392] compared fractures in 148 CD patients (53% classically symptomatic, 36% subclinical CD, and 11% silent CD-detected by screening} to 296 controls (functional GI disorders). The fracture data was self-report obtained

by interview/and pre-designed questionnaire. Moreno et al. found an increased number of fractures in the peripheral skeleton for classically symptomatic subjects compared with controls, but did not find an increased number of fractures in the subjects with subclinical or silent CD.

BMD. BMD is a surrogate outcome for fracture, and it is easier to evaluate in short-term studies. Previous studies of osteoporosis therapies in postmenopausal osteoporosis have shown that there may not, however, be a direct correlation between fracture reduction and increases in BMD. Osteoporosis/osteopenia may be a sign of subclinical CD and persisting osteopenia/osteoporosis in a patient with known CD may be a sign that the mucosa has not normalized.

BMD is an areal two-dimensional measure of bone mass and does not give a true volumetric measure and, therefore, may not be an accurate reflection of bone mass in children.

We found 11 articles that addressed the outcome of BMD/BMC in newly diagnosed subjects with CD.[348,352,353,355,356,375-378,386,387] The study characteristics are summarized in the Evidence Tables (see Appendix H).

The majority of these studies assessed BMD at baseline and the percentage change after a variable follow-up period (1 to 5 years in duration). Two studies evaluated the BMD of children with CD,[352,377] one study evaluated a mixed population,[348] and the remaining studies evaluated adults. All studies included individuals with biopsy-proven CD and in most of the studies BMD was compared with a control population. Only two studies had patients with CD act as their own controls.[353,376] The female to male prevalence ratio in CD is 2:1, and in these studies the proportion of females varied from 50% to 80%.

Five studies included assessments of dietary compliance to a GFD and three studies included data on whether subjects were on co-interventions (e.g., vitamin D or calcium), which may have impacted the BMD results. Only two studies[356,376] looked at the potential relationship between the change in histological grade on small bowel biopsy and change in BMD.

Prevalence of osteoporosis/osteopenia. The studies consistently found that BMD results were lower in untreated subjects with CD compared with controls. Regarding the prevalence of osteopenia/osteoporosis in newly diagnosed patients with CD, the estimates varied. Satgena-Guidetta et al.[353] noted a mean Z-score of -1.5 at lumbar spine, and -1.8 at the femoral neck, with 34% of subjects having normal BMD, 40% having osteopenia and 26% osteoporosis. Valdimarsson et al.[355] found the prevalence of severe osteopenia, as defined by a Z-score less thatn -2, to be 15% at the spine, 9% at the femoral neck, and 22% at the forearm. The prevalence of mild osteopenia (defined as $-2 \leq Z < -1$) was 23% at the lumbar spine and 24% at the forearm. There was not any difference in lumber spine BMD between those patients who presented with malabsorption, compared with those patients without malabsorption. Valdimarsson et al., found that 27% of subjects had secondary hyperparathyroidism. After 1 year on a GFD, the prevalence of those with severe osteopenia decreased from 23% to 14%.

In a recent review the authors pooled prevalence results and found that patients with untreated CD had a mean Z-score of -1.42, and a hip Z-score of -1.14.[381]

Valdimarsson et al.,[356] in a prospective study of 105 newly-diagnosed CD patients, performed follow-up small bowel biopsies. Of the 105 subjects, 28 had secondary hyperparathyroidism. They found a greater reduction in BMD in individuals who had secondary hyperparathyroidism (PTH>65). In this group, the BMD increased significantly, but did not completely normalize after 3 years of a GFD. In contrast, in those with normal PTH at diagnosis, the baseline BMD was not as low and there was a 2.5% increase after 1 year with the

BMD normalizing after 2 years of a GFD. Valdimarsson also noted that 22 patients with stage III-IV had lower median Z-scores than 76 patients with mucosal changes grade I-II. In this study, compliance with the GFD was 100% in those with high PTH, and lower at 87% in those with normal PTH levels.

Kemppainen et al.,[376] in a 5-year cohort study of 28 patients in which the cases served as own controls, found that BMD increased or remained stable in 69% of patients at the lumbar spine and in 67% of patients at the femoral neck. In this study, the authors did not notice an effect of the grade of villous atrophy on the mean BMD values or percentage change in BMD. They also did not observe any correlation between adherence to the GFD and the change in BMD.

Bai,[375] in a small cohort of 45 (25 completed) newly-diagnosed CD patients, assessed compliance with the GFD and found that 84% of patients increased their lumbar spine BMD (mean increase of 12%) and total body BMD (mean increase of 7.3%), compared with 151 control subjects. The greatest increase in BMD was noted within the first year. Bai[375] documented prior fractures in two patients, but did not report any fractures during the 4-year follow-up period.

Sategna-Guidetti et al.,[353] in a longitudinal study of 86 CD patients, noted a similar proportion of patients (83.7%) increased their spine BMD after 1 year, with an increase of 5.3% in LS BMD after 1 year (change in Z-score of 0.5 at the spine).

Ciacci et al.,[386] in a retrospective cohort of 41 consecutively diagnosed patients with CD, noted a significant increase in BMD (14% lumbar spine, and 10.4% femoral neck), after 1 year on a GFD. The authors also found that pretreatment BMD predicted response to treatment.

Mustalahati et al.,[378] noted a significant increase in lumber spine and femoral neck BMD with treatment after 1 year compared with controls, and noted that the BMD was lower in symptom-free patients (n=15), suggesting patients with silent CD may have mucosal lesions for longer periods of time.

Bardella,[348] in a case-control study of 71 CD patients (43 who had started a GFD in childhood and 28 who were diagnosed as adults and were on a GFD and in remission), found that the BMD of the adult CD patients was significantly lower than the control value (0.9 g/cm^2 vs 1.1 g/cm^2, $p<0.01$).

McFarlane et al.,[387]in a case control study of 21 biopsy-confirmed subjects with CD, documented that the baseline lumbar spine BMD was 85% of that seen in controls, and the increase in lumbar spine BMD over the first year was 6.6% (95% CI: 3.1-10.1) and 5.5% in the femoral neck.

Children/adolescents. Mora et al.,[377] in a study of 19 patients (211 controls), noted a lower BMD in CD patients versus controls at baseline, and an increase in total body BMD (using DXA) during the first year when compared with controls (15.2%).

Rea et al.,[352] noted an improvement in forearm Z-score after 1 year on a GFD in 23 newly diagnosed children with CD.

Mortality. There were seven cohort studies that addressed mortality data in CD. Two were Italian studies, [335,362] one was from Denmark,[343] one from Sweden,[331] and three were from the UK.[336,367,368] All seven were cohort studies.

Corraro et al.,[362] identified 1,072 biopsy-proven CD subjects from the records of 11 GI units between Jan 1962 to Dec 1994. The inclusion criteria were complete records and reliable

diagnosis of CD. The ratio of men to women was 1 to 3, the mean age at diagnosis was 35.7 years, mean follow-up was 6.0 years and median diagnostic delay was 17 months. Forty-five percent of the population had mild (39%) or asymptomatic disease, and 50 patients were lost to follow-up. Data were collected over accumulated 6,444 patient years of follow-up, with a mean follow-up of 6 years. Adherence to a GFD was assessed. Fifty-three CD patients died compared with 25.9 expected deaths. An increase in mortality was noted in the entire cohort population (SMR 2.0 [95% CI: 1.5-2.7]). The overall SMR did not differ by sex, age of diagnosis, or year of presentation. Diagnostic delay by more than 1 year significantly increased the SMR (2.6 [95% CI: 1.6-4.1]). There was significant mortality among patients presenting with malabsorption (SMR 2.5 [95% CI: 1.8-3.4]). No excess mortality was seen with patients with mild or asymptomatic CD. Significant mortality was also seen when patients did not adhere to a GFD on clinical records (SMR 10.7 [95% CI: 6.0-17.1]) and on patient interview (SMR 6.1 [95% CI: 4.2-8.6]). The causes of death showed an excess of death from malignancy (24 observed cases, SMR 2.6 [95% CI: 1.7-3.9]) and diseases of the respiratory (SMR 3.6 [95% CI: 1.1-8.4]) and digestive tracts (SMR 6.1 [95% CI: 3.0-10.9]). NHL was seen in two-thirds of the malignant cases (n=16). The other malignancies included gastric (n=2), small intestinal (n=1), liver (n=2), pancreatic (n=1), pleura (n=1), and leukemia (n=1). (Table 45)

Cottone et al.[335] evaluated mortality in a prospective cohort study of 228 biopsy-proven CD subjects in Sicily. Mortality was ascertained by reviewing hospital medical records and pathology specimens. Records were incomplete for 5% of patients. The mean age at diagnosis was 34.7 years and 100% of patients were on a GFD. Seventy-six percent were females. The clinical presentation was anemia in 60% of cases, malabsorption in 20% of cases, and asymptomatic in another 10% of cases. The mean follow-up was 73 months. Twelve deaths were observed, with 3.12 deaths expected and the SMR from all causes was 3.8 (95% CI: 1.9-6.7). The mortality rate was increased within the initial 4 years from diagnosis, giving an SMR of 5.8 (95% CI: 2.5-11.5).

Nielsen et al.[343] from Denmark, conducted a retrospective cohort study of 98 CD patients between 1964-1982. Sixty-one percent of patients were females and the median age at diagnosis was 41 years (range 2 to 74 yrs). Twenty-four percent of patients had unclassified CD and were treated with prednisone, since they did not respond to a GFD and had probable refractory CD. Twenty-three deaths occurred during the study (four due to malignancy). Nielsen et al. found that the 5-year survival rate was 88%, the 10-year survival rate 68.5%, and that mortality exceeded that of age- and sex-matched controls in the general population by a factor of 3.4 ($p<0.025$). There was no difference in mortality between males and females (2.7 and 2.3, respectively). Subjects who responded to a GFD had an extra mortality factor of 2.2 ($p<0.025$), and those who did not respond to a GFD had an extra mortality factor of 5.8 ($p<0.005$). Causes of death were poorly documented.

Peters et al.,[331] in a retrospective cohort study, compared 10,032 symptomatic subjects with CD who had been discharged at least once from hospital, to controls who were age/sex-matched for the calendar period cancer incidence rate. Fifty-nine percent were females. Mean follow-up was 9.8 years. Mortality was ascertained from a national death register. There were 828 deaths, with 419.3 expected, resulting in a SMR of 2 (95% CI: 1.8-2.1). Mortality risk decreased slightly with increasing number of years of follow-up (p for trend, 0.004). Mortality risks were increased for patients with NHL, cancer of the small intestine, autoimmune diseases (RA), allergic disorders, inflammatory bowel disorders, diabetes, and tuberculosis.

The first UK study was conducted in Birmingham, by Holmes et al.[367] Series I included 202 patients with idiopathic steatorrhea or CD, followed from 1965-1975. Ten patients had a positive biopsy for CD. Eleven patients could not be traced. In the 10-year period, 20 deaths were seen, with ten due to malignancy. Series II (1989) had 210 patients (94 males and 116 females) with biopsy-proven CD. Seventy patients were on a normal diet and 134 were on a GFD for more than 12 months at the end of the survey. Forty-three patients had died from all causes (expected was 20.82 deaths, $p<0.001$); 21 deaths were due to malignancy—13 reticulum cell sarcomas, six GI tract cancers and two other malignancies. Of the 21, 13 had a GFD for a mean of 41 months. Deaths from all malignancies, irrespective of diet, were statistically increased as a whole (expected 5.048 vs observed 21, $p<0.001$) and divided by sex (men expected 2.878 vs observed 12, $p<0.001$ and women expected 2.170 vs observed 9, $p<0.001$). Patients taking a normal diet were at increased risk of developing a malignant tumor ($p<0.05$). Clinical response did not predict the risk of developing malignancy.

Johnston et al.[368] examined CD in subjects from Northern Ireland using the Belfast MONICA project. MONICA I was the first survey, and began in Oct 1983 with 1,204 subjects. Of the subjects, 102 (52 males and 50 females, mean age 58.1 years) had positive serology, 72 consented to follow-up (34 males and 38 females) for 11.6 years (range 11.3-11.9 years), and 20 of the 72 gave consent to biopsy. Three subjects had villous atrophy. Thirteen subjects in MONICA I (seven males and six females) died (mean age at death 67.3 yrs; range 56-75 yr). Cause of death was obtained from death certificates from the General Register Office or General Practitioner records. Four patients died with malignant disease–pancreas, stomach, bile duct lymphoma and metastatic melanoma. None of the patients had CD, but all had positive serology. The number of cancer-related deaths and all cause mortality in the MONICA I follow-up study did not show an excess number of deaths compared with the general population of Northern Ireland.

Logan et al.[336] followed a prospective cohort of 653 patients with CD in Edinburgh between 1979 and 1981. All patients had biopsy-proven CD and mortality was ascertained from death certificates. Sixty percent of the patients were females and the mean follow-up was 13.5 years. Six percent of subjects were lost to follow-up. Clinical presentation was not reported. The subjects with CD were compared with age/sex-matched controls. There were 115 deaths from all causes; the expected number was 61.8 for a SMR of 1.9 (95% CI: 1.5-2.2). The increased mortality was greatest during the initial year after diagnosis and declined over time. The mortality rate for those diagnosed during childhood was similar to that of the general population.

Quality Assessment

The majority of studies included in this objective were single group “before–after” studies, although some studies also included a comparative healthy control group. We could not identify any quality instruments for this type of study design and in general, this type of study is considered weak, particularly in the absence of a control group. Overall, however, the strength of the evidence for this objective was fair to good (Appendix J, Tables 6-8).

Celiac 5: Promoting or Monitoring Adherence to a GFD

Out of 502 citations identified by the search strategy for the Celiac 5 objective, 189 met level 1 screening criteria (Appendix F). Of these, 86 met level 2 screening criteria and 20 studies met level 3 inclusion criteria.[396-415]

Of the included studies, eight studies offered correlation between serology and mucosal histological grade,[397,398,403,404,407,409,413,415] and eight reported on serology only.[396,399-402,408,410,412] Four studies focused on histologic changes without serology.[405,406,411,414] Nine of the included studies were conducted in an adult population, six in a pediatric or adolescent population, and five studies in mixed populations consisting of adults and children.

Included articles were divided by study population (adult/children/mixed), antibody type (IgG or IgA), and by antibody methodology (e.g., ME or HU).

None of the identified studies directly assessed the efficacy of a specific intervention on the promotion of adherence to a GFD. Six studies hint at interventions that could potentially be effective.[416-421] Four of these studies were applicable to a pediatric population and two studies were applicable to adults.

Monitoring Adherence to a GFD

Biopsy. To evaluate serology in assessing adherence, some information regarding mucosal recovery on GFD must first be known. Although mucosal recovery is generally assumed to occur within 6 to 12 months after starting GFD, there is evidence that recovery may be slower and more incomplete than previously assumed.

In a mixed population, Wahab et al.[405] followed the histologic profiles of 158 patients after institution of a GFD. Histological recovery, defined as the absence of villous atrophy (Marsh 0-II), was seen in only 65% of the patients within 2 years. Within 5 years, 85.3% of patients showed recovery, and an incremental improvement to 89.9% occurred after 5 years. Of the 10.1% of patients not achieving histological recovery during the follow-up period, 11 had symptoms of CD and were therefore, considered to have refractory CD (7% of all patients). Patients with Marsh IIIb and IIIc histology initially had lower rates of recovery, compared with those with Marsh IIIa histology. In a subgroup analysis of 25 children, recovery seemed to occur faster—96% showed histological recovery within 2 years ($p<0.01$ vs adults) and 100% recovered in long-term follow-up. It is important to point out that the validity defining a Marsh II lesion as histological recovery is uncertain. If these patients were not included, rates of histological recovery would be even slower. Nonetheless, clinical improvement was seen despite the slow histological improvement.

An early study by McNicholl et al.,[406] is consistent with the finding of more complete mucosal recovery in children. Thirty-six children on a GFD for a mean of 5.8 years underwent duodenal biopsy. Mucosal morphology was normal in 16 (44%) patients, while the remainder of the patients had minimal changes. Villous atrophy was not seen. IEL counts were normal in 30 (83%) patients. A subsequent gluten-challenge confirmed the diagnosis in all 36 children.

Lee et al.,[411] in a retrospective cohort of 39 adult patients, also found incomplete mucosal recovery. After a mean duration of a GFD for 8.5 years (range 1 to 14 years), histology was normal in only 21% of patients, and partial and total villous atrophy was seen in 69% and 10% of patients, respectively. These patients were felt not to have refractory CD since they had a good

clinical response to the GFD. Also of concern were the results of serologic testing at the time of follow-up biopsy in 31 patients. Despite the relatively high number of patients with some degree of villous atrophy, IgG-AGA, IgA-AGA and IgA-EMA were negative in the majority of patients. In fact, 77% of the 31 patients having serologic tests were negative for all the listed serological tests. The exact number of these 31 patients who had some degree of villous atrophy was not reported, but would be expected to be similar to the overall numbers listed above.

Selby et al.[414] investigated whether the failure of mucosal recovery was due to noncompliance with a GFD. Eighty-nine adult patients with CD on a GFD for a mean in excess of 8 years underwent dietary assessment by a dietician, questionnaire and food diary. They were then classified as either Codex GFD, which allows up to 0.03% of protein from a gluten source, or no-detectable gluten GFD (NDG-GFD). Villous atrophy persisted at high rates in both groups, with 46% of those on Codex GFD and 40% of those on NDG-GFD having persistent villous atrophy. The patients in this study did not have clinical features of refractory sprue. Based on the fact that there were similar histologic profiles in both groups, the authors postulate that persisting mucosal abnormalities may be unrelated to gluten non-compliance. Of course, gluten intake in the NDG-GFD group undetected by study protocols cannot be ruled out.

Serology. The studies assessing the utility of serology in monitoring adherence can be divided into those with,[397,398,403,404,407,409,413,415] and those without[396,399-402,408,410,412] biopsy correlation. The studies without biopsy correlation are reviewed first. They establish an association between serologic positivity and patient compliance.

Bartholomeusz et al.[396] demonstrated higher rates of IgA-AGA positivity in non-compliant as compared with compliant CD patients in a mixed population. How compliance was ascertained is not described. Three of the 17 (17.6 %) patients compliant with a GFD for greater than 6 months were IgA-AGA positive as compared with 11 of 12 (91.6%) non-compliant patients. The PPV for non-compliance was calculated to be 78.5%.

Burgin-Wolff et al.[400] showed that, as expected, serology becomes positive with gluten challenge. One hundred and thirty-four children with CD underwent gluten challenge and were assessed for IgA-AGA and IgA-EMA-ME. At baseline, the rate of serologic positivity was 23% for AGA and 13% for EMA. Within 3 months of gluten challenge, 97% of children were positive for AGA and 65% positive for EMA. Between 3 months and 1 year, 85% of children were positive for AGA and 84% positive for EMA.

In a mixed population, Fabiani et al.[408] demonstrated significantly higher IgA-tTG-GP values in patients deemed to be non-compliant with a GFD as compared with compliant patients.

Bardella et al.[399] demonstrated that the positivity of various serologic markers falls in adults with duration on a GFD (Evidence Tables, Appendix I). The five groups in this study were untreated CD, poor GFD compliance, GFD less than 2 years, GFD greater than 2 years, and a control group. As expected, IgA-AGA, IgA-EMA-ME and IgA-tTG-GP were positive in virtually all untreated CD patients. Also, as expected, there was a low rate of positive serology in the control group, with a higher percentage being IgA-AGA positive than either IgA-EMA-ME or IgA-tTG-PG. In the poorly-compliant CD group, all were positive for all three serologic tests. In patients on a GFD less than 2 years, the rates of positive AGA, EMA and tTG were 40.9%, 54.5%, and 63.6%, respectively. In patients on a GFD for more than 2 years, the rates were 16.2%, 9.5% and 14.2%, respectively. The overlap of CIs intervals was such that no differences between the serologic tests could be determined.

Vahedi et al.[402] studied IgA-EMA and IgA-tTG in adult CD patients. Based on dietary inquiry, patients were divided into those on a strict GFD, those with minor transgressions and those with major transgressions. It was not reported whether the EMA was ME or HU, nor was it reported whether tTG was GP or HR. The median duration of GFD was 75 months. Among those on a strict GFD, 2.5% and 3% were IgA-EMA and IgA-tTG positive, respectively. Among those with minor transgressions, positivity was only 37% and 31%, respectively. Among those with major transgressions, positivity was 86% and 77%, respectively. The sensitivity of IgA-EMA for any dietary transgression was 66%, and for minor transgression it was 37%. For IgA-tTG, the sensitivities were 52% and 31%, respectively. No statistically significant differences were detected between the two serologic tests.

In a mixed population, Scalaci et al.[401] showed a low reliability for IgA-EMA in picking up dietary transgressions reported at interview. It is not reported whether ME or HU was used. In patients on a GFD for at least 6 months, only 11.1% those patients reporting one dietary transgression per month were positive, and only 19% reporting one dietary transgression per week were positive.

Fabiani et al.[410] showed a similarly low rate of serologic detection of non-compliance in screen-detected adolescents. Of 6,315 screened students, 28 biopsy-proven CD patients were found. Of these, 23 agreed to participate in a follow-up study. The mean duration of GFD was 23 months. IgG-AGA, IgA-AGA and IgA-EMA were measured. Whether EMA was ME or HU was not reported. Of the 11 patients reporting any dietary transgression, only two patients (19%) were positive for any of the serologic tests.

Pacht et al.,[412] in a similar study, showed different results. Seventeen children deemed compliant with GFD for at least 1 year were all IgA-EMA-ME-negative, whereas, 22 children deemed non-compliant were IgA-MA-ME-positive. This study suggests a much higher sensitivity for EMA than in other studies.

A number of further studies include serology and biopsy correlation. These are reviewed below.

Sategna-Guidetti et al.[413] looked at 47 adults with CD. All were IgA-EMA-ME positive at diagnosis. After 8 to 30 months of GFD, a second biopsy was taken and IgA-EMA-ME was remeasured. Total AGA was also measured in 39 patients. No patient in which the mucosa recovered to normal had a positive EMA. Only one patient with normal histology had a positive AGA (2.6%). EMA was positive in only five of 23 patients with partial villous atrophy, three of 13 patients with subtotal villous atrophy, and one of two patients with total villous atrophy. AGA was positive in only seven of 20 patients with partial villous atrophy, five of ten patients with subtotal villous atrophy, and two of two patients with total villous atrophy. The PPV of EMA for abnormal histology was 100%, but the NPV was only 23%. The PPV-AGA (total) for abnormal histology was 93.8%, whereas the NPV was only 25%. There was a clear inability of serology to adequately reflect the mucosal state in this study, and serology was negative in a significant number of patients with villous atrophy.

Valentini et al.[407] also found a significant rate of negative serology despite the presence of villous atrophy. In an adult population on a GFD for a mean of 9.9 months (range 6-12 months), 24 patients were IgA-EMA-ME negative on a GFD. Seventeen of these 24 patients (71%) had varying degrees of villous atrophy on biopsy (14 had partial villous atrophy and three had subtotal villous atrophy).

Dickey et al.[409] also showed that disappearance of IgA-EMA-ME did not necessarily indicate mucosal recovery. In adults on GFD for 1 year, IgA-EMA-ME was positive in only two of 22 (9%) with partial villous atrophy, and three of ten (30%) with subtotal/total villous atrophy.

Mengozzi et al.[403] investigated adult CD patients on a GFD for 1 year. Most (95%) had a Marsh III histology at diagnosis. In general agreement with the prior studies, only 12% had normal histology at follow-up biopsy 1 year later. Fifty percent were Marsh I and 38% were Marsh II or III (individual results for Marsh II and III were not reported). IgA-EMA-ME, IgA-tTG-HR (four different assays: DRG Diagnostics, Eurospital, Immunodiagnostik, and Celikey), and IgA-tTG-GP were measured. Taking complete mucosal recovery as a negative biopsy and all other biopsies as positive, the authors looked at concordance of serology to biopsy results. Concordance for EMA, tTG1, tTG2, tTG3, tTG4 and tTG5-PG were 48%, 29%, 65%, 14%, 16%, 19%, respectively. The validity of a Marsh I or perhaps Marsh II histology being classified as positive is unclear, and it would have been interesting to know the corresponding concordance rates if Marsh 0-I and Marsh 0-II were considered normal.

Kaukinen et al.[398] similarly found a lack of correlation between IgA-EMA-HU, IgA-tTG-GP and histologic state. Of 87 adult patients on a GFD for a median of 1 year, 27 still had a Marsh III villous atrophy. Among those with Marsh III villous atrophy, EMA was negative in 74% and tTG was negative in 59% of patients. Furthermore, of 11 patients admitting regular dietary lapses, 55% were EMA and tTG negative. The sensitivity, specificity, PPV, and NPV of EMA for Marsh III villous atrophy was 26%, 93%, 63%, and 74%, respectively. The values for tTG were 41%, 88%, 61% and 77%, respectively.

The issue arises as to whether serology might more accurately reflect mucosal state in long-term follow-up. In patients on GFD over 5 years,[398] two of four patients with Marsh III villous atrophy were EMA and tTG negative, and five of nine patients (56%) admitting dietary transgressions were EMA and tTG negative. In this study, there was no clear advantage of tTG over EMA.

One study by Fotoulaki et al.[397] did show a good correlation between serology and mucosal state. In a mixed population of 30 patients, IgG AGA, IgA AGA and IgA-EMA-ME was measured after 12 months of GFD. Contrary to the preceeding studies, all patients had either a Marsh I or II biopsy on a GFD, and all were IgA AGA and IgA EMA negative, while 40% were still IgG-AGA positive. The age range of patients in this study was much younger (1 to 24 years).

Troncone et al.[415] demonstrated that serology could miss dietary transgressions in children. Twenty-three adolescents were divided into four groups, depending on assessment of gluten intake. IgA-EmA-ME was present in seven of seven patients assessed to be taking >2 g/day of gluten. All seven also had villous atrophy. Conversely, four patients on a strict GFD, had normal histology and negative EMA. For patients with intermediate levels of gluten intake, one of six patients with a gluten intake of less than 0.5 g/d had a positive EMA. This patient also had partial villous atrophy. Three patients in this group had lesser mucosal abnormalities (increased IELs) and negative serology. For patients ingesting 0.5 to 2 g/d of gluten, three had a positive EMA; two of these had villous atrophy. Five patients had increased numbers of IELs.

Interventions to Promote Adherence to a GFD

Anson et al.[416] investigated 43 Jewish Israeli children with CD, and their parents. Thirty-one of the children (70%) were judged compliant based on a combination of clinical symptoms,

biopsy and AGA. It is unclear if serology and biopsy was performed in all children to assess compliance. Parental knowledge was studied using a structured questionnaire. A significant positive correlation between the father being a professional and compliance was found ($p<.01$). Parental level of education was also significantly correlated with compliance. Significant differences in parental ability to choose GFD items from a specific menu were found. Ninety three percent of parents of compliant children were able to pick all five GFD items out of an eight-item menu. This compared with only 67% of parents of non-compliant children ($p<.05$).

In another parental questionnaire, Jackson et al.[418] found that 30 of 50 (60%) parents reported their children to be on a strict GFD. Dietary compliance correlated with membership in the Celiac Society ($p<0.0001$). It also correlated with parental score on an eight-question test related to knowledge of CD ($p<0.001$).

Ljungman et al.[420] found self-reported GFD compliance in children to be positively associated with knowledge of CD. In this study of 47 Swedish children, those deemed compliant scored 14.03 out of 15 on a knowledge test related to CD. This compared with an average score of 12.44 in the non-compliant group.

Lamontagne et al.[419] surveyed 617 past and present members of the Quebec Celiac Foundation. A final sample size of 234 was obtained. Self-reported compliance difficulty with a GFD was inversely correlated with a high level of confidence in treatment information from gastroenterologists and dieticians ($p<.005$).

Hogberg et al.[421] looked at the effect age of diagnosis might have on compliance. In a study population of 29 adults with CD, 15 were deemed compliant with a GFD on the basis of a questionnaire and serology (IgA EMA, IgG EMA and IgA tTG). Eighty percent of patients diagnosed prior to age 4 were GFD compliant compared with 36% of patients diagnosed after age 4 ($p<.05$). A drawback of this study is that serologic markers were collected about 3 years prior to the dietary questionnaire. This risks misclassification of patients if their compliance varied over time.

In an important study with relevance to outcomes of population screening, Fabiani et al.[417] showed a lower compliance in 22 adolescents identified by a mass screening program as compared with 22 age-matched controls with identified CD on the basis of symptoms. All patients had been prescribed a GFD for more than 5 years. Twenty-three percent of screen-detected patients reported being on a strict GFD as compared with 68% of those diagnosed with CD on the basis of symptoms. Patients in the screen-detected group were diagnosed at a later age (mean 14.0 yrs) versus patients identified on the basis of symptoms (mean 4.3 yrs).

A colouring book intervention has been developed to promote GFD compliance,[422] but the effectiveness of this intervention has not been assessed in children with CD.

Quality Assessment

The majority of studies in this objective were of a "before–after" design. In this setting, this design may not pose a major limitation for monitoring studies, since the purpose of the study was to assess the change in serology and histology after introduction of a GFD. In this regard, the strength of the evidence for monitoring adherence to a GFD was fairly good. However, there is almost a complete absence of studies of interventions for the promotion of adherence to a GFD.

Chapter 4. Discussion

Celiac 1: Sensitivity and Specificity of Tests for CD

Serology

Systematic reviews of studies of diagnostic accuracy are similar in many ways to reviews of other study types, such as randomized controlled trials. However, important differences exist in large part because of the weaknesses inherent to the diagnostic-accuracy study design and its potential sources of bias.[24] In addition to these considerations, the topic of CD introduces further difficulties, and bias because of the nature of how the disease itself is defined, and the methods of patient selection for inclusion in the study. Ideally, a diagnostic-accuracy study should include a consecutive or randomly selected sample of patients from a clinically relevant patient population. That is to say, a study population who's characteristics match those of the population in which the test will ultimately be used, and both patients and controls are selected from this population. Unfortunately, selection spectrum bias is common in studies of diagnostic tests in general, and in practice it is easier for investigators to select cases and controls as separate groups in a case-control design. The practice of choosing cases that have previously been identified as having the disease, especially if more severe, introduces bias in the estimates of sensitivity (artificially raising it), while choosing completely healthy individuals as controls introduces bias in the estimates of specificity—artificially raising it as well.[24] The importance of these biases comes back to the issue of the relevant clinical population. If the test is to be used in screening healthy individuals, then the estimate of the reported sensitivity is higher than it should, but the specificity estimate is likely valid. On the other hand, if the test is to be applied to suspected cases of the disease, then the reported estimate of sensitivity may not be that far off, but the specificity estimate would be higher than it should. Other important sources of bias also exist in relation to the study population, such as the mix of other diseases present in the population with similar features as the disease in question, and ensuring an appropriate mix of disease severity in the tested population. This last point regarding disease severity is especially important for this report, and is discussed at length below.

Lijmer et al.[423] reviewed 11 meta-analyses of diagnostic tests, and assessed the characteristics of the included studies using multivariate regression analysis. The authors identified several threats to the validity of a diagnostic study's results. Case-control designs overestimated diagnositic odds ratios (DORs) by three-fold compared with studies using a clinical cohort (relevant clinical population). As well, studies that applied different reference tests to those with and without disease (in case control) or to those testing positive or negative (in relevant clinical populations) overestimated the DOR by 2.2-fold. Interpreting the reference test, with knowledge of the results of the test under study, overestimated the DOR by 1.3-fold. DORs from studies without adequate descriptions of the test or study population were 70% and 40% higher, respectively, than in studies reporting these details. Inadequate descriptions of the reference test were also identified as sources of bias.

With this information at hand we tried to minimize bias in this report, by using what some may consider fairly strict inclusion criteria which also eliminated many poor quality studies. We included both case-control studies and cohort (relevant clinical population) designs but grouped

Note: Appendixes and Evidence Tables are provided electronically at http://www.ahrq.gov/clinic/tp/celiactp.htm

them separately. Studies were only included if an adequate description of the test under study and the reference test (biopsy, and a statement of the criteria defining CD) were provided, and both the cases and controls had to have had the same reference test (i.e., biopsy) applied at the same definition or level (i.e., biopsy grade).

The results of the systematic review demonstrate that in the studied populations IgA-EMA and IgA-tTG have sensitivities and specificities each in excess of 90% in both children and adults. In fact, the pooled specificity of EMA was 100% in adults using either EMA-ME or EMA-HU. In studies of children, the specificity of EMA using these two substrates was 97% and 95%, respectively, with overlapping 95% CIs, suggesting no statistical difference between these values. In adults, the pooled specificity of tTG-GP and tTG-HR were 95% and 98%, respectively, with overlapping CIs. Similarly, in children the specificities were 96% and 99%, again with overlapping CIs. Among the three studies in adults,[32,45,70] and four studies in children[35,52,70,79] that assessed both EMA and tTG, the specificities were nearly identical. Overall, these results suggest that EMA and tTG antibodies demonstrate extremely high specificities in both adults and children.

We identified a tendency towards greater variability in sensitivity between studies and between antibodies, compared with specificity. IgA-EMA-ME demonstrated sensitivities of 97% and 96% in adults and children, respectively. EMA-HU demonstrated a similar sensitivity of 97% in children, although the pooled estimate in adults was somewhat lower at 90%. Among two studies that assessed both EMA-ME and EMA-HU in adults, one demonstrated identical sensitivities of 95%,[81] whereas, the other[57] showed a lower sensitivity of HU compared with ME (90% vs 100%). This last study only included 20 untreated patients with CD, all of whom were ME positive, but two of whom were HU negative. None of the included mixed-age studies assessed both of these antibodies. Heterogeneity existed in the analyses of sensitivity of tTG-GP in the adult, but it is likely close to 90%. In children, the pooled estimate was 93%. The sensitivity of tTG-HR was 98% in adults and 96% in children, although in both cases the CIs included a low of 90%. In studies of mixed-age populations the sensitivity was 90%.

Estimates of the sensitivity of the IgG class antibodies of EMA and tTg suggest that these tests have poor sensitivities around 40%, although the specificities were quite high at around 98%. These finding suggest that this class of antibody would be inappropriate as a single test for CD, but may be useful in IgA deficient patients, or in combination with an IgA class antibody. One study that assessed the use of IgA-tTG-HR with IgG-tTG-HR found a sensitivity of 99% and a specificity of 100% for the combination.[72]

The analyses of all the AGA subgroups demonstrated significant heterogeneity, making pooled estimates impossible. Be that as it may, the sensitivity of IgA-AGA in adults is likely not much higher than 80%, but seems somewhat higher in children. The specificity likely lies between 80% and 90%, in adults and children, although the studies of serial testing of AGA followed by EMA or tTG in the prevalence section of this report suggest that the specificity is low as well. Even if one considers an optimistic range, the performance of IgA-AGA in both adults and children is inferior to that of the other antibodies discussed above.

The analyses of IgG-AGA suffered from significant clinical and statistical heterogeneity, making even general summary statements difficult. With this in mind, the typical sensitivity of this test likely lies below 80% in adults, and between 80% and 90% in children. The specificities are likely close to 80% in adults and between 80% and 90% in children with the same warning coming from the prevalence studies, suggesting that in the era of EMA and tTG, testing for CD with AGA has a limited role.

In assessing the PPV and NPV of these tests it is important to keep in mind the prevalence of CD in the tested population. In all the included studies, the prevalence of CD would be considered quite high, the minimum study prevalence was 9%, and many studies demonstrated prevalences in excess of 40%. In comparison, Fasano et al.[15] found the prevalence of CD in at-risk first-degree relatives of CD patients to be 4.55%. In general, based on our report, the prevalence of CD in high-risk groups such as suspected CD patients, and first-degree relatives was less than 20% (in non-tertiary centers), and the prevalence in patients with anemia and diabetes was generally less than 10% (Celiac 2 section). As expected, overall the included studies demonstrated the classic relationship between prevalence and the PPV and NPVs. At the relatively high prevalence of CD in these studies, the PPV (the chance that a positive test represents a true positive test) was quite high (>90%), but started dropping at a prevalence below 35% to values generally below 80%. Figures 21 and 22 represent the actual unweighted individual study data. It is therefore not surprising that the studies maintaining a high PPV at a low prevalence were all studies of small sample sizes. In the expected reverse relationship, at a prevalence above 45% the included studies showed a drop in the NPVs. However, in contrast to the situation with the PPV, the NPV would be expected to be between 95% and 100%, if not actually close to 100%, at the expected prevalence of CD in most clinical situations. The same relationship was seen when the pooled estimates of the sensitivity and specificity for each analysis group was used to calculate the PPV over a range of prevalences (Figure 23). Therefore, the potential problem with EMA and tTG serological testing lies in their performance in situations of "low" prevalence of CD (i.e., less than 20%, a value that is still higher than the prevalence of CD in most at-risk groups). Unfortunately, it was difficult to directly estimate the PPV of EMA and tTG based on the prevalence studies, such as the one by Fasano et al., since many of the studies only performed serology testing, or there was incomplete biopsy confirmation. However, in studies where it could be estimated using the best performing EMA or tTG serological test, the PPV ranged from 66.7% to 95.0%,[209,211,212,214,215,220,223,323] with all but one study having a PPV of less than 88.9%. Most of the studies had PPVs in the range of 70% to 80%. In this same group of studies that assessed the prevalence of CD in a general population, five studies showed 100% PPV, however, in all these studies there was less than ten confirmed CD cases,[213,217,222,225,231,269] and in three studies there were three or fewer confirmed cases.[217,222,231] The PPV of IgA/IgG AGA screening alone was considerably worse, and it was not uncommon in serial testing studies to see a ten-fold drop in potential cases when moving from AGA to subsequent EMA and tTG confirmation.

From the preceding discussion it is clear that in the diagnostic studies of the serological tests, the sensitivities of EMA and tTG antibodies for the detection of CD are quite high. Furthermore the specificities and NPVs are nearly perfect, making these antibodies appealing candidates for screening, as well as for the diagnosis of suspected CD patients. However, the pressing question is whether the reported high sensitivities and PPVs in these studies, and the enthusiasm surrounding these antibody tests, will hold true when these tests are applied to different clinically relevant populations. Of concern, is the true PPV of these tests when they are applied in populations with a relatively "low" prevalence (<10%-20%) of CD. This is an important issue, since the proportion of patients who would undergo unnecessary further testing will rise as the PPV falls. For example, if the PPV falls to a value of 80% (based on the examination of Figure 21), then 20% of screen-positive individuals would undergo unnecessary testing and/or treatments. From the estimates discussed above derived from the population screening studies,

and from the plots of PPV versus prevalence, it would appear that the PPV of these tests is potentially lower than the diagnositic test studies suggest it is.

The vast majority of studies, as well as our own TEP, required that the small intestinal mucosa show at least partial villous atrophy histologically for the diagnosis of CD to be made. In fact, most of the studies used patients with subtotal or total villous atrophy. Furthermore, inherent to the clinical definitions of classic, atypical, and silent CD described in the methods, is the requirement of having a "fully developed" villous atrophy. However, Fasano et al.,[15] in a large American prevalence study, found that only 34% of biopsied EMA-positive subjects had subtotal or total villous atrophy (modified Marsh IIIb or IIIc). In this study, no EMA-positive patient had a Marsh I lesion, 26% had a Marsh II lesion and 40% had a Marsh IIIa lesion. It is clear from this study, and from the discussion about biopsy later in this section, that true CD exists in patients with histologic grades less severe than classic Marsh III lesions, and that patients with silent CD do not have to have fully developed villous atrophy. The problem that then arises is whether the reported sensitivities of these antibodies holds in the majority of patients who have CD, yet with less severe histology. As well, if the sensitivity is not as high as reported then, by definition, the nearly perfect NPV of IgA EMA and tTG would also be expected to suffer.

This question has been answered in several studies that have correlated histology with the sensitivity of these serological markers, and also mirrors to some extent the antibody response that occurs once patients with CD are placed on a GFD. A description of results of these studies follows below, while a full narrative with tables is located in the Appendix H.

Rostami et al.[16] evaluated the diagnostic value of IgA EMA and AGA in 101 untreated patients with CD. The combination of the two tests showed an overall sensitivity of 76%. But, alarmingly, the sensitivity of EMA in these patients dropped precipitously with milder histological grades. EMA demonstrated a sensitivity of 100% in Marsh IIIc, 70% in Marsh IIIb and only 30% in Marsh IIIa. The authors did not consider patients with Marsh I or II lesions as having CD.

Tursi et al.[424] assessed the relationship of the histologic grade to tTG positivity in 119 consecutive adult CD patients defined by characteristic duodenal biopsy and "permanent gluten sensitive enteropathy." In this study, the frequency of tTG-positivity (sensitivity) and mean tTG levels, were greatest with the highest modified Marsh grade, and dropped steadily with milder histologic grades reaching a low of only 8% positivity in CD patients with Marsh I lesions. The sensitivities of tTG in Marsh IIIc, IIIb, IIIa, and II were 96%, 84%, 56%, and 33%, repectively. In another publication, likely using the same population of "permanent gluten-sensitive enteropathy," Tursi et al.[425] demonstrated similar results with AGA and EMA in a population of atypical CD (defined in methods). The sensitivities of EMA in Marsh IIIc, IIIb, IIIa, II, and I, were 97%, 92%, 89%, 40%, and 0%, respectively. The results with AGA showed a similar pattern, with the sensitivity dropping from 90% to 30% in March IIIc to Marsh II.

Furthermore, in likely the same population of "permanent gluten-sensitive enteropathy," Tursi et al.[426] found a relationship between clinical manifestation of CD and EMA sensitivity. EMA was positive in 77 of 96 (80.8%) patients with atypical CD and in 17 of 27 (63.0%) patients with silent CD. EMA was negative in patients with Marsh I lesions. Once again, assuming that all these patients with "permanent gluten-sensitive enteropathy" are truly CD patients, then EMA would miss 19% of atypical CD, and 37% of silent CD that were picked up on the basis of biopsy.

Demir et al.[427] studied the presentation and clinical features of 104 newly diagnosed Turkish children. EMA and biopsy correlation was available for 72 children. Similar to what was described above, EMA was positive in 92% of patients with Marsh III lesions versus 66.6% of patients with Marsh I-II lesions. Kotze et al.[428] assessed 47 symptomatic subjects with CD with intestinal biopsy, tTG and EMA antibodies. The authors found a statistically significant correlation between antibody titres of EMA and tTG, and histologic grades.

Hoffenberg et al.[317] studied a group of children at risk of CD who were part of a large prospective study of the genetic and environmental factors associated with autoimmune diseases. No relationship was found between Marsh grade and the genetic risk factor leading to screening, but a significant correlation was found between Marsh grade and tTG ($r=0.57$, $p<0.01$).

In a small case-control study assessing the diagnostic value of EMA, Sategna-Guidetti et al. also found that in patients with documented CD, EMA positivity correlated with the severity of the histologic grade.[429] In this study, EMA was falsely negative in 50% of CD patients without villous atrophy.

The findings of the large prevalence study by Fasano et al.,[15] however, require further discussion within this context. This study demonstrated a very high prevalence of CD of 0.95% (1:105) in asymptomatic not-at-risk adults using IgA-EMA. Additionally, 34% of biopsied EMA positive subjects had subtotal or total villous atrophy (modified Marsh IIIb or IIIc), 40% had a Marsh IIIa lesion, and 26% had a Marsh II lesion. No CD patient in this study had a Marsh I lesion, although this is in part likely due to how they defined CD. In any case, there are at least two ways to interpret these results. The first is that EMA testing does pick up the mild Marsh grades, given the high prevalence of CD in this study. While the second interpretation is that based on the preceding discussion and the serology monitoring data, this study has missed an unknown number of CD patients with milder histological grades. Unfortunately, since we do not have follow-up data on the screen-negative patients in this study, this question will be difficult to answer and arguments can be made on both sides.

The question that remains, however, is whether subjects with low grade histologic lesions are at the same risk of long-term complications as those with more advanced histologic grades. On the one hand, it is apparent that symptoms may not correlate with histologic grade but rather with the length of affected small bowel. When the distribution of histological grades is compared among patients with CD who are clinically asymptomatic versus symptomatic, the same distribution of grades is seen. For practical reasons, few of the studies we identified assessed length of small bowel involvement with CD. But another question arises: are patients with early March lesions who test positive for serology the ones who have more extensive small bowel disease?[430] These questions add to the uncertainty regarding the true performance of serological testing, and whether missing early grade histologic lesions is important. Although we could not find direct evidence comparing outcomes in patients based on their histologic grades, it is not unreasonable to think that a patient with Marsh I-II lesions would still have an increased risk of CD complications (see Celiac 4 and 5 for some data regarding this point).

In summary, it is clear that from our pooled estimates of the included studies that IgA-EMA and IgA-tTG antibodies provide excellent specificity for the diagnosis of CD. However, the high reported sensitivities may only apply to the selected group of patients with villous atrophy. Furthermore, if the sensitivity is in fact lower when the entire biopsy spectrum of CD is considered, then the nearly perfect NPV of these tests, particularly in low prevalence populations, would also be expected to suffer. Finally, the PPV of these tests may not be as high as suggested when the tests are applied in low-prevalence populations, as demonstrated by our

estimates of PPV from the population screening studies. These potential limitations of serological testing can have profound implications for population screening initiatives, and verification of the sensitivity of these antibodies in a large population of CD patients showing the full histological spectrum is urgently required.

HLA DQ2/DQ8

The HLA DQ2 haplotype represents the occurrence of the HLA class II heterodimer alleles DQA1*0501 and DQB1*0201. These typically occur in a cis position as HLA DR3-DQ2 or in a trans position as HLA DR5/DR7- DQ2. The HLA DQ8 haplotype DQA1*0301/DQB1*302 typically occurs in association with DR4. HLA DQ2 occurs in about 20% to 40% of the general population,[9,10,15,100,135,136,138-141,143,146,147,150-157,159,167] 48% to 65% of healthy relatives of patients with CD,[158,161,164,166,167,169,172,177] and in up to 73% of non-CD patients with type I diabetes.[97,165] In one study, 100% of patients with enteropathy associated T-cell lymphoma (EATCL) were HLA DQ2 positive.[151] Non-CD patients with Down Syndrome appeared to have the same frequency of HLA DQ2 as the general population.[109,134,160]

Populations of non-Western European descent demonstrated very wide variations in the frequencies of HLA DQ2 both in CD patients and controls.[120,137,142,148,159,163]

Overall, it can be seen that HLA DQ2 alone offers a sensitivity in excess of 90%, which can be improved to close to 100% if a strategy of testing for both HLA DQ2 and HLA DQ8 is utilized (either test being positive). The specificity of both tests together, or either test alone, is not as good as the sensitivity, falling in the range of 55% to 80%. The specificity becomes considerably worse if a population with a higher expected frequency of HLA DQ2 or HLA DQ8, such as first-degree relatives of patients with CD or patients with type 1 diabetes, is tested. The PPV, (the probability that a positive test represents a true positive result) of testing for HLA DQ2/8 in an average population is generally low. One, however, needs to keep in mind the dependence of predictive values on the prevalence of CD in the population to be tested. Therefore, in high-risk groups, such as first-degree relatives or patients with type I diabetes, the PPV tends to be higher. Conversely, it appears that the value of testing for HLA DQ2/8 is highest when a negative test is found. Given the high NPV of this test, average-risk patients can have the diagnosis of CD excluded based on a negative test. The situation is more complex in high-risk groups, since the NPV decreases with increasing prevalence, and with the recognition that there are HLA DQ2/DQ8-negative patients with CD. These findings, along with the cost of HLA testing, make routine use of this modality for screening or diagnosis inappropriate. However, the use of this test is most useful in cases of diagnositic uncertainty or as part of a multi-test gold standard in clinical studies.

Biopsy

Unfortunately, we could not identify any studies that assessed the sensitivity or specificity of biopsy for the diagnosis of CD. This is perhaps not surprising considering that CD has historically been, and for the most part continues to be, diagnosed based on characteristic histological features. These histologic features have been classified and categorized by Marsh and others,[1,16] and criteria for the diagnosis of CD have been proposed,[2] and modified[4] (Appendix A). A biopsy showing characteristic features that improves with a GFD and recurs with gluten challenge is by definition the gold standard for the diagnosis of CD and therefore would be expected to be highly specific (some patients such as those with refractory sprue will not improve on a GFD but are still considered to have CD, so the specificity of this definition is not absolute nor perhaps completely valid). Although we do not have actual numbers, it would appear from the qualitative assessment of the identified articles that a biopsy classified as a Mash IIIa or higher is likely to have a high specificity for the diagnosis of CD. However, as seen in the study by Fasano et al.,[15] such criteria would be expected to have a low sensitivity. Alternatively, one would expect that biopsy could have a very high sensitivity if a Marsh I lesion was used to define CD, though clearly given the wide differential of mild histologic changes (Table 1, Appendix A), the specificity would be expected to drop. Therefore, to try to estimate the sensitivity and specificity of biopsy, and particularly the lower histology grades, we have compiled some articles below that provide "uncontrolled indirect information" on this subject.

Inter-observer agreement in the histologic assessment of small bowel pathology. As previously described, there are several potential criteria for the diagnosis of CD. The original and modified ESPGAN criteria[2,4] appear direct. Most of these criteria, as well as the assembled TEP, felt that some degree of villous abnormality is required for the diagnosis of CD. In practical terms, even distinguishing between a Marsh II (no villous abnormality) and a Marsh IIIa (minimal villous changes) can be difficult.[431]. This concern is further confounded by potential problems with the biopsy specimens themselves such as size, orientation, quality, and proper biopsy sampling. Hence, agreement between different pathologists and between the same pathologist at different times becomes important. The biopsy literature search identified a few articles that addressed pathologist aggreement.

Weile et al.[432] assessed inter and intra-observer agreement among three experienced Swedish and Danish pathologists reading the small bowel histology of patients suspected of having CD. Ninety small-bowel biopsies taken by capsule near the ligament of Treitz from 73 children were selected at random from a larger sample taken from 1987 to 1994. The final diagnosis was made on the basis of evaluation of specimens by dissecting microscopy, formalin-fixed H&E-stained slides, intestinal disaccaridases, serology and clinical presentation. The initial biopsy reports from patient files were sorted into normal (66; normal or minor nonspecific abnormalities—85% were on a gluten-containing diet [GCD]), pathological (17; total and severe villous atrophy, all on GCD), and inconclusive (seven; because of poor orientation, small sample, or autolysis). Several years later (1997) the same three pathologists who read the initial biopsies, performed a second reading of the slides given to them in random order. In comparison with the first reading, the number of inconclusive readings rose from seven to 22, there was a corresponding fall in the number biopsies read as normal and pathological. Considering the overall biopsy reading and diagnosis, the Kappa statistics (a statistical measure of agreement "correcting" for chance[433])

were (0.57, 0.63, and 0.75) for the three pair-wise comparisons of the three pathologists. These kappa values were reported to be "moderate" (for two out of the three agreement kappa scores) to "substantial" in terms of agreement, and suggest that agreement is far from perfect even when the same pathologist reads the same slide twice

Vilela et al.[431] also assessed inter-observer agreement among Brazilian pathologists in the diagnosis of CD. Three experienced masked pathologists independently read the slides of 34 patients with CD based on ESPGAN criteria. Agreement differed among the three possible pair-wise comparisons, with the best agreement occurring between pathologists A and C. Good to excellent agreement (kappa 0.61-0.85) was obtained for the assessment of villous structure. Reasonable to good agreement was observed for increased number of crypt mitosis (kappa 0.63), and decrease in the overall number of villi (kappa 0.47-0.53). However, agreement about the number of IELs using standard staining was weak (kappa 0.39). Interestingly, the agreement regarding overall histologic grade was also weak between two pathologist pairs, and reasonable to good for the last pair. As with the above study, it is difficult to comment on the generalizability of these results. The authors suggest that the number of CD cases seen was fewer than expected, and qualitative rather than quantitative measures of such parameters as villous height and IELs were used. Still, the findings suggest that agreement regarding the histologic grades should not be taken for granted.

Several authors have suggested that quantitating various histologic features, such as the number of IELs per 100 or more enterocytes, results in greater reproducibility of biopsy readings.[434] Authors that used quantitiative criteria during studies of inter-observer agreement likewise showed better agreement than reported above.[435-437] These studies suggest that the use of quantative methods in the reading and reporting of small bowel histology, by pathologists experienced in the reading of CD biopsy specimens, leads to greater agreement among pathologists and presumably more uniform and standardized reporting.

Latent CD. The presence of latent CD is a threat to the diagnostic accuracy of biopsy, since these patients truly have normal intestinal histology.

Stenhammar et al.[438] conducted an initial study of 100 first-degree relatives of 32 patients with CD. All 100 relatives were biopsied and two cases of CD were identified. In a 20-year follow-up study, Hogberg and Stenhammar[247] performed serological evaluation (AGA, EMA, tTg) on these same 100 relatives and their offspring, with positive results prompting intestinal biopsy. All relatives with initial "mild or moderate mucosal" abnormalities remained unchanged and were not considered to have CD. Eight new CD cases were identified, two of these were relatives of the two cases diagnosed in the first study. One of these, a parent of an affected child, had a grade II-III lesion in the first study that normalized on a GFD, and remained normal after 3 years of a GCD; she was not classified as CD, though in retrospect she likely represents a late relapser rather than transient gluten intolerance or a true latent CD. The other patient had a grade II lesion, but initially was not regarded as having CD because of the absence of symptoms. She was also found to be DQ2 positive. The remaining six newly diagnosed subjects were offspring of index CD cases and were not part of the initial cohort. In all, only two subjects of the initial biopsied cohort were "missed" in the first study. In retrospect, these subjects should have been included. This suggests that biopsy has the potential of high sensitivity and specificity for CD. Unfortunately, in the follow-up study, the number and HLA status of those with mild-to-moderate mucosal abnormalities (serology negative) was not reported, and since not all subjects

were rebiopsied it is also unclear if there is a group of serology-negative, initially normal biopsy relatives that have developed higher grade histology at follow-up, suggesting latent CD.

Maki et al.[62] likewise after an initial biopsy screen of 113 first-degree relatives of CD patients, discovered 13 relatives with villous atrophy and crypt hyperplasia. During a 3-year follow-up period another three relatives, with previously "normal biopsies" who were AGA positive, were found to have CD. Unfortunately, the authors do not report on the number of relatives with low-grade histologic lesions, and whether the new cases were in patients with completely normal (Marsh 0) lesions or normal in terms of absence of villous atrophy.

Troncone et al.[439] searched the medical records of 25 centres in Italy over a 10-year period to identify children with latent CD defined as either individuals with initial normal biopsies who later developed villous atrophy and responded to a GFD (Group 1), or people who were previously diagnosed with CD by ESPGAN criteria and who were subsequently found to have normal histology on a GCD for 2 years (Group 2). Nineteen such cases were found. All these patients had normal morphometric analysis and IEL counts on the initial biopsy. Four of the 14 GFD responders were considered at risk of CD (first degree, diabetes). The authors suggested that the five Group 2 patients could either represent true transient gluten-intolerance, or, in their opinion, more likely be late relapsers. These results of apparent post-pubertal recovery from CD are similar to those reported by Maki et al.[440] and by Schmitz.[441] Although the authors do not report on the number of charts or children screened, the findings of this study suggest that latent CD is very rare and unlikely to impact on the diagnostic accuracy of biopsy. It, however, underscores the importance of a time dimension in studies of CD, to accurately assess the true false positive and negative rates of diagnostic tests for CD.

IELs with normal villous structure. CD exists in patients with normal villous structure. The biopsy can pick up these patients on the basis of crypt changes and/or changes in the number and type of IELs.

Ferguson et al.[442] assessed the relationship of raised levels of IELs to the final diagnosis among children with diarrhea. The authors found a lack of correlation between IEL counts and morphologic grading of the biopsy. However, among seven children ultimately found to have no organic disease, all had normal IEL counts in the range of 14-25/100 epithelial cells (ECs). Two of three children with CD on a GFD also had normal IEL counts. In contrast, the values were elevated to greater than 38 IEL/100 ECs in untreated CD patients. High counts were also found in three children with failure to thrive or diarrhea of unknown etiology, and in three of nine children with giardiasis. Though in these cases, the mean values were lower than in the untreated CD cases. Interestingly, among 14 children with gastroenteritis, ten had abnormalities of the villi, crypts or lamina propria, but all but one had IEL counts within the normal range. Although, the differential of mild mucosal changes is large, this study suggests that one of the histologic features of CD can distinguish between CD and other mild enteropathies, and could potentially allow for a relatively high sensitivity by allowing CD to be defined by a low-grade Marsh lesion, while maintaining some of the specificity. This theme will be revisited in studies that follow.

Iltanen et al.[136] assessed the γδ+ IELs in patients with and without CD. One hundred and seven patients were evaluated for possible CD. Twenty seven were found to have CD (25%) on the basis of ESPGAN criteria. As well, 28 biopsy-negative adults who underwent endoscopy for dyspepsia were used as controls. Table 46 details the main study findings.

Table 46: Results of study assessing γδ+ IELs in patients with and without CD[136]

Test	Celiac (n=27)	CD excluded on biopsy (n=79)	Biopsy-negative controls (n=28)
Mean # of γδ+ IELs	40.4 (95%CI: 32.7-48.2)	6.7 (95%CI: 4.8-8.5)	1.6 (95% CI: 1.1-2.1)
Elevated γδ+ IELs (> 4.4 cells/mm)	27 (100%)	39 (49%)	n/a
AGA positive	21/26 (81%)	33/66 (50%)	n/a
Reticulin antibodies	27/27 (100%)	18/78 (23%)	n/a
HLA DQ2	19/21 (90%)	20/67 (30%)	

The mean density of γδ+ IELs was significantly greater in CD patients compared with those patients where CD was excluded on biopsy, and compared with biopsy-negative controls. The density of these IELs was also significantly higher in patients with CD excluded on biopsy compared with controls. Because the authors used the ESPGAN criteria, which requires some degree of villous atrophy, the 50% of subjects with CD excluded based on this criteria who were AGA positive begs the question of how many of these were actually CD patients. However, based on the reported data, elevated γδ+ IELs were calculated to have a sensitivity of 100%, but a specificity of only 50.6%, although the true specificity is likely higher. In the biopsy-negative suspected CD group, 66 out of the 79 underwent testing for HLA DQ2. Out of these patients, 46 tested negative for HLA DQ2. Given the high NPV of this test, it is likely that most of those patients do not have CD. Recalculating the specificity based on this assumption would raise its value, but unfortunately a breakdown of the number of patients with normal and elevated IEL in relation to HLA DQ2 was not reported. In any case, a better comparison would have been with the biopsy-negative control subjects, but the number of control subjects with raised IELs is not reported. Based on the mean density of IELs in this group, the number of patients with elevated IELs is likely to be low. During follow-up of the children suspected of having CD, but with normal mucosal biopsy and positive serology, four patients developed CD and responded to a GFD, further suggesting that this "control" group of patients with CD "excluded" on biopsy likely contained true CD patients who did not have villous atrophy. The results also suggest that the measurement of γδ+ IELs can be valuable in the diagnosis of CD, and hints at the fact that the requirement of villous atrophy on biopsy may miss some subjects with CD, particularly if they have raised IEL levels , positive serology and are HLA DQ2 positive.

Kutlu et al.[443] also studied the density of γδ+ IELs in untreated CD, treated CD and control patients (Table 47). The study population was made up of five children with classic CD with total villous atrophy and improvement on a GFD (Group A), seven patients studied after 1 to 11 years of a GFD with mucosal recovery (Group B), and 22 patients with CD by ESPGAN criteria who were left on a normal diet for 1 month to 10 years (Group C). The control group consisted of 15 children with various GI disorders other than CD, and 15 adults undergoing intestinal surgery for gastric and pancreatic disorders. The report aggregated data from groups A and C.

Table 47: Results of study assessing density of γδ+ IELs in patients with untreated CD, treated CD and control patients[443]

	Sub-total/total villous atrophy (n=18)	Moderate villous atrophy (n=7)	Normal mucosa (n=9)	Pediatric controls (n=15)	Adult controls (n=15)
Diet	normal		GFD	n/a	
γδ+ IELs/100 ECs	14.8	17.5	14.5	3.1	3.6

The density of γδ+ IELs/100 enterocytes was significantly higher in CD patients (15.4, n=34) compared with pediatric and adult control patients (3.1 and 3.6, respectively). However, the density did not correlate with histologic grade or with a GFD. Unfortunately, this study has several methodological flaws, and estimates of the sensitivity and or specificity of IEL in CD could not be derived. However, the study does indicate the potential usefulness of measuring γδ+ IELs in the overall evaluation of biopsy specimens for possible CD, and again demonstrates that CD patients can have a biopsy with normal villous structure which can be distinguished from normals by assessing the number of IELs.

In an interesting comparative study of the correlation of IELs with AGA positivity by ELISA, O'Farrelly et al.[444] studied 25 patients who had typical histologic features of CD and who were subsequently placed on a GFD. Ten of these were AGA positive, whereas 15 were negative. The second group consisted of 28 subjects suspected of CD but with "normal" small bowel histology. Twelve were AGA positive and 16 were negative. Increased levels of IELs were seen in both AGA positive (82.5) and negative (74.3) CD patients (difference not significant). On the other hand, among those with "normal" histology, AGA positive subjects had a significantly higher density of IELs than those who were AGA negative (42.4 vs 17, $p<0.001$). This data suggests that subjects suspected of CD with normal villous atrophy who have raised IEL densities should be further evaluated for CD, especially if serology is positive. These are also the types of patients where response to a GFD may be invaluable to firmly establish the diagnosis and help clarify the diagnostic value of low-grade histologic lesions.

Saputo et al.[445] compared the density of IELs between patients with confirmed CD, those undergoing investigation for CD, and control subjects (Table 48). The normal IEL range was determined to be between 4.68 and 17.60 based on the control group mean +/- 2 SD.

Table 48: Results of study comparing density of γδ+ IELs in patients with confirmed CD, those undergoing investigation for CD, and control subjects[445]

	Confirmed CD (n=9)	CD under investigation (n=40)	Controls (n=143)
IELs/50 ECs	68.55	51.21	11.14
# with raised IELs (estimated from figure)	9	40	2

These results again suggest the usefulness of IELs in the evaluation of histology of patients being assessed for CD, and suggest a sensitivity of raised IELs of 100%, and a specificity of 98.6%. Unfortunately, the authors do not report the number of individuals under investigation for CD who actually ended up having CD, so as to estimate the diagnostic parameters in this group.

Similarly, Jarvinen[436] studied IEL density and villous/crypt ratio in 928 Finnish patients with a suspicion of CD, and 59 biopsy-negative controls with dyspepsia (Table 49). CD was diagnosed on the basis of a suggestive small intestinal biopsy showing some degree of villous atrophy with subsequent later improvement on GFD. The main results excluding DH patients are presented below.

Table 49: Results of study comparing IEL density and villous/crypt ratio in patients with a suspicion of CD, and 59 biopsy-negative controls with dyspepsia[436]

	Untreated CD (n=138)	Treated CD (n=198)	Suspicion of CD with normal villi (n=545)	Controls (n=59)
CD3 + IELs	68*	40*	26	30
γδ+ IELs	19.8*	12*	3.2	2.3
Villous/crypt ratio	0.6*	1.9*	2.8	3.0
*statistically different from control				

The authors noted that using a cut off of 37 cells/mm for CD3+ and 4.3 cells/mm for γδ+ IELs, the sensitivities and specificities were 93% and 73% for CD3+, and 93% and 88% for raised γδ+ IELs, respectively. The PPVs and NPVs for raised γδ+ IELs were 95% and 85%, respectively, in this population. However, these results are based on the well-documented clear-cut CD group, and did not take into consideration the CD patients that might be in the suspicious but normal villi group. Among the patients with a suspicion of CD but normal villi and high γδ+ IELs (>4.3), 28% were EMA positive compared with only 8% with normal γδ+ IELs (<4.3). Unfortunately, the outcomes of these patients are not reported, so one cannot comment further based on this study about the usefulness of IELs in Marsh I or II patients.

Mino et al.[446] assessed the density of IELs in routinely stained specimens compared with specimens stained with the readily available CD3 antibody. Twenty-eight subjects with architecturally normal duodenal biopsies, which were well-oriented and demonstrated greater than 20 IELs/100 ECs were included in the study. AGA, EMA and tTG antibodies were measured. Subjects were divided in the groups listed in Table 50. Controls consisted of seven normal individuals, two patients with reflux, and two patients with irritable bowel syndrome.

Table 50: Results of study assessing IEL density in routinely stained specimens compared with specimens stained with the CD3 antibody[446]

	CD (n=8)	Treated CD (n=4)	Non-CD (n=16)	Controls (n=11)
Mean age	33.5	46.3	46.4	39.1
IELs/100 ECs by H&E staining	42.1	29.2	36.8	Not increased
IEL/100 ECs in villous tip by CD 3 staining	47.5	29.4	33.2	8.2

There were no statistically significant differences between any of the groups when IELs were measured with H&E staining. However, all pair-wise comparisons were statistically different, except between the treated CD group and the non-CD group, when villous-tip IELs were counted with CD3 staining. The authors conclude that villous tip IELs are more specific indicators of CD, particularly with CD3 staining (which is more readily available than staining for γδ+ IELs),

and suggest that the specificity of low grade Marsh lesions could be improved by these techniques.

In a similar study, Goldstein et al.[447] compared IEL density and villous distribution among patients suspected of CD. Twelve patients were diagnosed with CD based on histologic features and response to a GFD, whereas in 66 patients the diagnosis of CD was excluded based on biopsy, and supported by negative serology (and in some cases a lack of response to a GFD). Control cases consisted of patients with dyspepsia who underwent endoscopy and biopsy. The main results are summarized in Table 51.

Table 51: Results of study comparing IEL density and villous distribution among patients suspected of CD[447]

	CD (n=12)	Non-CD (n=66)	Controls (n=24)
Mean age	35.2	36.1	34.5
Iga EMA	8	3 (no response to GFD)	n/a
IgA AGA	5	13 (all EMA neg.)	n/a
Villous tip IELs	11.6	4.3	2.2
IELs distributed evenly along the villi	9/12 (75%)	3/68 (4%)	0
n/a = not applicable			

The authors found that the mean villous tip IEL density was significantly greater in the CD group than in the non-CD and control group. A more even distribution of IEL along the villi was also found to be significantly more common in the CD group compared with the other groups. However, this last point is controversial. Unfortunately, given that this is a small study, the authors did not look at differences in these characteristics among CD patients with different histologic grades.

Kuitumen et al.[448] compared the histologic features of children with untreated CD, treated CD, other GI disorders (cow's milk allergy, DH, congenital lactase deficiency, acrodermatitis enteropathica, and giardiasis) and a group of control subjects without GI pathology. Of the 52 children with CD in this group, all had severe villous atrophy. CD patients had the lowest enterocyte height, and the most intense IEL infiltration of the studied groups. The authors found no overlap between CD patients and controls for the density of IELs, villous height, crypt depth, and villous height to crypt depth; all these parameters were statistically different between the CD patients and controls.

Kaukinen et al.[449] studied 96 consecutive adults found to be ARA or AGA positive and compared them with 27 ARA- and AGA-negative patients with dyspepsia. All patients underwent duodenal biopsy and CD was diagnosed on the basis of a villous height to crypt depth of less than two and crypt hyperplasia. Twenty-nine patients met their biospsy criteria of CD (18 ARA- and AGA-positive patient, nine ARA-positive patients, and two AGA-positive patients). The 29 CD patients were placed on a GFD and of the 21 who were rebiopsied at 6 to 12 months, all showed unequivocal histologic improvement. The mean density of IELs in CD, serology positive, biopsy negative, and control patients were 87, 38, and 25 cells/mm, respectively. These numbers were statistically different. The mean density of γδ+ IELs among the CD patients was 16.6. Eleven serology-positive patients with normal villous structure (presumably Marsh I and II) expressed HLA DR and had higher levels of γδ+ IELs (mean of 13.4 cells/mm) than the non-CD controls. A repeat biopsy (time unspecified) was performed in 12 serology-positive patients

with normal villous structure at the time of the first biopsy. Ten of these had raised γδ+ IELs density on biopsy (Marsh I or greater). Five of these 12 were found to have villous atrophy (Marsh IIIa or greater). This study further illustrates the later development of CD in subjects with mild histologic changes, and suggests that although the specificity of villous atrophy may be high (all patients responded to a GFD), the sensitivity of villous atrophy (Marsh IIIa or higher) is lower than that of the serological test used in this study. This suggests that using a lower biopsy cut-off grade could improve sensitivity, albeit at the cost of specificity.

Using another approach, Wahab[450,451] identified 38 patients with symptoms of malabsorption who only demonstrated raised epithelial lymphocytes on duodenal biopsy (Marsh I). These patients were given a gluten challenge of 30g/day for 2 months, while maintaining their normal GFC. Twelve of 38 patients developed worsening mucosal lesions of crypt hyperplasia and partial or subtotal villous atrophy. After institution of a GFD all 12 patients showed improvement of their malabsorption, and improvement of their histology, suggesting that they truly had CD.

The same authors,[451] similarly studied 27 patients referred for malabsorption who were found to have a Marsh II lesion. HLA DQ2 or DQ8 was found in 21 of 27 patients (78%). The authors motivated 25 patients to follow a GFD, and all showed symptomatic improvement. The two patients who refused the GFD progressed to a Marsh IIIa lesion at follow-up. Although these data provide evidence of the true existence of CD in patients with Marsh II lesions, the frequency is unlikely to be as high as reported here. The high NPV of HLA DQ2/DQ8 suggests that at least some of the six testing negative likely don't have CD. In any case, this study adds further evidence to the notion that a Marsh III cut-off will miss some patients with CD.

In a very interesting study, Mahadeva et al.[452] identified all duodenal biopsies performed over a 1-year period with increased levels of IELs, yet normal villous structure. Biopsies were formalin fixed and stained with H&E. Other biopsies showing at least subtotal villous atrophy and increased IELs were considered as "suggestive of CD." Two normal control duodenal biopsies for every case of increased IELs with normal villous structure were also obtained. The upper limit of normal for IEL levels in this study was 22 IELs/100 ECs. Out of 626 biopsies assessed, 14 (2.2%) were found to have increased IEL and normal villous structure, whereas 15 (2.4%) cases of CD were identified. Normal histology was found in 502 (80.2%) of the biopsies. The biopsies with raised IELs had a mean of 38 IELs/100 ECs (range of 27-46). Control biopsies on the other hand had a mean of 12.4 IELs/100 ECs (range of 2-20). The presence of GI symptoms did not differentiate those with raised IELs from controls or CD patients in this cohort. Six of the 14 patients with raised IELs had positive EMA and/or unexplained anemia and were suggested as having "latent" CD by the authors. Unfortunately, follow-up in this group was incomplete with only three of these patients undergoing repeat biopsy. As with the previously described studies, the presence of patients evaluated for possible CD who have isolated increased IELs may contain a subset of true CD patients. In fact, if one assumes that the six EMA positive subjects with raised IELs do in fact have CD, then one can estimate that using a lower histologic grade to define CD in this population would have resulted in a sensitivity of biopsy of 100%, and a specificity of 98%—since only eight patients out of the studied sample of 531 would have been misclassified as having CD when in fact they did not. Of course, the expected specificity would not be as high as the one produced in this exercise since the authors do not tell us the histologic features or the diagnoses of the remaining 95 patients (626 biopsied, minus 502 normal, minus 15 CD, minus 14 raised IEL and normal villous structure = 95). However, taking this exercise further, if we assume that all of the other 95 patients were

misclassified as having CD, then the specificity would drop to a still respectable 83%. Clearly, this type of study is the starting point in assessing the diagnostic parameters of the biopsy itself as a test. However, what is needed to fully assess biopsy as a test is a clearer measure of the false positive and negative rates. This can only be accomplished by using a battery of tests (biopsy, serology, HLA) to act as a gold standard to initially identify all potential cases, and then a follow-up period (response to GFD or gluten challenge) to assess the permanence of the diagnosis and the utility of biopsy at various cut-offs when used alone.

Kaukinen et al.[453] performed a study partially fulfilling the above requirements. Ten patients with suspected CD but only Marsh I or II lesions were compared with 27 biopsy-normal controls. The suspected cases were assessed before and after a GFD. The main results are presented in Table 52.

Table 52: Results of study assessing patients with suspected CD and Marsh I or II, before and after a GFD[453]

	Histology	EMA+	TTG+	HLA DQ2	γδ+ IELs
Initially	Marsh III – 2 (patchy) Marsh II – 7 Marsh I – 1	8/10	9/10	9/9	Marsh III – 25 cells/mm Marsh I-II – 13 Controls – 1.4
After GFD	All Marsh II re-biopsied Marsh I – 2 Marsh 0 – 5	0/10	1/10 (Slightly elevated)	Same	Reported as decreased values not reported.

Although this is a small study with possible selection bias, the authors demonstrate that in a subset of patients suspected of having CD but without villous abnormalities, CD was diagnosed in all on the basis of a response to a GFD. Raised levels γδ+ IELs, positive serology, and HLA DQ2 positivity, supported the diagnosis of CD. Patients with CD and Marsh I-II lesions had significantly higher levels of IELs than controls. Unfortunately, this study did not include a larger sample of patients with Marsh I-II histology that included serology-negative subjects. Although it is clear based on this study that CD can exist in patients with Marsh I-II lesions with raised γδ+ IELs, it is difficult to generalize these results to an unselected sample of suspected CD patients.

In a somewhat complicated but important study, Kuakinen et al.[98] assessed 271 patients with suspected CD by biopsy. Forty-five patients were classified as having definite CD on the basis of a Marsh III lesion. While in 136 patients, CD was excluded on the basis of a Marsh 0 lesion and normal levels of γδ+ IELs. The remaining 76 patients had an uncertain diagnosis of CD based on biopsy (absence of villous atrophy) and underwent HLA DQ2 and DQ8 testing. In 59 of these patients, there were minor mucosal lesions or positive serological markers, while 17 were already on a GFD prior to biopsy. CD was excluded in 11 of these 17 patients on a GFD. Of the remaining 59 patients, CD was excluded in 22 because of a negative HLA DQ2/8 given the high NPV of this test, whereas 37 were DQ2/8 positive and remained with the suspicion of CD. Overall, CD was excluded in 33 of 76 patients. Among patients suspected of CD, but without villous atrophy, Marsh I-II lesions were found in 20 DQ2/8-positive patients versus in five DQ2/8-negative patients. Elevated levels of γδ+ IELs were found in 20 patients who were DQ2/8 positive compared with seven patients who were DQ2/8 negative, and IgA-EMA was found in 16 patients who were DQ2/8 positive compared with 0 patients who were DQ2/8 negative. Although data is not provided for some patients, one can estimate the sensitivity of

using a Marsh III cut-off. We know that CD was diagnosed outright in 45 out of 271 patients, but with subsequent testing a further 37 patients were found to be positive for HLA DQ2 or DQ8. At least 16 (EMA positive) and likely 20 (increased IEL counts) of these patients likely have CD. Based on these assumptions, the sensitivity of a Marsh III cut-off is between 69% (20 DQ2/8 patients with increased IELs have CD) and 74% (16 EMA and DQ2/8-positive patients have CD). The sensitivity would be lower if more of the DQ2/8 positive patients turned out to have CD. The specificity of that cuff-off would appear to be 100%, although we are not told if the Marsh III patients all improved on a GFD. Clearly using a biopsy cut-off lower than Marsh III would have increased the sensitivity, but unfortunately we are not given enough information to estimate this reliably.

This study with its battery of tests comes closer to the ideal design to estimate the diagnostic characteristics of biopsy, but unfortunately, it has significant short comings. To be fair the intent of the study was not to determine the sensitivity of a Marsh III cut-off. However, for the sake of future studies in this area, several design changes could have allowed this estimation. This study had two important positive aspects: it used a relevant clinically important population of patients suspected of having CD, and all the subjects underwent biopsy. However, it would have been ideal, if all the subjects also underwent HLA testing and serology. Furthermore, a follow-up of positive and negative patients, and or the assessment of the response to a GFD or the use a gluten-challenge in difficult to diagnose patients, would have allowed for the estimation of false positive and negative cases.

Relationship of serology to histology. As the data from the previous discussion suggests, CD clearly exists in patients with histological grades milder than Marsh IIIa. The fact that the sensitivity of biopsy is improved by using a lower grade as a cut-off brings up an important question. If the preceding statement is true, then what test is most sensitive for detecting CD with mild histologic changes—biopsy or serology? The issues surrounding this discussion have been addressed in the later portion of the serology discussion section, and a detailed narrative summary of the studies of the relationship of serology to histology can be found in Appendix H. However, to summarize, data from these studies as well as some data from Celiac 5 suggest that the sensitivity of serology drops with milder histologic grades, and suggests that serology alone would miss CD patients with mild histology grades.

In summary, CD exists in patients with histology grades less than Marsh IIIa. The sensitivity of biopsy at a Marsh IIIa or higher cut-off is likely less than that of serology with EMA or tTG. If lower Marsh grades are used, the sensitivity of biopsy increases, and it is possible that if morphometeric techniques including assessing IEL densities are used, the specificity may not suffer greatly. Ultimately, the question of the true sensitivity of biopsy can only be answered with a well-conducted study that attempts to identify all possible CD patients in a given clinically relevant population using multiple simultaneous tests (e.g., serology, HLA) in addition to biopsy. All patients, those who clearly have CD, those in whom CD seems excluded, as well as equivocal cases, need to be followed for the assessment of the permanence of their "diagnoses." Equivocal cases could also be considered for further testing, either with assessing response to a GFD or gluten challenge, to help in the clarification of their diagnosis. Although there are other potential variables to consider, with these measures, assessment of the false positive and false negative rates of biopsy, and hence a clearer estimate of the sensitivity and specificity, can be determined.

Celiac 2: Incidence and Prevalence of CD

Incidence in the General Population—Different Geographic and Racial/Ethnic Populations

The crude incidence of CD among western European and North American countries over the past 25 years has varied between 1 and 51 per 100,000, and the cumulative incidence by age 5 between 0.118 and 9 per 1,000 livebirths. Notable variations in CD incidence have not only been striking between neighbouring countries, such as is the case for Sweden and Denmark, but also between time periods for the same region, such was noted in the UK between the 70's and 80's as well as in Sweden over the 90's.

It is important to note that there were important methodological differences among the studies, from using patient registers[200] to actively screening at-risk patients.[128] Clinical practice also varied between time periods and regions. The advent of serological testing in the early 90's changed attitudes towards screening and identifying populations at risk with resulting higher detected incidences of CD. In some studies, active efforts were made to detect CD among asymptomatic subjects, such as the case in Finland where all subjects referred for endoscopy underwent small intestinal biopsy, independent of the cause for referral.[199] The incidence of CD is also expected to vary according to the genetic make-up of the studied population, although the prevalence of at-risk HLA haplotypes was only noted in one study.[128] These observations also highlighted the importance of dietary factors in triggering so-called CD epidemics among genetically predisposed populations. It would appear that breastfeeding bears a protective role, while early introduction of gluten, as well as the amount of gluten content in the diet may promote the early serological and pathological manifestations of CD. It is unknown whether these factors trigger an earlier expression of a disease which would become manifest anyway, or whether they trigger the appearance of a disease which may not otherwise occur, even later on in life.

In conclusion, caution should be exercised when extrapolating the noted incidence for one given region to a whole country, in particular in countries such as the US where there are differing population ethnicities among regions, between rural and urban areas, as well as between small and large cities. However, it remains that the true incidence and prevalence of CD are if anything greater than reported in clinical settings, since observations derived from screening and case-finding efforts were consistently greater than those relying on the diagnosis of clinically suspected cases. Lastly, it is important to bear in mind that, considering the large proportion of subjects with silent CD (the so-called celiac iceberg), observed incidences will depend upon the efforts spent screening cases, as is well illustrated by the difference in the relatively low incidence observed over 30 years in Olmstead county, where the majority of cases had clinically overt disease, as opposed to the very high incidence noted in Denver Colorado that resulted from a systematic and prospective screening of newborns and children at risk.

Prevalence in the General Population—Different Geographic and Racial/Ethnic Populations

The included prevalence studies demonstrated important differences in execution, tests for prevalence assessment, and in patient sampling, making pooled estimates of prevalence unreliable. Furthermore, the discussions regarding the operational characteristics of the serological tests themselves, the influence of disease prevalence on the PPVs and NPVs of these tests, and the criteria by which clinical and histological CD is defined, have to be kept in mind when considering the results of this section. The last point regarding the histologic definition of CD is particularly important in this setting, since one-third of the included studies did not seek histologic confirmation of serology diagnosed CD, and in another four studies, a large proportion of the serology-diagnosed patients did not undergo histologic confirmation. Finally, because of the previously discussed concerns regarding the sensitivity of serological tests in lower grade histological lesions, and the potential for missing true CD patients based on histologic criteria that require villous atrophy, the true prevalence of CD in the general population may still have been underestimated in these studies.

With these points in mind, the results of this report suggest that the prevalence of CD in the general unselected populations of North America and Western Europe is quite high and likely falls within the range of 0.5% to 1.26% (1:200 to 1:79). Smaller sample-size studies tended to give wider estimates ranging from 0.17% to 2.67%. Among the studies from the US, the range of prevalence was 0.4% to 0.95% in adults, and 0.31% in children. In Italy, the range of prevalence was between 0.2% and 0.8%, whereas the Scandinavian countries, Ireland and the UK, tended to show a higher prevalence of CD of approximately 1.0% to 1.5%, although there were also studies from those same countries that showed a lower prevalence.

In summary, the prevalence of CD in Western populations is likely close to 1% (1:100) and may be higher in Northern European countries. A firm estimate of the prevalence is impeded by between-study differences, and uncertainties regarding the performance of serological tests at these relatively "low" prevalences, compared with the 40% to 60% prevalences in the studies of the diagnostic characteristics of these same tests (Celiac 1).

Prevalence of CD in Patients with Suspected CD

The prevalence of CD is greatly affected by the study population. In populations where the diagnosis of CD is clinically suspected, either because of the presenting symptoms or the presence of associated conditions, its prevalence varied between 1.1%[307] and 50%.[301] This illustrates well how the patient selection process will influence the prevalence of the condition—studies reporting very high prevalence had populations that originated from tertiary, referral centers, while studies reporting low prevalence had populations that tended to originate from general practice. Although the report of the large American study of CD prevalence in at-risk and not-at-risk individuals did not specify how their subjects had been gathered,[206] we can assume that these were derived from community practices, considering their large number.

Altogether the variations between the study populations, the diagnostic criteria and the study design were such that it was inappropriate to statistically combine the observed prevalence to obtain a summary measure. Nonetheless, considering studies with subjects who were not originating from a specialized referral centre, the observed prevalence of CD in subjects with symptoms or conditions associated with CD ranged between 1% and 4%.

Prevalence of CD in Patients with Type I Diabetes

The findings of this report suggest that the prevalence of CD in patients with type I diabetes is higher than the prevalence in the general not-at-risk population. These findings appear to be consistent across the studied age groups, and by the screening method. Although the magnitude of the risk of CD among patients with diabetes varied to some degree from study to study, many of these differences can be explained by issues of study design. An overall pooled estimate of the prevalence of CD in diabetes could was not calculated due to these study differences.

Almost uniformly, the prevalence of CD by biopsy was to some degree lower than the prevalence by serology. This may reflect the fact that there were some false-positive serology results in the prevalence of CD seen in these studies. Additionally, all these studies used some degree of villous atrophy to make a diagnosis of CD, which may underestimate the true biopsy prevalence of CD, since CD patients with Marsh I or II lesions were not considered. The prevalence by biopsy seemed to be lower still in studies that require subtotal or greater villous atrophy to make a diagnosis of CD. Furthermore, the prevalence by biopsy was uniformly low, as would be expected, in studies in which a large proportion of the screen-positive patients did not undergo biopsy. In these studies, the prevalence by biopsy was typically less than two percent, which likely represents an underestimation of the true prevalence of CD in this population.

The prevalence of CD by serology varied greatly with lows near 1% and highs close to 12%. However, the majority of studies, and particularly those using EMA or tTG, demonstrated prevalences in the range of 4% to 6%. Although the prevalence by biopsy also varied, the typical study with complete biopsy confirmation of serology-positive patients demonstrated prevalences in the range of 3% to 6%.

This evidence report has gathered the reported studies examining the relationship between diabetes and CD. Baring in mind the limitations noted above, we believe there is sufficient evidence to show individuals with type I diabetes are at higher risk of CD. The prevalence of CD in this population is likely between 3% and 6%.

Prevalence of CD in Relatives of Patients with CD

The prevalence is CD in relatives of patients with CD is elevated, both in first-degree and second-degree relatives. That prevalence varied between 2.8%[246] and 17.2%[235] in first-degree relatives and between 2.6%[206] and 19.5%[235] in second-degree relatives. The prevalence remains elevated among first cousins, and was 17% in the only study of these subjects.[235]

We have identified several factors that can be responsible for the variation in the observed prevalence. In particular, the selection of the families, of the relation to the index case, the diagnostic criteria, and the choice of study design.

The prevalence of CD appears to be generally higher in families with multiple known cases, such as reported by Book et al.[235] and Mustalahti et al.[241] Most other studies referred to their subjects as originating from a "CD family," without systematically documenting the proportion of families with multiple known cases of either CD or DH.

As expected, in studies that looked at various degrees of relation, the risk was greatest in the first-degree relatives.[206,235,239] However, Book et al.[235] found no difference in prevalence

between second-degree relatives and first cousins, i.e., 19.5% (95% CI: 15.1-23.9) and 17.0% (95% CI: 6.4-27.7), respectively.

Also, the age of the screened population might be a factor even beyond infancy, since it has been observed by prospective serological[248] and histological[237] follow-up studies that the serological and histological markers of CD can develop after an initial negative screen in a genetically predisposed individual. Therefore, a one-time assessment or screen in these individuals may be insufficient.

The serological diagnosis of CD will be affected by the diagnostic accuracy of the test. Fortunately, 11 out of 12 studies that used serological screening were EMA-based, a test with good diagnostic accuracy in populations with relatively high prevalence, such as relatives of CD patients. The single non-EMA study[236] used AGA, a test with a lower sensitivity and specificity than EMA, but all seropositive subjects underwent a confirmatory intestinal biopsy.

The histologic diagnostic criteria also affect the reported prevalence, as was well illustrated by the study by Tursi et al.,[249] where Marsh grades of I and II were also considered diagnostic, resulting in a prevalence of 44.1%.

The study design, especially whether all at-risk individuals are biopsied as opposed to solely those that satisfy a non-invasive criteria, is also to be considered. The EMA-based serological tests can miss milder forms of enteropathy as has been discussed, and this may explain why the prevalence of CD was generally higher in studies where all identified relatives were biopsied.

Prevalence of CD in Patients with Anemia

The results of this report demonstrate an increased prevalence of CD in patients with IDA. The prevalence is highest (between 10% and 30%) in studies of patients with GI symptoms, or in patients who have no gross lesions seen at initial investigation. CD appears to also be common in premenopausal women, both with (4.5%) and without (33%) heavy periods. Overall, in asymptomatic IDA patients assessed by serology or biopsy, the prevalence of CD was between 2.3% and 6%. Therefore, patients with IDA, particularly those without a clearly identifiable cause, should be evaluated for CD as part of their investigation.

Prevalence of CD in Patients with Low BMD

The studies of the prevalence of CD in patients with low BMD suggest that between 0.9% and 3% of patients with osteoporosis have CD. As a comparison, Fasano et al.[15] found that in the United States 0.75% of the general not-at-risk population, and 4.55% of first degree relatives of CD patients were found to have CD.

The results from these studies should be interpreted within the context of some methodological limitations. Three of them used AGA as the initial screening test to prompt further investigation, and we have shown that the sensitivity of this test is not high. Furthermore, the biopsy criteria used to define CD was either not reported, or required the presence of subtotal, or greater villous atrophy (Marsh IIIb or greater). We have also shown that CD exists in patients with lower grade histological lesions. Furthermore, the study results are contradictory. Two showed a risk of CD higher than the general population,[296,298] while the other two did not. In particular, the study by Mather et al.[297] found that seven out of the 96 screened patients were positive for EMA-ME, but none of these were positive on biopsy. From what we have seen regarding the specificity of this test being close to 100% (and therefore the

PPV would be expected to be high as well), it is unlikely that there are so many false positives even if the prevalence of CD was low, and raises the question of whether early grade CD patients remained undiagnosed. As such, it is difficult to draw any firm conclusions about the true prevalence of CD in this population, given the contradictory results, the fact that lower grade lesions were not considered, and that no follow-up data was provided on the patients who screened positive for serology but did not meet the biopsy criteria. Taking into account these limitations, it is likely that the prevalence of CD in patients with osteoporosis is higher than that in the general population.

Celiac 3: Risk of Lymphoma in CD

The association between malabsorption and lymphoma is a concept that has evolved over the past century. The observation that a significant proportion of patients with intestinal lymphoma also had villous atrophy at a distance from the malignancy, or had previously been diagnosed with CD, led to the publication of several series on the topic.

Although the objective of the task order was not to determine the risk of CD in lymphoma per se, the broad coverage of our search strategy also allowed us to systematically appraise the literature on this question, and were able to identify only two controlled studies on this association, which we describe here.[454,455]

Johnson et al.[455] performed a retrospective search of the five main pathology laboratories serving Northern Ireland to identify all the incident cases of small bowel lymphomas (SBL) and small bowel adenocarcinoma from 1987 to 1996. The clinical presentation of the cases, as well as the presence or absence of villous atrophy at a distance, were noted. The prevalence of CD in this group of SBLs was compared with that of the general population in Northern Ireland, as observed from serological screening of the population at large.[188] There were 13 cases of CD (gender not reported) out of 69 cases of SBL, all of which were ETCLs. Only one out the 13 CD cases was known to have CD prior to the diagnosis of SBL. The OR of CD in SBL was 27.98 (95% CI: 11.88-65.81) compared with the general population. The OR of unrecognized CD in SBL was 15.72 (95% CI: 9.71-25.45) compared with the general population.

In a prospective multicenter Italian study conducted between 1996 and 1999, Catassi et al.[454] screened newly diagnosed adult patients with NHL for CD using EMA and AGA testing; EMA-positive or IgA-deficient patients underwent small bowel biopsy. There were six cases of CD out of 653 patients with NHL (prevalence 0.92%). Three had B-cell and three had T-cell lymphomas. Four out of six cases had lymphoma primarily located in the gut. Two patients were known to have CD for more than 1 year, one of whom was poorly adhering to a GFD. Two cases had been diagnosed with CD within 1 year of the diagnosis of NHL, whereas two other cases had no prior CD diagnosis. The prevalence of CD among these NHL patients was compared with that observed in two Italian studies which performed large scale screening for CD.[126,222] The OR of CD in NHL was 3.1 (95% CI: 1.3-7.6) compared with an age-and sex-matched population.

These observations point to a clear association between CD and lymphoma. To determine the degree of association, or to quantify the risk of lymphoma in CD, we searched the literature for controlled studies of the incidence of lymphoma in CD. Unfortunately, the majority of publications on lymphoma in CD were uncontrolled. Typically, patients diagnosed with CD in a single institution were followed over time and the incident cases of lymphoma were described,

along with characteristics of the affected patients, the course of their CD and the histological type of lymphoma. Unfortunately, such studies provide little confidence to estimate the true risk of lymphoma in CD, since lymphoma per se will occur in the general population. The incidence of lymphoma has to be compared with "controls," matched on various characteristics such as age, sex, period and population. Any study that did not adjust the observed incidence to the expected incidence for age- and sex-matched individuals of the same population was deemed uncontrolled and excluded.

Cohort studies, either prospective or retrospective, constituted the majority of controlled studies. The incidence of lymphoma in a cohort of biopsy-proven CD patients, calculated as the number of lymphomas divided by the number of patient-years of follow up, was compared with that of an age- and sex-matched population from the same geographic area and time-period.

The SIR therefore represents the likelihood of lymphoma in CD patients relative to those who do not have CD in the same population. The value of the denominator reflects the incidence of lymphoma in a given population, so that it is not possible to pool SIR's from different populations.

The AR, however, is a measure of association that provides information about the absolute excess risk of disease in CD patients compared with "non-afflicted" individuals. This measure is defined as the difference between the incidence rates in the CD patients and normal population and, in a cohort study, can be calculated as the difference of cumulative incidence (risk difference) or incidence densities (rate difference) depending on the study design. The AR is a measure of risk which can be pooled; however, since incidence rates were reported in only two studies, we had insufficient data to generate a representative summary statistic.

Furthermore, studies varied greatly at several levels, in particular with respect to the definition of an incident case of lymphoma, the reported outcome measure, and the CD population selection.

Studies differed in their definition of observed cases of lymphoma, in the following manners:

1. Inclusion of malignancies that antedated the diagnosis of CD. In one American study, the number of at-risk years was calculated both from the time of CD diagnosis and from the time of onset of symptoms that could be attributed to CD.[340] In a prior national survey to patients with CD,[456] these authors had collected evidence to support that there is usually a long duration of symptoms before a diagnosis of CD is made in the United States, so that they considered this account justifiable. However, authors from other countries would specifically exclude the malignancies that were diagnosed prior to CD, assuming that it was unknown whether these were truly "at-risk" periods and that this account could falsely inflate the incidence of lymphoma in CD.[333] Considering that publications uniformly calculated and reported the incidence ratio based on the time period from the CD diagnosis, this is the measure of risk that we selected.
2. Inclusion of malignancies that were recognized simultaneously to the diagnosis of CD (i.e., within 1 to 12 months of diagnosis). In some cases, the diagnosis of CD can be unknown until the presentation of lymphoma. This fact highlights the possibility that lymphoma can occur in asymptomatic patients with CD. Although the importance of such cases is undeniable, the account of such cases can introduce bias and inflate the incidence of lymphoma in CD. In other words, the simultaneous diagnosis of CD and lymphoma is similar to an incident case in a patient with a "zero" duration of follow-up, i.e., is closer to a measure of prevalence than incidence. The inclusion of cases of

lymphoma occurring in patients with previously undiagnosed CD should theoretically be related to all cases of CD, diagnosed and undiagnosed, in order to give an accurate estimate of incidence, which is obviously impossible. However, some studies chose to include such cases, while others excluded them from the incidence calculation. This distinction was noted in the results presentation.

3. Exclusion of malignancies that were diagnosed incidentally at autopsy. In their large Swedish cohort of individuals hospitalized with CD, Askling et al.[337] also excluded unsuspected autopsy diagnoses of lymphoma, assuming that such entities would have been silent during life, and that they therefore could not be controlled for in the comparator group.
4. Case definition of lymphoma. Lymphomas are broadly categorized as Hodgkin's lymphomas and NHLs. The lymphomas that have been associated with CD have typically been of the NHL type, and so the majority of studies sought cases of NHL, with the exception of the Scottish study from Logan,[336] where both Hodgkin's and NHLs were reported.

The reported outcome measures also varied and impaired our ability to combine observations. Some studies reported the incidence of lymphoma, while others, relying on death certificates for ascertainment of outcomes, reported on the mortality from lymphoma.

Finally, the patient selection also varied, along with the reporting of the circumstances that led to the diagnosis of CD. These factors limited our ability to draw conclusions on the risk of lymphoma in symptomatic versus asymptomatic patienst with CD.

We were also unable to find controlled data on the risk of lymphoma in refractory CD, an objective which had been suggested by the TEP. We did find, however, two prospective studies and one retrospective study that could lend support to the notion that the risk of lymphoma in refractory CD is greater than that of responsive CD.[457-459]

In the Netherlands, Wahab et al.[457] prospectively followed 158 biopsy-proven CD patients to assess the recovery of histological changes with a GFD over time. There were 11 incident cases of refractory CD with more than 5-years of follow-up, five of whom developed ETCL, in contrast to none of the remaining GFD-responding CD patients.

Goerres[458] reported on 18 patients diagnosed with refractory CD between 1998 and 2000, gathered from all over the Netherlands, whom they treated with azathioprine and prednisone. There were three men and 15 women, with a mean age of 58 years (range 39-82). Subtypes of IEL populations were analyzed by flow cytometry, allowing for the classification of refractory CD patients into two types: type I refractory CD (n=10), in which a normal IEL population is seen, and type II refractory CD (n=8), in which an aberrant IEL population is present. All of the patients with type I refractory CD responded to combined azathioprine-prednisone therapy, whereas none of the patients with type II refractory CD showed a response. In fact, six of the eight patients with type II refractory CD developed EATL within a 3-year period, and a seventh patient died with blastic T-cell-like cells in the small bowel and the liver, and myeloproloferative changes in the bone marrow. The authors concluded that type II refractory CD is a premalignant condition with a very poor prognosis.

In a French national cooperative study, the clinical information and tissue specimen necessary for IEL subpopulation analysis were gathered from 21 patients diagnosed with refractory CD between 1974 and 1998.[459] There were five men and 16 women, with a mean age of 51 years (range 29-73 years). Nine of the 21 patients (43%) died from severe malnutrition

and/or lymphoma (three patients) after a mean of 6.7 (range 1-14) years after the onset of symptoms of refractory CD. A phenotypically abnormal IEL population associated with evidence of clonality was found in eight of the nine patients that could be tested. The authors suggested that refractory CD may be the missing link between CD and ETCL.

This systematic review identified nine controlled studies that met inclusion criteria. The major observation of our review is that the risk of lymphoma in CD was significantly increased compared to an age-matched population from the same region and period in 8 out of 9 studies. The SIR (NHL) varied from 2.66[338] to 42.7,[333] whereas, the SMR from NHL or lymphoma in CD varied from 11.4[337] to 69.3.[339] This increased risk persists even when the cases that are diagnosed with lymphoma simultaneously or within 1 year of the diagnosis of CD are excluded from the calculation.

Some observational studies suggest that the risk of lymphoma, relative to patients of the same age without CD, may be highest in individuals who were diagnosed during adulthood,[336,337] and appears to decrease with adherence to a GFD, as shown by several authors.[333,336-339] It is also interesting to note that the only study that did not report a significant increased risk of lymphoma was one where 75% of patients were on a strict GFD.[338]

The differential risk of lymphoma among patients diagnosed with CD in adulthood versus childhood may indicate that early diagnosis and treatment with a GFD is protective. The possibility that a GFD may be protective is also supported by Askling et al.[337] who found that the risk of lymphoma dropped to unity after 15 years of follow up. Limitations in the designs of these studies, however, prevents firm conclusions. These studies have followed relatively few patients diagnosed as children through middle age when the risk of lymphoma rises, and they may not have accounted for other factors (severity of symptoms, or other marker of disease activity) which might affect risk. The distinction between childhood and adult diagnosis of CD in the published cohorts relies on the presence or absence of CD-related symptoms during childhood, which has historically been a key factor in CD diagnosis. Based on the observations from these groups of patients, it would seem that continuous gluten exposure and ongoing mucosal damage sets the stage for malignancy later on in life. It remains unclear, however, why some individuals would have persistent mucosal damage in the absence of symptoms. Would these individuals also carry other characteristics that modulate their risk of malignancy? As we tap into the base of the "celiac iceberg" through systematic screening, we will hopefully in the future be able to observe the incidence of lymphoma in child and adult CD populations who were identified through population screening, and placed on a GFD despite them being asymptomatic during that period of their lives. The notion that lymphoma arises from prolonged antigenic stimulation should be confirmed if the risk of lymphoma is, as expected, lower than historical CD cohorts in those individuals.

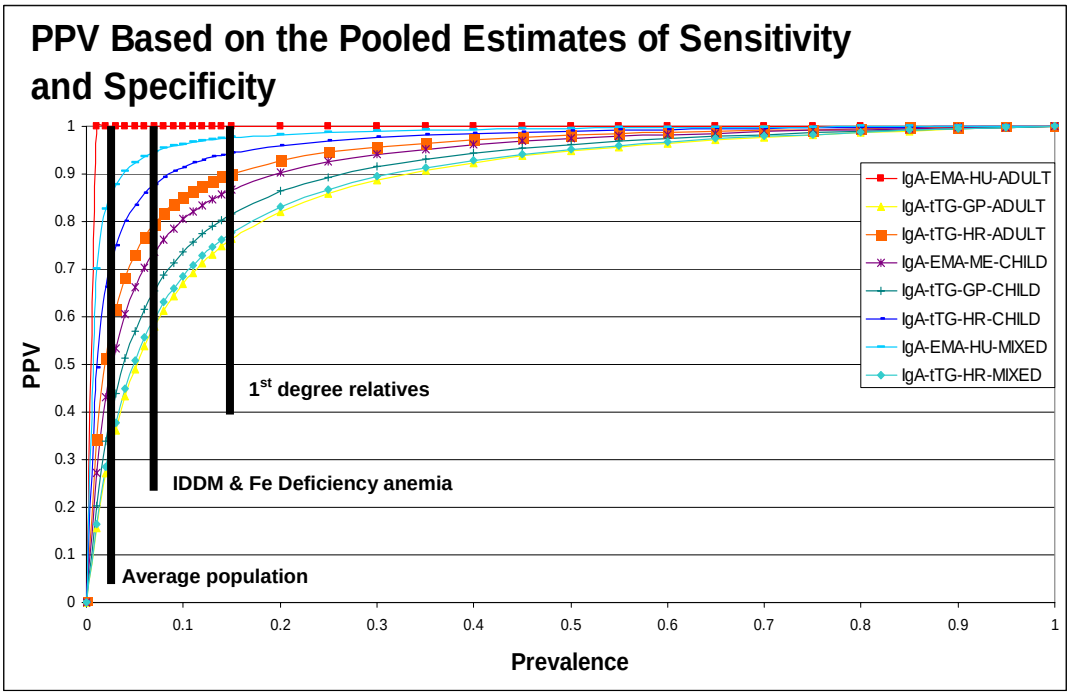
Celiac 4: Consequences of Testing for CD

The search strategy did not identify any studies that would allow us to address the specific benefits and harms of testing with different strategies for CD. At present, there is inadequate information from the published literature on the benefits and harms of screening and the potential risks of undetected CD. Prospective trials of screening would be helpful to provide the data necessary to construct the tables that depict the consequences of screening specific populations.

Information on the consequences of screening will come from the currently ongoing large population based prevalence studies.

The consequences of such issues as false-positive results were dealt with in the Celiac 1 Discussion. As discussed in that section, the definition of CD used and the prevalence of CD in the test populations, have a great impact on the diagnostic parameters of the available tests. We have presented data that show that the sensitivity of the available tests declines considerably when applied to patients with low-grade histological lesions. Unfortunately, there is insufficient data to address the question of what is the consequence of missing patients with low-grade histological lesions if serological screening alone is used. As described in Celiac 1, all the diagnositic test studies of the various serological markers were undertaken in study populations in which the prevalence of CD exceeded the that observed in most clinical situations. We have shown that the positive predictive value, which is predominately influenced by the test specificity and the prevalence of CD in the test population, drops from the reported values to much lower values when the test is applied in typical clinical populations. To illustrate this point, Figure 31 highlights the expected PPV when applied to different test populations.

Figure 31: PPV based on pooled estimates of sensitivity and specificity



As can be seen from Figure 31, the PPV—the probability that a positive test result actually represents true CD—drops with the prevelance of the population in which the test is applied. This relationship holds true for all the summary curves, but differ in degree. It is important to note that the PPV is predominantly influenced by the specificity of the test and prevalence. Since we have identified that the specificity of EMA and tTG is quite high, the major influence on the PPV in these analyses is the prevelance of CD in the population being tested. The practical importance of this discussion, is that despite having very high specificity, the use of these serological markers in low-prevalence populations would be expected to result in high false-positive rates. Below a prevalence of 5%, the false-positive rates may be as high as 30% to

50% based on our estimates. This may seem counterintuitive, given that the specificity is greater than 95% and close to 100% in some cases. One must keep in mind that unless the specifity actually equals 100%, the prevalence of CD will influence the PPV. As the specificity approaches 100%, the influence of the prevalence decreases. The same interplay occurs between the negative predictive value (the probability that a person with a negative test does not have CD), and the sensitivity of the test. However, in this case, the NPV rises as the prevalence of the disease falls (see Celiac 1 Figures). Given that we have identified that EMA and tTG have a sensitivity in the range of 95%, the NPV would be expected to be very high (>96%), particularly in low-prevalence populations. This would mean that the false-negative rates with these tests are less than 1% to 4%. These data would then suggest that a negative test result would have a high probability of being a true negative result, but that a positive test would have to be considered in light of the expected prevalence of CD in the tested population. If the expected prevalence is in the range of 10% or lower, then the possibility that the result represents a false-positive should be considered. Lastly, one must not forget the discussion regarding the true sensitivity of these serological markers when lower grade CD lesions are considered. The studies by Rostami et al.[16] and others, suggest that the sensitivity can be lower than 80%. In fact, both Rostami et al.[16] and Tursi et al.[424] suggest that the sensitivity for grades less than Marsh IIIa, is in the range of 30% to 40%. If this is the case, then the nearly perfect NPV discussed above would be expected to fall, particularly in groups with a higher prevalence of CD. For example, if the sensitivity was really 75%, then the NPV would drop to 88% (12% false negatives) if a population of patients with suspected CD was tested. However, because of the strong influence of a low prevalence (<15%) on the NPV, the NPV will remain higher than 90%, as long as the sensitivity of the test is greater than 50%.

Expected Outcomes of Treatment of CD

The four studies of diabetes and CD in children/adolescents that evaluated the impact of a GFD found that body composition parameters improved on the GFD, but HbA1c levels did not improve. Some studies observed an increase in the insulin requirements after introduction of a GFD, which could be explained by improved absorption of nutrients.

The results of studies on anthropometrics and body composition in CD patients are variable due to differences in populations, and methods used to evaluate body composition. Overall, weight and BMI improves after starting a GFD. Individuals with CD may have a lower BMI when compared with controls because of lower daily energy intakes, particularly in those who strictly follow a GFD.

A few small studies have evaluated the impact of the diet on nutritional parameters in newly diagnosed symptomatic CD patients. These studies found that nutritional status does improve in the majority of subjects with CD on a GFD. Certain biochemical parameters such as ferritin may take longer to normalize. There is evidence that the recovery of nutritional status is linked to improvement of villous atrophy. Larger studies of nutritional status in those with classical and silent CD patients and the relationship of biochemical values to changes in histological grade on small bowel biopsy and compliance with the GFD would be helpful.

Compliance with the GFD was assessed in adolescent populations in three studies and the results varied. Compliance with a strict GFD was greater in those who were symptomatic, compared with those who were diagnosed via a screening program. Another study in adults by Ciacci et al.[460] looked at the correlation between intestinal biopsy and compliance (assessed by

dietary interview) and found that that intestinal damage was significantly associated with dietary compliance. Low or very low compliance with a GFD had a PPV of 92.8%, and good compliance had a negative PPV of 96.8%. This study also suggested that those with more severe symptoms at diagnosis were more likely to have better compliance. Given the poorer compliance in those without symptoms, different strategies to promote adherence with the GFD may need to be developed if screening for CD is promoted.

The justification for screening the general population for CD would be strengthened by well-conducted comprehensive cost-effective analyses. Only one study[360] appeared to include the majority of the components that have been recommended for the reporting of cost-effectiveness analyses (CCOHTA, Guidelines for Economic Evaluation of Pharmaceuticals: Canada, 1997). None of the analyses incorporated the use of health related quality of life or utility assessments.

Fractures/BMD/Osteoporosis/Osteopenia

There were a number of methodological limitations in the studies that examined bone-related consequences of CD. Limitations included: selection of representative cases and controls, ascertainment of the outcome and failure to identify and control for relevant co-interventions such as calcium and vitamin D.

The issue of whether fractures are increased with individuals with CD appears to be somewhat controversial based on results of the included studies. Both Thomason et al.[394] and Vestergaard et al.[388] did not find increased fracture rates for CD subjects, whereas, the recent population-based study by West et al.[385] did find an increased rate of fractures. This is an important issue to clarify since osteoporotic fractures are one of the key reasons for promoting strict adherence to the GFD and for making decisions about screening. In some studies, the sample sizes were small and may not have been large enough to detect an increased risk in fractures in subjects with CD relative to controls. In addition, methodologies and study populations varied, and not all studies controlled for duration of CD. Moreno et al.[392] found that the risk of fracture in subclinical and silent cases of CD was not significantly different from that of controls. Overall, the risk of fracture seemed to increase with age as one would anticipate and may be greater in those patients who were clinically symptomatic. Based on results of current studies, the risk of fracture appears to be highest prior to diagnosis of CD and diminishes once individuals are on GFD. This latter finding would be consistent with the increase in BMD that is seen after 1 year on a GFD. Additional population based fracture studies would be useful to clarify the relative and absolute risk of fracture in CD and to determine if it differs in asymptomatic cases.

Overall, the studies consistently documented an increased prevalence of osteoporosis/osteopenia in newly diagnosed patients relative to controls. There was a significant increase in BMD, especially within the first year of being on a GFD. Some of the variability in the results could be attributed to proportion that were compliant with the diet and use of co-interventions such as calcium and vitamin D. Moreno et al.[392] found that the lumbar spine BMD did not differ in groups according to clinical presentation, but they did find a significantly lower T score of the femoral neck BMD in classically symptomatic cases versus subclinical or silent cases. Mustalahti et al.,[378] however, found that BMD in the spine was lower in asymptomatic cases.

Based on the two studies in children,[352,377] BMD appears to normalize in children after treatment with a GFD. The normalization of BMD in children would support the need for early

diagnosis of CD and treatment. However, in children skeletal growth may affect BMD, with some of the change relating to changes in growth. Most studies of BMD in adults on a GFD have found that the BMD is still reduced at all sites when compared to normal controls. One study suggested that those without secondary hyperparathyroidism at time of diagnosis may normalize their BMD, but this finding was not replicated. A large BMD study with baseline and follow-up small bowel biopsy data, and documentation of clinical presentation, percent compliance with the GFD and adjustment of co-interventions is recommended to give us accurate information on bone-related consequences of CD.

Mortality

The majority of observational studies have demonstrated an increase in overall mortality rate (SMR of 2 or greater) in subjects with CD when compared with the general population. The increase in mortality can be attributed to deaths from malignant diseases, respiratory, and digestive diseases. The increase in mortality appears to be greatest within the first 3 years after diagnosis and declines over time. The mortality rate seems to increase with longer delays in diagnosis and poor adherence to the GFD. Perhaps one of the most important points from the Corraro study,[362] is that the mortality rate was not increased compared to the general population for those individuals who had mild symptoms or were asymptomatic. This latter result has potential implications for population screening for CD.

Celiac 5: Promoting or Monitoring Adherence to a GFD

Monitoring Adherence to a GFD

Some of the same concerns expressed in the other celiac objectives, regarding clinical definitions, histological criteria, and the performance of the serological tests, are repeated when the results of the studies on monitoring adherence to a GFD are considered. Foremost in facilitating the interpretation of these studies is the question of what to consider as the histological criteria to define recovery on a GFD. Certainly normalization to Marsh 0 would constitute recovery, but what about improvement to Marsh I or II, or even accepting Marsh IIIa? The distinction has important implications for assessing the strength of the correlation between histological and serological improvement, and in this regard, different studies have adopted different cut-offs.

It is clear from the presented studies that improvement of symptoms does not offer an accurate assessment of adherence to a GFD as judged by interview or by biopsy. This point is illustrated in the study by Kluge et al.[461]. In follow-up of 18 adult patients with CD, all patients felt well and appeared to be clinically in remission. Nonetheless, only 17% of the patients reported being on a strict GFD. Biopsy assessment of eight patients showed six with total villous atrophy including one patient who reported strict adherence to GFD. The remaining two patients did not have villous atrophy but the mucosa was not normal, including an excess of IELs. Thus, small amounts of gluten may provoke a histologic change without clinical symptoms which may be an important reason why adherence to GFD may be less than perfect. In other words, non-compliance does not necessarily translate into noticeable consequences for the patient.

Furthermore, it is increasingly recognized that most CD patients don't have symptoms, so reliance on symptomatic improvement is clearly not adequate.

There is good evidence that mucosal recovery following institution of GFD is slower and more incomplete than previously assumed, especially in adults.[405,411,414] Whether this slow recovery is due to dietary transgression, inadvertent gluten intake or whether this is simply the natural history of the disease is less clear. This has definite implications for the interpretation of both biopsy and serology results in monitoring adherence to GFD, particularly in the short run.

With the advent of the newer and more sensitive serologic tests for CD (EMA, tTG), the possibility of a reduction in the need for follow-up biopsies and a move towards non-invasive serological monitoring has been proposed. The question arises as to whether serology can detect dietary transgressions and reasonably mirror histological improvement on a GFD.

A number of studies show that values of serologic markers will fall with increasing duration of GFD, whether one looks at IgA-AGA, IgA-EMA, or IgA-tTG. As well, several studies suggest that in both adults and children, increasing degrees of non-compliance with a GFD, are more likely to be associated with positive serologic tests.[396,402,408]. The question, however, is *not* whether serology can pick-up major transgressions such as with a gluten challenge which it is clearly capable of assessing,[400,404] but rather if serology can pick-up milder degrees of dietary non-compliance and reasonably reflect histological status. A high rate of falsely-negative serology with lesser degrees of dietary transgression would diminish serology as a means of accurately monitoring adherence.

In both adults and children, the sensitivity of serology for picking-up dietary transgressions based on interview or self-reporting is disappointing.[401,402,410,415] One conflicting study[412] showed a good correlation between serology and adherence. This likely reflects the way patients were categorized, and it is likely that in this study, patients with lesser degrees of dietary transgression were categorized as compliant. In general, there is a significant rate of normal serology in patients identified as not adhering to a GFD. Furthermore, evidence from several studies suggests that serology, regardless of the actual test used, does not adequately reflect the mucosal state in adults.[398,403,407,409,409,413] Surprisingly, it seems that serology may be normal, not only in Marsh I or II lesions, but also when there is villous atrophy present.[398,407,409,413] Although the specificity of various serologic markers for villous atrophy seems better than sensitivity,[398] the NPV of serology would suggest that a negative test does not offer high assurance of the absence of villous atrophy.

As discussed earlier, mucosal recovery can be a slow process. It may be that serologic markers may better reflect histology in long-term follow-up. Certainly, in the range of follow-up of these studies (6-30 months), serology may be negative despite villous atrophy. There is evidence that even in longer follow-up, serology does not accurately reflect adherence.[398,402,410]

In younger patients, IgA-AGA and IgA-EMA-ME may better represent the mucosal state.[397,415] These studies are in keeping with the impression that in children and adolescents, mucosal recovery is faster and more complete. In children, serology seems to be a better marker of the absence of villous atrophy. Still, serology may be negative in the face of lesser degrees of histologic abnormality without villous atrophy.[397] The significance of such lower-grade biopsy abnormalities, although, is unclear.

It is possible that IgA-AGA may rise faster with non-compliance to GFD than other markers.[396,400] However, there is little direct evidence to show superiority of one serologic test over another in monitoring adherence.

Perhaps an important question that arises from this discussion, with particular relevance to symptomatic CD patients, is: "is it good enough for CD patients to show symptomatic improvement and a corresponding fall in, or normalization of, a sensitive serological marker without need for 'normalization' of the intestinal mucosa?" Unfortunately, this question is not an easy one to answer since many of the outcome studies in CD, particularly for lymphoma and mortality, did not specifically address differences in histologic grade. Furthermore, we identified no clear evidence suggesting that refractory sprue was the result of dietary indiscretion as opposed to a different spectrum of CD. Nonetheless, histological improvement appears to be important. For example, one study[356] demonstrated that osteoporotic patients with CD on a GFD who had Marsh III lesions had lower median Z-scores than those with grades less than Marsh III, while another study demonstrated a significant correlation of nutritional status measured by histomorphometric index, with the severity of the histological biopsy grade.[346] In the former study as well as one other study,[358] histologic grade correlated with degree of IDA, all suggesting that the goal of monitoring should be to assess degree of histological improvement.

It can be concluded that the return of serologic markers to normal is associated with duration of GFD and degree of patient compliance. Unfortunately, the correlation remains imperfect, especially in adults, and seems to reflect gross rather than minor degrees of dietary transgressions. Serological tests seem to have a higher specificity than sensitivity for dietary transgressions. It is recognized that this area is controvercial and that clinicians are moving away from routine follow-up biopsy as a means to assess dietary compliance. It seems reasonable to suggest that improvement in clinical parameters, and disappearance of serological markers would be an adequate measure of response to a gluten free diet. In children, because of their faster and more complete mucosal recovery, this strategy of using serology may be an appropriate means to monitor adherence. In adults, however, the situation is somewhat more complex. Therefore, while serology certainly can be an adjunct means to monitor adherence to a GFD, consideration should be given to assessing histological improvement since some evidence exists to suggest that mucosal improvement to at least below a Marsh III appears to be important from an outcomes perspective. If biopsy is to be utilized as a means of assessing adherence to a GFD in adults, the timing of the biopsy needs to take into consideration the slower mucosal healing in adults, and should therefore be performed after 1 year to 1.5 years of a GFD.

Interventions to Promote Adherence to a GFD

Changes in dietary habits are difficult to attain and maintain. The barriers to compliance are many. No interventions to promote compliance with GFD have been studied and found to be effective. Adding to the difficulty of assessing any proposed intervention is the lack of certainty as to how best to measure GFD compliance.

The existing evidence suggests a positive correlation between parental socioeconomic status, education, knowledge of CD, and the compliance of their children.[416,418] Compliant children may also have a better knowledge of CD[420] than those children who are non-compliant. Improved knowledge in adults also appears to correlate with compliance.[419] It is, therefore, not unreasonable to suggest that interventions designed to improve knowledge about CD in general, and about GFD, and specifically how to identify gluten-containing products, would likely improve compliance with a GFD. Improving knowledge regarding gluten-containing food products and additives would also likely improve self-confidence in choosing gluten-free foods as suggested by Lamontagne et al.[419] Improved knowledge of outcomes of untreated CD may

also improve compliance. Such information interventions, however, would need to be prospectively evaluated to ensure that they perform as expected.

Membership in a local celiac society appears to be an effective means of promoting compliance with a GFD. This is not surprising since such organizations provide CD patients with not only improved knowledge regarding their disease, and the intricacies of the GFD, but also provide emotional and social support.

It is interesting that one study[417] has demonstrated lower rates of compliance in children detected by screen as compared with those diagnosed on the basis of symptoms. It seems logical that if there are no obvious detrimental symptoms from a gluten-containing diet, that children and likely adults will be less likely to be compliant. The authors speculate that since screen-detected patients had a higher mean age of diagnosis, compliance might be promoted by earlier identification. They speculate that earlier detection would avoid the difficulty of changing formed eating habits.

Is early detection of CD an effective intervention to promote compliance? It appears rational that it would be easier to follow a GFD if it were introduced at an earlier age. There are some interesting observations [417]that suggest that diagnosis in early childhood is associated with improved compliance.[421] Unfortunately, the issue of compliance in asymptomatic screen-positive individuals casts doubt on the positive downstream effects of screening asymptomatic populations for CD, particularly if the low-compliance rates in asymptomatic individuals can be reproduced in other studies.

In summary, it is suggested by the results of this report that a multidisciplinary approach to patient and parent education and support by physicians, dieticians, and celiac societies, possibly employing formal knowledge and decision support interventions that involve the patient (and parent) directly, are likely to improve compliance in individuals diagnosed with CD. Formal testing of interventions and programs would be valuable.

Strength of the Body of Evidence

Celiac 1

Overall, the quality of the diagnostic studies assessed in the Celiac 1 objective was quite good, due largely to our stringent inclusion criteria. However, 59% of the included studies reported using a selected patient population that may not be representative of a clinically-relevant population. This is likely related to study design. In addition, only 11% of the studies reported on whether the reference test was reported without knowledge of the index test. However, we felt that this was not a major threat to the validity of the studies.

Two other factors that affect the interpretation of these results, yet were not captured in the quality assessments, are the threshold effects for determining the positivity of a serological test, and the high prevalence of CD in these studies (see above). With these considerations in mind, the overall strength of the evidence is quite good.

Celiac 2

The overall quality of reports of the included studies in the Celiac 2 objective was found to be marginal to fair. For example, most of the studies did not report on whether the patients were consecutively enrolled, a factor that could contribute to selection bias. However, setting aside the quality of individual studies, from a policy perspective, the strength of the evidence is fairly good in that the study populations were selected to reflect that of a North American/Western European descent, that should reflect the demographics of the US population.

Celiac 3

The studies included in the Celiac 3 objective were found overall, to be of good quality. Again, the overall strength of the evidence is due largely to the stringent inclusion criteria, such as the requirement for the reporting of standardised rates for the outcomes based on rates from the local general population, and the overall good quality of the included studies.

Celiac 4

The majority of studies included in this objective were single group "before–after" studies, although some had in addition a comparative healthy control group. We could not identify any quality instruments for this type of study design and in general, this type of study is considered weak, particularly in the absence of a control group. Overall, however, the strength of the evidence for this objective is fair to good and suggests that the results can be used for policy decisions with the understanding that this area of CD research is still relatively new and requires further high quality studies.

Celiac 5

The majority of studies in this objective were also of a "before–after" design. However, in this setting, this design may not pose a major limitation, since the purpose of the study is to assess the change in serology and histology after introduction of a GFD. In this regard, the strength of the evidence for monitoring adherence to a GFD is fairly good. However, there is almost a complete absence of studies of interventions for the promotion of adherence to a GFD.

Future Research

This review has allowed us to identify several areas in need of future research. Perhaps the most important of these is a need for the development of a consensus on the definition of CD in the era of advanced serological testing. As discussed in the report, this distinction of what one calls CD has profound implications for each of the requested task order objectives. Do screen-positive patients without villous atrophy have CD. Certainly the preliminary evidence suggests that this is the situation in many cases. However, what is required is a new definition of a gold standard for the diagnosis of CD. This new gold standard may include a combination of serology, biopsy and HLA testing. Such a gold standard, when used in studies with a time dimension (e.g., response to a GFD or gluten challenge; extended follow-up), would help answer some of the uncertainties identified in this report including: the real performance of the serological tests when low-grade lesions are considered CD; the diagnostic performance of biopsy alone; the outcomes of patients with these low-grade lesions; and, those that would be "missed" using current screening strategies. Even in the absence of a new gold standard, we could not identify a well-conducted study of the diagnostic performance of the various serological markers when applied to an average population (i.e., one with a prevalence of CD in keeping with the range identified for average risk), with the entire cohort being investigated equally (i.e., all are biopsied). Such a study would at least be able to shed light on the performance of these tests in average-risk patients, and since all patients are biopsied, the relationship of histology to serology could be further assessed.

On a similar theme, we have identified multiple studies that suggest the importance of histological improvement on a GFD. This is a controversial area since in common clinical practice, clinicians are moving away from routine follow-up biopsy. It seems reasonable to believe that improvement in clinical parameters with loss of serological markers is adequate evidence of response to a GFD. In children, this issue may be less important since histological improvement is much more rapid and complete than in adults, and correlation with serology seems better. However, we have identified multiple studies in adults that suggest poor correlation between serology and improvement of histology on a GFD, and other studies that suggest that serology is useful for detecting gross dietary indiscretion, but not minor occurrences. Therefore, the question that arises is what constitutes adequate improvement on a GFD, and what are the criteria to define this improvement. Based on the lymphoma literature that suggests that this malignancy may arise from chronic antigenic stimulation and immune activation, what are the outcomes of adults with clinical improvement, yet persistent histological abnormalities? Are some histological features, such as reduction of mucosal lymphocytes, more important markers of improvement and possibly prognosis than other features such as villous height?

We feel that clarification of these fundamental questions is necessary for the conduct of future studies in all areas of CD, and in particular studies of the diagnostic tests and the outcomes in CD, since these are so dependent on the definitions discussed above.

Conclusion

This report has provided a systematic review on five broad areas of CD, with each of these areas including important sub-components. Perhaps one of the most important findings of this report is the understanding of the importance of how one chooses to define CD in the era of serological testing, and how this apparently clear-cut task has profound implications on all the results presented in this report. Specifically, can CD be diagnosed solely on the basis of serology? Is some degree of villous atrophy necessary for the diagnosis of CD? These questions have important implications downstream of the diagnosis as well. Do CD patients without symptoms or villous atrophy have the same risk of complications as those with villous atrophy? Is serological improvement on a GFD sufficient to reduce CD complications or must there be documented histological improvement, and what degree of histological improvement is necessary?

The results of the Celiac 1 objective suggest that in the era of EMA and tTG antibody testing, AGA testing in both children and adults has a limited role. The sensitivity and specificity of EMA and tTG are quite high (over 95% for sensitivity, and close to 100% for specificity), as are their PPVs and NPVs, but as previously discussed, one has to be aware that the reported diagnostic parameters are taken from studies in which the prevalence of CD was, for the most part, much higher than that seen in usual clinical practice and certainly the PPV of these tests may not be as high as reported when these tests are applied in general population screening. The bulk of the evidence on the diagnostic characteristics of these tests was derived from studies that defined CD as having at least some degree of villous atrophy. We have identified studies that suggest that the sensitivity of these tests drops, at times significantly, when applied to populations with CD with lower-grade histological lesions. This not only has implications regarding those patients with "mild" CD who were missed during screening efforts, but also puts into question the nearly perfect NPV of these tests.

HLA DQ2/DQ8 testing appears to be a useful adjunct in the diagnosis of CD. The test has high sensitivity, in excess of 90% to 95%, but because around 30% of the general population and an even higher proportion of "high-risk" subjects including diabetics and family members also carry these markers, the specificity of this test is not ideal. The greatest diagnostic utility of this test appears to be its NPV.

Biopsy itself, when used with a strict cut-off requiring villous atrophy, appears to have high specificity, but poor sensitivity. Using lower grade cut-offs clearly improves sensitivity, but because of the wide differential of causes of histological lesions similar to Marsh I to IIIa, the specificity suffers. The use of histomorphometric measures, such as quantification of $\gamma\delta+$ IELs, are likely to allow for the use of lower-grade cut-offs while maintaining reasonable specificity. Ultimately, a trial utilizing multiple diagnostic tests in an attempt to capture as many CD patients in a clinically-relevant population as possible, with a time dimension including a response to a GFD or gluten challenge, is required to fully assess the diagnostic characteristics of biopsy alone. This type of study would be able to characterize the false-positive and false-negative rates if all studied patients are followed forward in time.

The included prevalence studies demonstrated important differences in execution, tests for prevalence assessment, and in patient sampling, and their results also have to be interpreted in the light of some of the limitations that have been identified regarding the diagnostic performance of the tests for CD. Nonetheless, the results of this report suggest that CD is a very common disorder with a prevalence in the general population that is likely close to 1:100 (1%).

Several high-risk groups with a prevalence of CD greater than that of the general population have been identified including those suspected of having CD, family members of CD patients, type I diabetics, and those with IDA or low BMD. Additionally, the review identified multiple other high-risk groups such as those with Down Syndrome, short stature, and infertility, to name a few, though their inclusion was beyond the scope of this report. These results would suggest that at the very least, high-risk groups should be screened for CD. If the performance of the noninvasive serological tests can be verified in the relatively "low prevalence" situations in general unselected populations, then population screening may also be advisable, particularly if a greater understanding of the consequences of missing early low-grade CD can be obtained, and the issues of low-compliance with a GFD of asymptomatic screen identified patients can be addressed.

CD is known to be associated with GI lymphoma. The results of this report confirm this strong association, with the limitations indicated in the text. Nonetheless, the report identified SIR for lymphoma that ranged from 4 to 40, and SMR that ranged from 11 to 70. GI lymphoma is believed to arise as a result of chronic antigenic stimulation, which leads to the development of a clonal T-cell population with usually a refractory intermediate stage. We have identified epidemiologic data that supports this notion, and suggests that a diagnostic delay, and in particular diagnosis of CD in adulthood, as apposed to in childhood, is associated with poorer outcomes. Fortunately, several studies suggest that adherence to a GFD reduces the risk of lymphoma in CD patients. These findings underscore the importance of early diagnosis and treatment of CD.

The consequences of testing for, and identifying CD patients, is expected to have a positive impact on patient outcomes be it either from a reduced risk of lymphoma with early diagnosis and treatment of CD or from improvements in nutritional status, BMI, and BMD. The consequences of testing in at-risk and symptomatic patients appears to be more straightforward since these patients appear to be more compliant with a GFD and would be expected to benefit from this intervention. The data is less clear for asymptomatic screen-identified patients, particularly those who are truly silent and/or don't have fully developed villous atrophy since, on the one hand the outcome of such patients has not been extensively studied, and on the other hand, compliance with a GFD appears problematic, particularly for those diagnosed in adulthood.

Finally, no specific interventions have been identified that promote adherence to a GFD, but education of patients and family members about CD and about the intricacies of the GFD through multidisciplinary teams, and participation in local CD societies, has been show to improve compliance. Therefore, the development and evaluation of formal educational interventions in collaboration between healthcare professionals and CD societies would appear to be a means to build on the methods that appear to already improve patient compliance. Monitoring of adherence to a GFD appears to be important, since improvement in histologic grade has been associated with improved BMD, IDA, and nutritional status. The serological markers appear to be adequate for detecting gross dietary indiscretion, and responding to gluten challenge, but unfortunately, they have poor sensitivity for detecting lesser degrees of dietary indiscretion, and have inadequate correlation with histological improvement at least in the short-term. It is true that histological improvement tends to lag behind clinical and serological improvement, especially in adults in whom improvement may never be complete, but even considering this, a negative serological test has been shown to miss patients with persistent villous atrophy. The recognition of persistent villous atrophy appears to be important since

improvement beyond this level is associated with the improved outcomes listed above. It should be noted, however, that we could not identify a controlled study that objectively determined the level of histological improvement that would be associated with improved outcomes, and this is an area for future study. Although somewhat controversial, nonetheless, based on this report it would appear that follow-up biopsy, at least 1 year after GFD in adults to document improvement of the histological grade, would be valuable.

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Abbreviations and Acronyms

95% CI-	Ninety-five percent confidence interval
AGA-	Antigliadin antibody
AR-	Attributable risk
BMD-	Bone mineral density
CD –	Celiac disease
DXA-	Dual energy X-ray absorptiometry
EGD-	Esophagogastroduodenoscopy
ELISA-	Enzyme-linked immunosorbent assay
EMA-	Endomysial antibody
ESPGAN-	European Society of Pediatric Gastroenterology and Nutrition
ETCL-	Enteropathy-associated T-cell lymphoma
GFD-	Gluten-free diet
GP-	Guinea pig
HLA-	Human leukocyte antigen
HR-	Human recombinant
HU-	Human umbilical cord
IDA-	Iron deficiency anemia
IDDM-	Type I diabetes (insulin dependent)
IEL-	Intraepithelial lymphocytes
IF-	Immunofluorescence
IgA-	Immunoglobulin A
IgG-	Immunoglobulin G
ME-	Monkey esophagus
NHL-	Non-Hodgkin’s lymphoma
NPV-	Negative predictive value
OR-	Odds ratio
PPV-	Positive predictive value
Prev-	Prevalence
PVA-	Partial villous atrophy
RR-	Relative risk
SD-	Standard deviation
Sens-	Sensitivity
SIR-	Standardized incidence ratio
SMR-	Standardized mortality ratio
SPA-	Single photon absorptiometry
Spec-	Specificity
SVA-	Subtotal villous atrophy
tTG-	Tissue transglutaminase
VA-	Villous atrophy

Appendix A.

Table 1: Various causes of villous atrophy (VA; Farrell and Kelly, Am J Gastro 2001;96:3237)

Celiac disease
Dermatitis herpetiformis
Cow's milk protein intolerance (children)
Post-gastroenteritis
Giardiasis
Peptic duodenitis
Crohn's disease
Small intestinal bacterial overgrowth
Eosinophilic gastroenteritis
Radiation or chemotherapy
Tropical sprue
Severe malnutrition
Diffuse small intestinal lymphoma
Graft versus host disease
Hypogammaglobulinemia
Alpha chain disease

Table 2: Marsh (Gastroenterology 1992;102:330) and Rostami (Am J Gastroenterol 1999;94:888) modified histological criteria for CD

Criteria		Rostami modification (1999)	Original Marsh (1992)
Marsh 0		Same as original	**Pre-infiltrative:** • Normal mucosal and villous architecture
Marsh I		Same as original	**Infiltrative:** • Normal mucosal and villous architecture • Increased numbers of IELs
Marsh II		Same as original	**Hyperplastic:** • Similar to above but with enlarged crypts, with increased crypt cell division
Marsh III	**a**	**Partial VA:** • Shortened blunt villi • Mild lymphocyte infiltration • Enlarged hyperplastic crypts	**Destructive lesion:** • Flat mucosa – complete loss of villi • Lymphocyte infiltration • Enlarged hyperplastic crypts
	b	**Sub-total VA:** • Clearly atrophic villi – but still recognizable • Enlarged crypts whose immature epithelial cells are generated at an increased rate • Influx of inflammatory cells	
	c	**Total VA:** • Nearly total VA • Severe Marsh atrophic, hyperplastic and infiltrative lesions	
Marsh IV		Same as original	**Hypoplastic:** • Total VA • Normal crypt height but hypoplasia • Normal IEL count • Many feel this doesn't exist and represents severe malnutrition
VA=villous atrophy IEL=intraepithelial lymphocytes			

Table 3: Revised ESPGAN criteria

Criteria	ESPGAN*- 1979	ESPGAN†- Revised 1990
Initial histology	- Absent or nearly absent villi - Recognized existence of less severe lesion - No consensus on verification of less severe lesions but recommended if possible continuing gluten diet and assess histology, or re-challenge after GFD, given the large differential of milder histologic lesions	- Biopsy must remain the initial step in the diagnosis (mandatory) - Recommend capsule over endoscopic biopsy - Large well oriented biopsy - Histology: hyperplastic VA with hyperplasia of the crypts and an abnormal surface epithelium. The IEL count is raised - Morphometery and histochemistry are important aids to diagnosis. - Monoclonal antibodies to IEL may be a future aid
Antibody studies	- n/a	- Recognize that IgA AGA, and EMA have a high degree of sensitivity and specificity for the diagnosis of CD - When such antibodies are present at the time of diagnosis in a child with a typical small intestinal mucosa, and when they disappear in parallel to a clinical response to a GFD, weight is added to the diagnosis of CD that may now be said to have been finally established - When biopsy is unavailable in communities were other causes of enteropathy are rare, the presence of abnormal concentrations of two antibodies strongly suggests that CD is a diagnostic possibility - Antibodies can be a marker of response to a GFD and a guide to dietary compliance
Improvement on GFD	- Recognized as central to the definition - Recognized that improvement need not be complete	- Second mandatory requirement remains a reasonably rapid (weeks rather than many months) clinical remission on a strict GFD - Control biopsy is always a suitable way of verifying the effect of GFD, and is required in asymptomatic pts
Gluten Challenge	- Importance of gluten challenge and re-biopsy emphasized to document “permanence” of gluten intolerance - However, the panel recognized that challenge was not being performed in routine practice (only 652 were performed among several thousand children with gluten intolerance)	- No longer a requirement - Should be used in equivocal cases such as when no initial biopsy was done, biopsy was inadequate or atypical, in communities with high rates of other enteropathies, or in situations when pts plan to abandon the GFD in an uncontrolled way - Challenge should be performed after obtaining a control biopsy on a GFD - Re-biopsy is performed 3-6 months later with the recognition that relapse can take 5-7 years or more to occur.
2-year rule	- To address the issue of transient gluten intolerance, the panel emphasized the usefulness of the 2-year rule after stopping a GFD - 619 of 652 gluten challenges redeveloped histology compatible with CD by 2 years	- The 2-year rule is practical in most cases, but several reports of relapse occurring 5-7 years after gluten rechallenge

*McNeish et al., Arch Dis Child 1979;54:783
†Walker-Smith et al., Arch Dis Child 1990:65:99
CD=celiac disease; n/a=not applicable; GFD=gluten-free diet

Appendix B. Search Strategies

Search Strategy 1

Celiac 1 – Diagnostic Tests

Test 1. EMA

MEDLINE on DIALOG

1. s anti(w)endomysial(w)antibod? OR antiendomysial(w)antibod?
2. s anti(w)endomysium(w)antibod? OR antiendomysium(w)antibod?
3. s endomysial(w)antibod? OR endomysium(w)antibod? OR endomysial(w)autoantibod? OR endomysium(w)autoantibod?
4. s endomysial(n3)iga OR antiendomysial(n3)iga OR iga(n)ema
5. s endomysium(n3)iga OR antiendomysium(n3)iga OR igg(n)ema
6. s immunoglobulin?(n3)endomysial OR immunoglobulin?(n3)antiendomysial
7. s immunoglobulin?(n3)endomysium OR immunoglobulin?(n3)antiendomysium
8. s ema(n3)antibod? OR ema(n3)autoantibod? OR anti(w)ema OR ema(n3)positiv?
9. s aea AND (endomysial OR endomysium OR antiendomys?) OR aea(n3)positiv? OR aea(n2)igg OR aea(n2)iga
10. c 1 OR 2 OR 3 OR 4 OR 5 OR 6 OR 7 OR 8 OR 9
11. s (celiac OR celiacs OR coeliac OR coeliacs OR gluten OR glutens OR glutenin OR glutenins OR gliadin OR gliadins OR celiac disease/de) AND (ema OR aea)
12. s (celiac OR celiacs OR coeliac OR coeliacs OR gluten OR glutens OR glutenin OR glutenins OR gliadin OR gliadins OR celiac disease/de) AND autoantibod?(n2) positiv?
13. c 10 OR 11 OR 12
14. s epithelial(w)membrane(w)antigen
15. c 13 NOT 14
16. s s15/human
17. s s16/eng

EMBASE on DIALOG

1. s anti(w)endomysial(w)antibod? OR antiendomysial(w)antibod?
2. s anti(w)endomysium(w)antibod? OR antiendomysium(w)antibod?
3. s endomysial(w)antibod? OR endomysium(w)antibod? OR endomysial(w)autoantibod? OR endomysium(w)autoantibod? OR endomysium antibody/de
4. s endomysial(n3)iga OR antiendomysial(n3)iga OR iga(n)ema
5. s endomysium(n3)iga OR antiendomysium(n3)iga OR igg(n)ema 6. s immunoglobulin?(n3)endomysial OR immunoglobulin?(n3)antiendomysial
7. s immunoglobulin?(n3)endomysium OR immunoglobulin?(n3)antiendomysium
8. s ema(n3)antibod? OR ema(n3)autoantibod? OR anti(w)ema OR ema(n3)positiv?
9. s aea AND (endomysial OR endomysium OR antiendomys?) OR aea(n3)positiv? OR aea(n2)igg OR aea(n2)iga
10. c 1 OR 2 OR 3 OR 4 OR 5 OR 6 OR 7 OR 8 OR 9
11. s (celiac OR celiacs OR coeliac OR coeliacs OR gluten OR glutens OR glutenin OR glutenins OR gliadin OR gliadins OR celiac disease/de) AND (ema OR aea)
12. s (celiac OR celiacs OR coeliac OR coeliacs OR gluten OR glutens OR glutenin OR glutenins OR gliadin OR gliadins OR celiac disease/de) AND autoantibod?(n2)positiv?
13. c 10 OR 11 OR 12

14. s epithelial(w)membrane(w)antigen
15. c 13 not 14
16. s s15/human
17. s s16/eng

Test 2. tTG

MEDLINE on DIALOG

1. s tissue(w)transglutaminase?? OR tissue(w)trans(w)glutaminase??
2. s antitissue(w)transglutaminase?? OR anti(w)transglutaminase??
3. s human(w)transglutaminase?? OR antitransglutaminase??(n3)antibod?
4. s (immunoglobulin? OR immunoglobulin a/de OR immunoglobulin g/de) AND (transglutaminase OR transglutaminases)
5. s ttg(n3)antibod? OR ttg(n3)autoantibod? OR ttg(w)(kit OR kits) OR ttga OR httg OR anti(w2)ttg OR human(w)ttg OR elisa(n)ttg OR attga
6. s (transglutaminase?? AND antibod?) OR (transglutaminase?? AND autoantibod?)
7. s transglutaminase??(n3)iga OR transglutaminase??(n3)igg OR tg2(n5)transglutaminase?? OR human(w) recombinant(w)tg2
8. s anti(w)gamma(w)glutamyltransferase AND (antibod? OR autoantibod?)
9. c 1 OR 2 OR 3 OR 4 OR 5 OR 6 OR 7 OR 8
10. s (celiac OR celiacs OR coeliac OR coeliacs OR gluten OR glutens OR glutenin OR glutenins OR gliadin OR gliadins OR celiac disease/de) AND (transglutaminase OR transglutaminases OR ttg OR tg2)
11. c 9 OR 10
12. s s11/human
13. s s12/eng

EMBASE on DIALOG

1. s tissue(w)transglutaminase?? OR tissue(w)trans(w)glutaminase??
2. s antitissue(w)transglutaminase?? OR anti(w)transglutaminase??
3. s human(w)transglutaminase?? OR antitransglutaminase??(n3)antibod?
4. s immunoglobulin OR immunoglobulin a/de OR immunoglobulin a1/de OR immunoglobulin a2/de
5. s immunoglobulin g/de OR immunoglobulin g1/de OR immunoglobulin g2/de OR immunoglobulin g2a/de OR immunoglobulin g2b/de OR immunoglobulin g3/de OR immunoglobulin g4/de
6. s transglutaminase OR transglutaminases
7. c 4 OR 5
8. c 7 AND 6
9. s ttg(n3)antibod? OR ttg(n3)autoantibod? OR ttg(w)(kit OR kits OR assay) OR ttga OR httg OR anti(w2)ttg OR human(w)ttg OR elisa(n)ttg OR attga
10. s (transglutaminase?? AND antibod?) OR (transglutaminase?? AND autoantibod?)
11. s transglutaminase??(n3)iga OR transglutaminase??(n3)igg OR tg2(n5)transglutaminase?? OR human(w) recombinant(w)tg2
12. s anti(w)gamma(w)glutamyltransferase AND (antibod? OR autoantibod?)
13. c 1 OR 2 OR 3 OR 8 OR 9 OR 10 OR 11 OR 12
14. s (celiac OR celiacs OR coeliac OR coeliacs OR gluten OR glutens OR glutenin OR glutenins OR gliadin OR gliadins OR celiac disease/de) AND (transglutaminase OR transglutaminases OR ttg OR tg2)
15. c 13 OR 14
16. s s15/human
17. s s16/eng

Test 3. AGA

MEDLINE on DIALOG

1. s gliadin(w)antibod? OR antigliadin(w)antibod? OR iga(n3)antigliadin OR igg(n3)antigliadin
2. s antigliadin AND (serology OR serological)
3. s iga(n2)aga OR igg(n2)aga OR aga(n3)positive? OR anti(w)aga
4. s (celiac OR celiacs OR coeliac OR coeliacs OR gluten OR glutens OR glutenin OR glutenins OR gliadin OR gliadins OR celiac disease/de) AND (aga OR antigliadin?)
5. c 1 OR 2 OR 3
6. c 5 OR 4
7. s s6/human
8. s s7/eng

EMBASE on DIALOG

1. s gliadin(w)antibod? OR antigliadin(w)antibod? OR iga(n3)antigliadin OR igg(n3)antigliadin
2. s antigliadin AND (serology OR serological)
3. s iga(n2)aga OR igg(n2)aga OR aga(n3)positive? OR anti(w)aga
4. s (celiac OR celiacs OR coeliac OR coeliacs OR gluten OR glutens OR glutenin OR glutenins OR gliadin OR gliadins OR celiac disease/de) AND (aga OR antigliadin? OR anti(w)gliadin?)
5. c 1 OR 2 OR 3
6. c 5 OR 4
7. s s6/human
8. s s7/eng

Test 4. HLA DQ2/DQ8

MEDLINE on DIALOG

1. s (leukocyte OR leukocytes OR leucocyte OR leucocytes) AND (antigen OR antigens)
2. s hla OR hla-dq antigens/de OR hla antigens/de OR hladq OR histocompatibility antigens/de OR histocompatibility testing/de OR histocompatibility
3. c 1 OR 2
4. s dq2? OR dq8? OR hladq2? OR hladq8? OR d2? OR d8?
5. c 3 AND 4
6. s celiac OR celiacs OR coeliac OR coeliacs OR gluten OR glutens OR glutenin OR glutenins OR gliadin OR gliadins OR celiac disease/de
7. s hla(w)antigen?? OR hla antigens/de OR hla-dq antigens/de
8. c 6 AND 7
9. c 5 OR 8
10. s s8/human
11. s s9/eng

EMBASE on DIALOG

1. s (leukocyte OR leukocytes OR leucocyte OR leucocytes) AND antigen
2. s (leukocyte OR leukocytes OR leucocyte OR leucocytes) AND antigens
3. s hla OR hla dq antigen/de OR hla antigen/de OR hladq OR histocompatibility antigen/de OR histocompatibility test/de OR histocompatibility/ti,ab,de
4. c 1 OR 2 OR 3
5. s dq2? OR dq8? OR hladq2? OR hladq8? OR d2? OR d8?
6. c 4 AND 5
7. s celiac OR celiacs OR coeliac OR coeliacs OR gluten OR glutens OR glutenin OR glutenins OR gliadin OR gliadins
8. s hla(w)antigens?? OR hla antigen/de OR hla dq antigen/de
9. c 7 AND 8
10. c 6 OR 9
11. s s10/human
12. s s11/eng

Test 5. Small bowel biopsy

MEDLINE on DIALOG

1. s celiac???(w)disease OR coeliac???(w)disease OR celiac(w)sprue OR coeliac(w)sprue OR celiac(w)syndrome OR coeliac(w)syndrome
2. s celiacs OR coeliacs OR celiac disease/de
3. s silent(w)celiac OR silent(w)coeliac OR asymptomatic(w)celiac OR asymptomatic(w)coeliac OR undetected(n3)(celiac OR coeliac)
4. s subclinical(w)celiac OR subclinical(w)coeliac OR sub(w)clinical(w)celiac OR sub(w)clinical(w)coeliac OR undiagnosed(n3)celiac OR undiagnosed(n3)coeliac
5. s nontropical(w)sprue OR non(w)tropical(w)sprue OR endemic(w)sprue
6. s gluten(n3)enteropath? OR gluten(n3)sensitiv? OR gluten(n3)hypersensitive? OR gluten(n3)intoleran? OR glutenin??(n3)hypersensitiv? OR glutenin??(n3) sensitiv? OR glutenin??(n3)intoleran?
7. s gliadin(n3)hypersentitiv? OR gliadin(n3)intoleran?
8. s wheat(n3)hypersensitiv? OR wheat(n3)intoleran? OR wheat hypersensitivity/de
9. s (wheat OR triticum OR gluten) AND food hypersensitivity/de
10. c 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9
11. s celiac disease(l)pathology OR celiac disease(l)diagnosis OR wheat hypersensivitity(l)diagnosis OR wheat hypersensitivity(l)pathology
12. s (wheat OR triticum OR gluten) AND food hypersensitivity(l)diagnosis
13. s (wheat OR triticum OR gluten) AND food hypersensitivity(l)pathology
14. c 11 or 12 or 13
15. s intestine, small(l)pathology OR duodenum(l)pathology OR jejunum(l)pathology OR ileum(l)pathology OR small(n2)bowel/ti,ab OR small(n2)intestine?/ti,ab
16. s biopsy! OR biopsy/ti,ab OR biopsies/ti,ab
17. c 14 AND 15 AND 16
18. s small(w)(bowel OR intestine OR intestines) OR intestinal(w)mucosa
19. c 10 AND 16 AND 18
20. s (villi OR villus OR villous OR microvilli)(n3)atroph?
21. c (10 AND 20) OR (14 AND 20)
22. c 17 OR 19 OR 21
23. s s22/human
24. s s23/eng

EMBASE on DIALOG

1. s celiac???(w)disease OR coeliac???(w)disease OR celiac(w)sprue OR coeliac(w)sprue OR celiac(w)syndrome OR coeliac(w)syndrome
2. s celiacs OR coeliacs OR celiac disease/de
3. s silent(w)celiac OR silent(w)coeliac OR asymptomatic(w)celiac OR asymptomatic(w)coeliac OR undetected(n3)(celiac OR coeliac)
4. s subclinical(w)celiac OR subclinical(w)coeliac OR sub(w)clinical(w)celiac OR sub(w)clinical(w)coeliac OR undiagnosed(n3)celiac OR undiagnosed(n3)coeliac
5. s nontropical(w)sprue OR non(w)tropical(w)sprue OR endemic(w)sprue
6. s gluten(n3)enteropath? OR gluten(n3)sensitiv? OR gluten(n3)hypersensitive? OR gluten(n3)intoleran? OR glutenin??(n3)hypersensitiv? OR glutenin??(n3) sensitiv? OR glutenin??(n3)intoleran?
7. s gliadin(n3)hypersentitiv? OR gliadin(n3)intoleran?
8. s wheat(n3)hypersensitiv? OR wheat(n3)intoleran? OR wheat allergy/de
9. s (wheat OR triticum OR gluten OR glutens) AND food allergy/de
10. c 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9
11. s small intestine/de OR duodenum/ti,ab,de OR jejunum/ti,ab,de OR ileum/ti,ab,de OR small(n2)bowel/ti,ab OR small(n2)intestine?/ti,ab OR duodenal/ti,ab OR jejunal/ti,ab OR ileal/ti,ab
12. s dc=a3.60.70? [small intestine]

13. s small(w)(bowel OR intestine OR intestines) OR intestinal(w)mucosa
14. s intestine mucosa/de or duodenum mucosa/de OR jejunum mucosa/de OR ileum mucosa/de
15. c 11 OR 12 OR 13 OR 14
16. s biopsy/ti,ab,de OR biopsies/ti,ab
17. c 15 AND 16
18. s intestine biopsy/de OR duodenum biopsy/de OR jejunum biopsy/de OR ileum biopsy/de
19. c 17 OR 18
20. c 10 AND 19
21. s (villi OR villus OR villous OR microvilli)(n3)atroph?
22. c 10 AND 21
23. c 20 OR 22
24. s s23/human
25. s s24/eng

Celiac 2 – Epidemiology

MEDLINE on DIALOG

1. s celiac???(w)disease OR coeliac???(w)disease OR celiac(w)sprue OR coeliac(w)sprue OR celiac(w)syndrome OR coeliac(w)syndrome
2. s celiacs OR coeliacs OR celiac disease/de
3. s silent(w)celiac OR silent(w)coeliac OR asymptomatic(w)celiac OR asymptomatic(w)coeliac OR undetected(n3)(celiac OR coeliac)
4. s subclinical(w)celiac OR subclinical(w)coeliac OR sub(w)clinical(w)celiac OR sub(w)clinical(w)coeliac OR undiagnosed(n3)celiac OR undiagnosed(n3)coeliac
5. s nontropical(w)sprue OR non(w)tropical(w)sprue OR endemic(w)sprue
6. s gluten(n3)enteropath? OR gluten(n3)sensitiv? OR gluten(n3)hypersensitive? OR gluten(n3)intoleran? OR glutenin??(n3)hypersensitiv? OR glutenin??(n3) sensitiv? OR glutenin??(n3)intoleran?
7. s gliadin(n3)hypersentitiv? OR gliadin(n3)intoleran?
8. s wheat(n3)hypersensitive? OR wheat(n3)intoleran? OR wheat hypersensitivity/de
9. s (wheat OR triticum OR gluten) AND food hypersensitivity/de
10. c 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9
11. s celiac disease(l)epidemiology OR celiac disease(l)ethnology OR wheat hypersensitivity(l)epidemiology OR wheat hypersensitivity(l)ethnology
12. s (wheat OR triticum OR gluten) AND food hypersensitivity(l)epidemiology
13. s (wheat OR triticum OR gluten) AND food hypersensitivity(l)ethnology
14. s epidemiolog?/ti,de OR occurrence/ti,de OR prevalence/ti,de OR incidence/ti,de OR pedigree/de OR seroprevalence OR seroepidemiol? OR epidemiologic studies! OR epidemiologic measurements/de [check!!]
15. s population characteristics! OR population! OR demography! OR demographic?/ti,ab OR population?/ti,de
16. s minority groups/de OR ethnic groups! OR racial stocks!
17. s anemia(w2)iron(w)deficiency/de OR anemia(w)hypochromic/de OR iron(w)deficiency(w)anemia OR osteoporosis/ti,ab,de OR diabetes mellitus, insulin-dependent! OR juvenile(w)diabetes
18. s diabetes AND type(w)(1 OR I OR one)
19. s celiac disease(l)genetics OR gluten(l)genetics OR wheat hypersensitivity(l)genetics
20. s (wheat OR triticum OR gluten) AND food hypersensitivity(l)genetics
21. s family! OR genetic predisposition to disease! OR genetic(w)predisposition OR family(n3)(member OR members) OR familial
22. s family/ti,ab OR families/ti,ab OR familial/ti,ab OR brother/ti,ab OR brothers/ti,ab OR sister/ti,ab OR sisters/ti,ab OR aunt/ti,ab OR aunts/ti,ab OR uncle/ti,ab OR uncles/ti,ab OR cousin/ti,ab OR cousins/ti,ab
23. s parent/ti,ab OR parents/ti,ab OR mother/ti,ab OR mothers/ti,ab OR father/ti,ab OR fathers/ti,ab OR wife/ti,ab OR wives/ti,ab OR husband/ti,ab OR husbands/ti,ab
24. s son/ti,ab OR sons/ti,ab OR daughter/ti,ab OR daughters/ti,ab OR children/ti,ab OR relatives/ti,ab OR sibling/ti,ab OR siblings/ti,ab OR offspring/ti,ab
25. c 21 OR 22 OR 23 OR 24
26. c 19 OR 20
27. c 25 AND 26
28. c 14 OR 15 OR 16 OR 17 OR 18
29. c 10 AND 28
30. c 27 OR 29 OR 11 OR 12 OR 13
31. s s30/human
32. s s31/eng
33. s animal/de
34. c 32 NOT 33

EMBASE on DIALOG

1. s celiac???(w)disease OR coeliac???(w)disease OR celiac(w)sprue OR coeliac(w)sprue OR celiac(w)syndrome OR coeliac(w)syndrome
2. s celiacs OR coeliacs OR celiac disease/de
3. s silent(w)celiac OR silent(w)coeliac OR asymptomatic(w)celiac OR asymptomatic(w)coeliac OR undetected(n3)(celiac OR coeliac)
4. s subclinical(w)celiac OR subclinical(w)coeliac OR sub(w)clinical(w)celiac OR sub(w)clinical(w)coeliac OR undiagnosed(n3)celiac OR undiagnosed(n3)coeliac
5. s nontropical(w)sprue OR non(w)tropical(w)sprue OR endemic(w)sprue OR refractory(w)sprue
6. s gluten(n3)enteropath? OR gluten(n3)sensitiv? OR gluten(n3)hypersensitive? OR gluten(n3)intoleran? OR glutenin??(n3)hypersensitiv? OR glutenin??(n3) sensitiv? OR glutenin??(n3)intoleran?
7. s gliadin(n3)hypersentitiv? OR gliadin(n3)intoleran?
8. s wheat(n3)hypersensitive? OR wheat(n3)intoleran? OR wheat allergy/de OR cereal(w)allergy 9. s (wheat OR triticum OR gluten?? OR glutinin??) AND (food allergy/de OR allergy/de OR hypersensitivity/de OR food allergen/de)
10. c 1 OR 2 OR 3 OR 4 OR 5 OR 6 OR 7 OR 8 OR 9
11. s epidemiology/de,id OR dc=c1.270? OR epidemiolog?/ti,ab OR seroepidemiol? OR epidemiological data/de 12. s occurrence/ti,de OR prevalence/ti,de OR incidence/ti,de OR pedigree/ti,de OR pedigree analysis/de OR dc=g1.385.170? OR seroprevalence
13. s dc= g1.250.710.715? OR dc= i1.700? OR demography/ti,ab,de OR demographic?/ti,ab OR population?/ti,de OR population research/de OR population risk/de
14. s minority(w)group?? OR ethnic(w)group?? OR minorities/ti,ab OR dc=g1.750? OR dc=i1.275? OR dc=m2? OR ethnology/ti,ab,de OR ethnic difference/de OR ethnicity/ti,ab
15. s iron(w)deficiency(w)anemia OR iron deficiency anemia/de OR anemia(n3)hypochromic/ti,ab
16. s osteoporosis/ti,ab,de OR dc= c2.275.540.110.650?
17. s insulin dependent diabetes mellitus/de OR juvenile(w)diabetes OR insulin(w)dependent(w)diabetes
18. s diabetes AND type(w)(1 OR i OR one)
19. s familial disease/de OR family study/de OR familial incidence/de
20. c 11 OR 12 OR 13 OR 14 OR 15 OR 16 OR 17 OR 18 OR 19
21. s celiac disease/de OR gluten??/ti,ab OR wheat allergy/de OR cereal(w)allergy
22. s (wheat OR triticum OR gluten?? OR glutinin??) AND (food allergy/de OR allergy/de OR hypersensitivity/de OR food allergen/de)
23. c 21 OR 22
24. s genetics/de OR q1.340?
25. c 23 AND 24
26. s genetic(w)predisposition OR family(n3)(member OR members)
27. s family/ti,ab OR families/ti,ab OR familial/ti,ab OR brother/ti,ab OR brothers/ti,ab OR sister/ti,ab OR sisters/ti,ab OR aunt/ti,ab OR aunts/ti,ab OR uncle/ti,ab OR uncles/ti,ab OR cousin/ti,ab OR cousins/ti,ab
28. s parent/ti,ab OR parents/ti,ab OR mother/ti,ab OR mothers/ti,ab OR father/ti,ab OR fathers/ti,ab OR wife/ti,ab OR wives/ti,ab OR husband/ti,ab OR husbands/ti,ab
29. s son/ti,ab OR sons/ti,ab OR daughter/ti,ab OR daughters/ti,ab OR children/ti,ab OR relatives/ti,ab OR sibling/ti,ab OR siblings/ti,ab OR offspring/ti,ab
30. c 26 OR 27 OR 28 OR 29
31. c 25 AND 30
32. c 10 AND 20
33. c 31 OR 32
34. s celiac disease(l)epidemiology
35. s (celiac disease/de OR wheat allergy/de OR cereal(w)allergy) AND epidemiology/de
36. s (wheat OR triticum OR gluten?? OR glutinin??) AND (food allergy/de OR allergy/de OR hypersensitivity/de OR food allergen/de) AND epidemiology/de
37. c 34 OR 35 OR 36
38. c 33 OR 37
39. s s38/human
40. s s39/eng

Celiac 3 – Lymphomas

MEDLINE on DIALOG

1. s celiac???(w)disease OR coeliac???(w)disease OR celiac(w)sprue OR coeliac(w)sprue OR celiac(w)syndrome OR coeliac(w)syndrome
2. s celiacs OR coeliacs OR celiac disease/de
3. s silent(w)celiac OR silent(w)coeliac OR asymptomatic(w)celiac OR asymptomatic(w)coeliac OR undetected(n3)(celiac OR coeliac)
4. s subclinical(w)celiac OR subclinical(w)coeliac OR sub(w)clinical(w)celiac OR sub(w)clinical(w)coeliac OR undiagnosed(n3)celiac OR undiagnosed(n3)coeliac
5. s nontropical(w)sprue OR non(w)tropical(w)sprue OR endemic(w)sprue
6. s gluten(n3)enteropath? OR gluten(n3)sensitiv? OR gluten(n3)hypersensitive? OR gluten(n3)intoleran? OR glutenin??(n3)hypersensitiv? OR glutenin??(n3) sensitiv? OR glutenin??(n3)intoleran?
7. s gliadin(n3)hypersentitiv? OR gliadin(n3)intoleran?
8. s wheat(n3)hypersensitive? OR wheat(n3)intoleran? OR wheat hypersensitivity/de
9. s (wheat OR triticum OR gluten) AND food hypersensitivity/de
10. c 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9
11. s (villi OR villus OR villous OR microvilli)(n3)atroph?
12. c 10 OR 11
13. s lymphoma/ti,ab,de OR lymphomas/ti,ab,de OR lymphoma! OR hodgkin?/ti,ab,de
14. s intestine/ti,ab,de OR intestinal/ti,ab,de OR duodenum/ti,ab,de OR duodenal/ti,ab.de OR jejunum/ti,ab,de OR jejunal/ti,ab,de OR ileum/ti,ab,de OR ileal/ti,ab,de
15. s small(n2)bowel/ti,ab OR small(n2)intestine?/ti,ab OR large(n2)intestine?/ti,ab OR large(n2)bowel/ti,ab OR intestines!
16. s gastric/ti,ab,de OR gastro?/ti,ab,de OR gi/ti,ab OR stomach/ti,ab,de OR pylorus/ti,ab,de OR pyloric/ti,ab,de OR esophagogastr?
17. c 14 or 15 or 16
18. c 12 AND 13 AND 17
19. s s18/human
20. s s19/eng

EMBASE on DIALOG

1. s celiac???(w)disease OR coeliac???(w)disease OR celiac(w)sprue OR coeliac(w)sprue OR celiac(w)syndrome OR coeliac(w)syndrome
2. s celiacs OR coeliacs OR celiac disease/de
3. s silent(w)celiac OR silent(w)coeliac OR asymptomatic(w)celiac OR asymptomatic(w)coeliac OR undetected(n3)(celiac OR coeliac)
4. s subclinical(w)celiac OR subclinical(w)coeliac OR sub(w)clinical(w)celiac OR sub(w)clinical(w)coeliac OR undiagnosed(n3)celiac OR undiagnosed(n3)celiac
5. s nontropical(w)sprue OR non(w)tropical(w)sprue OR endemic(w)sprue
6. s gluten(n3)enteropath? OR gluten(n3)sensitiv? OR gluten(n3)hypersensitive? OR gluten(n3)intoleran? OR glutenin??(n3)hypersensitiv? OR glutenin??(n3) sensitiv? OR glutenin??(n3)intoleran?
7. s gliadin(n3)hypersentitiv? OR gliadin(n3)intoleran? Or gluten free diet/de OR gluten(w)free
8. s wheat(n3)hypersensitive? OR wheat(n3)intoleran? OR wheat allergy/de OR cereal(w)allergy
9. s (wheat OR triticum OR gluten?? OR glutinin??) AND (food allergy/de OR allergy/de OR hypersensitivity/de)
10. c 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9
11. s (villi OR villus OR villous OR microvilli)(n3)atroph?
12. s refractory/ti,ab OR non(w)respond?/ti,ab OR nonrespond?/ti,ab OR non(w)responsiv?/ti,ab OR nonresponsiv?/ti,ab
13. s sprue OR celiac OR coeliac OR celiac disease/de
14. c 12 AND 13
15. c 10 OR 11 OR 14

16. s lymphoma/ti,ab,de OR lymphomas/ti,ab,de OR hodgkin?/ti,ab,de
17. s dc = c2.385.520.500.510? OR dc = c2.385.520.510.500? OR dc = c6.610.50.50? OR dc = c6.610.75.520.510? [var. lymphoma]
18. c 16 OR 17
19. s small intestine/de OR small(n2)bowel/ti,ab OR small(n2)intestine?/ti,ab OR duodenum/ti,ab,de OR duodenal/ti,ab OR jejunum/ti,ab,de OR jejunal/ti,ab OR ileum/ti,ab,de OR ileal/ti,ab
20. s large(n2)intestine?/ti,ab OR large(n2)bowel/ti,ab OR cecum/ti,ab,de OR colon/ti,ab,de OR colonic/ti,ab,de OR rectum/ti,ab,de OR rectal/ti,ab,de OR anus/ti,ab,de
21. s intestine/ti,ab,de OR intestinal/ti,ab,de OR dc = a3? [digestive system]
22. s intestine mucosa/de OR duodenum mucosa/de OR jejunum mucosa/de OR ileum mucosa/de OR intestinal(w)mucosa OR colon mucosa/de OR rectum mucosa/de OR small intestine mucosa/de
23. s gastric/ti,ab,de OR gastro?/ti,ab,de OR gi/ti,ab OR stomach/ti,ab,de OR cardia/ti,ab,de OR pylorus/ti,ab,de OR pyloric/ti,ab,de OR esophagogastr?
24. s digestive system cancer/de OR dc = c2.220.230.210? OR dc = c2.220.230.210? [digestive system cancer]
25. c 19 OR 20 OR 21 OR 22 OR 23 OR 24
26. c 15 AND 18 AND 25
27. s stomach lymphoma/de OR intestine lymphoma/de
28. c 15 AND 27
29. c 26 OR 28
30. s s29/human
31. s s30/eng

Celiac 4 – Screening

MEDLINE on DIALOG

1. s celiac???(w)disease OR coeliac???(w)disease OR celiac(w)sprue OR coeliac(w)sprue OR celiac(w)syndrome OR coeliac(w)syndrome
2. s celiacs OR coeliacs OR celiac disease/de
3. s silent(w)celiac OR silent(w)coeliac OR asymptomatic(w)celiac OR asymptomatic(w)coeliac OR undetected(n3)(celiac OR coeliac)
4. s subclinical(w)celiac OR subclinical(w)coeliac OR sub(w)clinical(w)celiac OR sub(w)clinical(w)coeliac OR undiagnosed(n3)celiac OR undiagnosed(n3)coeliac
5. s nontropical(w)sprue OR non(w)tropical(w)sprue OR endemic(w)sprue
6. s gluten(n3)enteropath? OR gluten(n3)sensitiv? OR gluten(n3)hypersensitive? OR gluten(n3)intoleran? OR glutenin??(n3)hypersensitiv? OR glutenin??(n3) sensitiv? OR glutenin??(n3)intoleran?
7. s gliadin(n3)hypersentitiv? OR gliadin(n3)intoleran?
8. s wheat(n3)hypersensitive? OR wheat(n3)intoleran? OR wheat hypersensitivity/de
9. s (wheat OR triticum OR gluten) AND food hypersensitivity/de
10. c 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9
11. s screen/ti,ab OR screens/ti,ab OR screening/ti,ab OR screened/ti,ab OR mass screening!
12. c 10 AND 11
13. s celiac disease(l)diagnosis OR celiac disease(l)pathology
14. s “sensitivity and specificity”/de
15. s physician’s practice patterns/de
16. s reference standards/de OR quality control/de OR evaluation studies/de OR predictive value of tests/de OR incidental findings/de
17. s reproducibility of results/de OR physical examination(l)standards OR diagnosis, differential/de OR diagnostic errors! OR follow-up studies/de
18. s public health/de OR public health practice/de OR population surveillance/de OR clinical protocols/de OR critical pathways/de
19. s quality assurance, health care! OR guideline adherence/de OR social control, formal/de OR “outcome assessment (health care)”/de
20. c 14 or 15 or 16 or 17 or 18 or 19
21. c 13 AND 20
22. c 12 OR 21
23. s s22/human
24. s s23/eng

EMBASE on DIALOG

1. s celiac???(w)disease OR coeliac???(w)disease OR celiac(w)sprue OR coeliac(w)sprue OR celiac(w)syndrome OR coeliac(w)syndrome
2. s celiacs OR coeliacs OR celiac disease/de
3. s silent(w)celiac OR silent(w)coeliac OR asymptomatic(w)celiac OR asymptomatic(w)coeliac OR undetected(n3)(celiac OR coeliac)
4. s subclinical(w)celiac OR subclinical(w)coeliac OR sub(w)clinical(w)celiac OR sub(w)clinical(w)coeliac OR undiagnosed(n3)celiac OR undiagnosed(n3)coeliac
5. s nontropical(w)sprue OR non(w)tropical(w)sprue OR endemic(w)sprue
6. s gluten(n3)enteropath? OR gluten(n3)sensitiv? OR gluten(n3)hypersensitiv? OR gluten(n3)intoleran? OR glutenin??(n3)hypersensitiv? OR glutenin??(n3) sensitiv? OR glutenin??(n3)intoleran?
7. s gliadin(n3)hypersentitiv? OR gliadin(n3)intoleran?
8. s wheat(n3)hypersensitiv? OR wheat(n3)intoleran?
9. s (wheat OR triticum OR gluten) AND (food allergy/de OR hypersensitivity/de)
10. c 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9
11. s mass screening/de OR mass(w)screen? OR genetic screening/de OR genetic(w)screen? OR newborn screening/de OR newborn??(n3)screen?

12. 10 AND 11
13. s screen/ti,ab OR screens/ti,ab OR screening/ti,ab OR screened/ti,ab
14. s dc=J2.10? [controlled study]
15. s dc=J2.40.10? [clinical study]
16. s dc=J2.50? [methodology]
17. s evaluation(w)study OR evaluation(w)studies OR physical examination/de OR follow-up studies/de
18. s dc=E1.215? [diagnosis]
19. s dc=N7.700? [practice guideline]
20. s dc=Q1.550.75? [social medicine]
21. c 14 or 15 or 16 or 17 or 18 or 19 or 20
22. c 10 AND 13 AND 21
23. c 12 OR 22
24. s s23/human
25. s s24/eng

CAB and AGRICOLA on DIALOG

1. s celiac???(w)disease OR coeliac???(w)disease OR celiac(w)sprue OR coeliac(w)sprue OR celiac(w)syndrome OR coeliac(w)syndrome
2. s celiacs OR coeliacs OR celiac syndrome/de
3. s silent(w)celiac OR silent(w)coeliac OR asymptomatic(w)celiac OR asymptomatic(w)coeliac OR undetected(n3)(celiac OR coeliac)
4. s subclinical(w)celiac OR subclinical(w)coeliac OR sub(w)clinical(w)celiac OR sub(w)clinical(w)coeliac OR undiagnosed(n3)celiac OR undiagnosed(n3)coeliac
5. s nontropical(w)sprue OR non(w)tropical(w)sprue OR endemic(w)sprue
6. s gluten(n3)enteropath? OR gluten(n3)sensitiv? OR gluten(n3)hypersensitiv? OR gluten(n3)intoleran? OR glutenin??(n3)hypersensitiv? OR glutenin??(n3) sensitiv? OR glutenin??(n3)intoleran?
7. s gliadin(n3)hypersentitiv? OR gliadin(n3)intoleran?
8. s wheat(n3)hypersensitiv? OR wheat(n3)intoleran?
9. s (wheat OR triticum OR gluten) AND (food allergies/de OR hypersensitivity/de)
10. c 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9
11. s mass(w)screen? OR genetic(w)screen? OR newborn??(n3)screen?
12. c 10 AND 11
13. s screen/ti,ab OR screens/ti,ab OR screening/ti,ab OR screened/ti,ab
14. s evaluation(w)study OR evaluation(w)studies
15. c 10 AND 13 AND 14
16. c 12 OR 15
17. s s16/human
18. s s17/eng

PsycInfo on DIALOG

1. s celiac???(w)disease OR coeliac???(w)disease OR celiac(w)sprue OR coeliac(w)sprue OR celiac(w)syndrome OR coeliac(w)syndrome
2. s celiacs OR coeliacs
3. s silent(w)celiac OR silent(w)coeliac OR asymptomatic(w)celiac OR asymptomatic(w)coeliac OR undetected(n3)(celiac OR coeliac)
4. s subclinical(w)celiac OR subclinical(w)coeliac OR sub(w)clinical(w)celiac OR sub(w)clinical(w)coeliac OR undiagnosed(n3)celiac OR undiagnosed(n3)coeliac
5. s nontropical(w)sprue OR non(w)tropical(w)sprue OR endemic(w)sprue
6. s gluten(n3)enteropath? OR gluten(n3)sensitiv? OR gluten(n3)hypersensitiv? OR gluten(n3)intoleran? OR glutenin??(n3)hypersensitiv? OR glutenin??(n3) sensitiv? OR glutenin??(n3)intoleran?
7. s gliadin(n3)hypersentitiv? OR gliadin(n3)intoleran?
8. s wheat(n3)hypersensitiv? OR wheat(n3)intoleran?
9. s (wheat OR triticum OR gluten) AND food allergies/de
10. c 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9

11. s mass(w)screen? OR genetic(w)screen? OR newborn??(n3)screen? OR screening/de OR screening tests/de OR health screening/de
12. c 10 AND 11
13. s screen/ti,ab OR screens/ti,ab OR screening/ti,ab OR screened/ti,ab
14. s evaluation(w)study OR evaluation(w)studies OR physical examination/de OR followup studies/de OR evaluation/de
15. c 10 AND 13 AND 14
16. c 12 OR 15
17. s s16/human
18. s s17/eng

Sociological Abstracts at CAB

Limits set to English

1. celiac (KW) OR celiacs (KW) OR coeliac (KW) OR coeliacs (KW)
2. wheat (KW) OR gluten* (KW) OR gliadin* OR food (DE) OR foods (KW)
3. allergy (KW) OR allergies (KW) OR hypersensitivit* (KW) OR sensitive* (KW) OR intolerance* (KW)
4. mass public (DE) OR screen (KW OR screens (KW) OR screening (KW) OR screened (KW)
5. 1 AND 4
6. 2 and 3 AND 4
7. 5 OR 6

Celiac 5 – Dietary Compliance

MEDLINE on DIALOG:

Part 1

1. s celiac disease(l)psychology OR diet therapy(l)education OR diet therapy(l)methods OR diet therapy(l)standards OR diet therapy(l)trends OR diet therapy(l)utilization OR diet therapy(l)psychology
2. s diet(l)methods OR diet(l)trends OR diet(l)psychology OR diet(l)standards OR diet(l)utilization
3. s psychological tests/de OR health behavior/de OR patient acceptance of health care! OR health education/de OR patient education/de OR nutrition(l)education OR teaching!
4. s quality of life/de OR menu planning/de OR food habits/de OR feeding behavior/de OR quality of health care/de OR compliance/ti,ab OR adherence/ti,ab OR motivation/ti,ab,de
5. s achievement/de OR motivation/de OR directive counseling/de OR counseling/de OR psychology, applied/de OR psychology, educational/de OR learning/de OR child guidance/de
6. s adaptation, psychological/de OR attitude/de OR attitude of health personnel/de OR professional-patient relations! OR attitude to health!
7. s health promotion/de OR decision making! OR risk reduction behavior/de OR early(w)intervention/de OR intervention/ti,ab OR interventions/ti,ab OR data collection/de
8. s diet, protein-restricted(l)methods OR diet, protein-restricted(l)trends OR diet, protein-restricted(l)psychology OR diet, protein-restricted(l)standards OR diet, protein-restricted(l)utilization
9. s health surveys/de OR nutrition assessment! OR Behavioral Risk Factor Surveillance System/de OR interviews! OR questionnaire?/ti,ab,de
10. s guideline adherence/de OR evaluation studies/de OR “outcome assessment (health care)” OR “process assessment (health care)” OR food labeling/de
11. c 1 OR 2 OR 3 OR 4 OR 5 OR 6 OR 7 OR 8 OR 9 OR 10
12. s celiac disease(l)diet therapy
13. c 11 AND 12
14. s triticum(l)adverse effects OR wheat hypersensitivity/de OR gluten(n2)withdraw? OR gluten(l)adverse effects OR gliadin(l)adverse effects OR gluten(w)free
15. s diet OR dietary OR diets OR nutrition OR nutritional
16. c 14 AND 15
17. c 13 OR 16
18. s s17/human
19. s s18/eng

Part 2

1. s food labeling/de
2. s celiac OR coeliac OR triticum OR wheat hypersensitivity/de OR gluten OR gliadin OR celiac disease/de
3. c 1 and 2
4. s s3/human
5. s s4/eng

EMBASE on DIALOG

1. s celiac???(w)disease OR coeliac???(w)disease OR celiac(w)sprue OR coeliac(w)sprue OR celiac(w)syndrome OR coeliac(w)syndrome OR celiac(w)syndrome
2. s celiacs OR coeliacs OR celiac disease/de
3. s silent(w)celiac OR silent(w)coeliac OR asymptomatic(w)celiac OR asymptomatic(w)coeliac OR undetected(n3)(celiac OR coeliac)
4. s subclinical(w)celiac OR subclinical(w)coeliac OR sub(w)clinical(w)celiac OR sub(w)clinical(w)coeliac OR undiagnosed(n3)celiac OR undiagnosed(n3)coeliac
5. s nontropical(w)sprue OR non(w)tropical(w)sprue OR endemic(w)sprue

6. s gluten(n3)enteropath? OR gluten(n3)sensitiv? OR gluten(n3)hypersensitive? OR gluten(n3)intoleran? OR glutenin??(n3)hypersensitiv? OR glutenin??(n3) sensitiv? OR glutenin??(n3)intoleran?
7. s gliadin(n3)hypersentitiv? OR gliadin(n3)intoleran?
8. s wheat(n3)hypersensitive? OR wheat(n3)intoleran? OR wheat allergy/de
9. s (wheat OR triticum OR gluten) AND (hypersensitivity/de OR food allergy/de)
10. c 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9
11. s diet restriction/de OR gluten(w)free(w)diet?? OR diet therapy/de OR diet?(w)intervention OR therapeutic(w)diet?? OR diet OR dietary OR diets OR nutrition OR nutritional OR dietary(w) restriction?? OR dietary(n3)guideline??
12. s intervention??/ti,ab,de OR feeding behavior/de OR gluten(w)free(w)food?/ti,ab OR nutritional intolerance/de
13. c 11 OR 12
14. c 10 AND 13
15. s health behavior/de OR illness behavior/de OR adaptive behavior/de OR behavior modification/de OR patient attitude/de OR patient compliance/de OR patient education/de OR patient guidance/de OR patient counseling/de
16. s quality of life/de OR coping(w)behavior/ti,ab OR adjustment??/ti,ab,de OR decision making/de OR early(w)intervention/ti,ab,de OR compliance/ti,ab OR adherence/ti,ab OR motivation/ti,ab,de OR coping behavior/de OR coping/ti,ab,de
17. s psychosocial(n3)aspect?? OR habit??/ti,ab,de OR attitude??/ ti,ab,de OR psychologic test /de OR counsel?/ti,ab,de OR psychological factor/de OR psycholog?/ti,ab,de
18. c 15 OR 16 OR 17
19. c 14 AND 18
20. s s19/human, eng

CAB and AGRICOLA on DIALOG

1. s celiac???(w)disease OR coeliac???(w)disease OR celiac(w)sprue OR coeliac(w)sprue OR celiac(w)syndrome OR coeliac(w)syndrome OR celiac(w)syndrome
2. s celiacs OR coeliacs OR celiac syndrome/de
3. s silent(w)celiac OR silent(w)coeliac OR asymptomatic(w)celiac OR asymptomatic(w)coeliac OR undetected(n3)(celiac OR coeliac)
4. s subclinical(w)celiac OR subclinical(w)coeliac OR sub(w)clinical(w)celiac OR sub(w)clinical(w)coeliac OR undiagnosed(n3)celiac OR undiagnosed(n3)coeliac
5. s nontropical(w)sprue OR non(w)tropical(w)sprue OR endemic(w)sprue
6. s gluten(n3)enteropath? OR gluten(n3)sensitiv? OR gluten(n3)hypersensitive? OR gluten(n3)intoleran? OR glutenin??(n3)hypersensitiv? OR glutenin??(n3) sensitiv? OR glutenin??(n3)intoleran?
7. s gliadin(n3)hypersentitiv? OR gliadin(n3)intoleran?
8. s wheat(n3)hypersensitive? OR wheat(n3)intoleran?
9. s (wheat OR triticum OR gluten) AND (hypersensitivity/de OR food allergies/de)
10. c 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9
11. s gluten(w)free(w)diet?? OR diet treatment/de OR diet?(w)intervention OR therapeutic diets/de OR diet OR dietary OR diets OR nutrition OR nutritional
12. s dietary(w)restriction?? OR dietary(n3)guideline?? OR intervention??/ti,ab,de OR feeding behavior/de OR gluten(w)free(w)food?/ti,ab OR
13. c 11 OR 12
14. c 10 AND 13
15. s behavior modification/de OR patient compliance/de OR patient education/de
16. s quality of life/de OR coping(w)behavior/ti,ab OR adjustment??/ti,ab,de OR decision making/de OR early(w)intervention/ti,ab,de OR compliance/ti,ab OR adherence/ti,ab OR motivation/ti,ab,de OR coping/ti,ab
17. s psychosocial(n3)aspect?? OR habit??/ti,ab,de OR attitude??/ ti,ab,de OR counsel?/ti,ab,de OR psychological factors/de OR psycholog?/ti,ab,de
18. c 15 OR 16 OR 17
19. c 14 AND 18
20. s s19/human

21. s s20/eng

PsycInfo on DIALOG

1. s celiac???(w)disease OR coeliac???(w)disease OR celiac(w)sprue OR coeliac(w)sprue OR celiac(w)syndrome OR coeliac(w)syndrome OR celiac(w)syndrome
2. s celiacs OR coeliacs
3. s silent(w)celiac OR silent(w)coeliac OR asymptomatic(w)celiac OR asymptomatic(w)coeliac OR undetected(n3)(celiac OR coeliac)
4. s subclinical(w)celiac OR subclinical(w)coeliac OR sub(w)clinical(w)celiac OR sub(w)clinical(w)coeliac OR undiagnosed(n3)celiac OR undiagnosed(n3)coeliac
5. s nontropical(w)sprue OR non(w)tropical(w)sprue OR endemic(w)sprue
6. s gluten(n3)enteropath? OR gluten(n3)sensitiv? OR gluten(n3)hypersensitive? OR gluten(n3)intoleran? OR glutenin??(n3)hypersensitiv? OR glutenin??(n3) sensitiv? OR glutenin??(n3)intoleran?
7. s gliadin(n3)hypersentitiv? OR gliadin(n3)intoleran?
8. s wheat(n3)hypersensitive? OR wheat(n3)intoleran? OR wheat(n3)(allergy OR allergies)
9. s (wheat OR triticum OR gluten) AND food allergies/de
10. c 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9
11. s gluten(w)free(w)diet?? OR diet?(w)intervention OR therapeutic(w)diet?? OR diet OR dietary OR diets OR nutrition OR nutritional OR dietary(w)restriction?? OR dietary(n3)guideline??
12. s intervention??/ti,ab OR gluten(w)free(w)food?/ti,ab
13. c 11 OR 12
14. c 10 AND 13
15. s health behavior/de OR illness behavior/de OR adaptive behavior/de OR behavior modification/de
16. s quality of life/de OR coping(w)behavior/ti,ab OR adjustment??/ti,ab,de OR decision making/de OR early(w)intervention/ti,ab,de OR compliance/ti,ab OR adherence/ti,ab OR motivation/ti,ab,de OR coping behavior/de OR coping/ti,ab
17. s psychosocial(n3)aspect?? OR habit??/ti,ab,de OR attitude??/ti,ab,de OR counsel?/ti,ab,de OR psycholog?/ti,ab,de
18. c 15 OR 16 OR 17
19. c 14 AND 18
20. s s19/human
21. s s20/eng

Sociological Abstracts at CAB

Limits set to English

1. celiac (KW) OR celiacs (KW) OR coeliac (KW) OR coeliacs (KW)
2. wheat (KW) OR food (DE) OR foods (kw) OR gluten* (KW) OR gliadin*
3. allergy (KW) OR allergies (KW) OR hypersensitive* (KW) OR intoleran* (KW) OR sensitive* (KW)
4. feeding practices (DE) OR feeding (DE)
5. diet (DE) OR nutrition (DE)
6. diet (KW) OR diets(KW) OR nutrition (KW)OR nutritional (KW)
7. 2 AND 3
8. 4 OR 5 OR 6
9. 1 AND 8
10. 7 AND 8
11. 9 OR 10

Appendix C. Data Assessment and Data Abstraction Forms

Data Assessment Forms

Level 1 Screening

Objective 1:

1. Does this refer to determining the sensitivity or specificity of one of the following tests for celiac disease? (biopsy, anti-htTG, anti-endomysial, anti-gliadin antibody, anti-gliadin antibody, HLA DQ2/DQ8).
    a. This citation refers to another objective and should be moved or copied
    b. **No** (move on to next citation)
    c. **Yes**
    d. Can't tell

Objective 2:

1. Does this refer to the prevalence or incidence of **celiac disease**?
    a. This citation refers to another objective and should be moved or copied
    b. **No** (move on to next citation)
    c. **Yes**
    d. Can't tell

Objective 3:

1. Does this refer to an association between **celiac and GI lymphoma**?
    a. This citation refers to another objective and should be moved or copied
    b. **No** (move on to next citation)
    c. **Yes**
    d. Can't tell

Objective 4:

1. Does this refer to expected consequences of testing for celiac disease
    a. This citation refers to another objective and should be moved or copied
    b. **No** (move on to next citation)
    c. **Yes**
    d. Can't tell

Objective 5:

1. Does this refer to identifying or assessing interventions for promoting or monitoring adherence to a gluten free diet?
    a. This citation refers to another objective and should be moved or copied
    b. **No** (move on to next citation)
    c. **Yes**
    d. Can't tell

Level 2 Screening

Objective 1:

1. Does this refer **specifically** to determining the sensitivity or specificity of one of the identified tests for celiac disease? **Note: for biopsy and HLA**, we may not see sensitivity or specificity – keep if you can get data on use as diagnostic test or accuracy as a test or if it distinguishes celiac from other diseases etc.
    a. **Yes.** If this citation also refers to another objective(s), please state objective number(s):
    b. **No.** If this citation refers to another objective(s), please state objective number(s): (move on to next citation)

2. Is this a review article?
    a. Yes (keep for references)
    b. No

3. What is the test(s) being studied? (Note: we are not interested in any other type of test!)
    a. Biopsy
    b. Anti-htTG
    c. Anti-endomysial antibody (EMA)
    d. Anti-gliadin antibody (AGA)
    e. HLA DQ2/DQ8 (note: we are not interested in pathophysiology does the article give data using HLA to distinguish celiac from non celiac)
    f. If none of above then – reject citation

4. What is the "gold standard" the test(s) is compared to?
    a. Biopsy
    b. Anti-htTG
    c. Anti-endomysial antibody (EMA)
    d. Anti-gliadin antibody (AGA)
    e. HLA DQ2/DQ8
    f. Other (list in box)

5. What is the patient population?
    a. Adults

b. Paediatric
c. General unselected
d. Specific ethnic groups (fill in box)
e. Patients with suspected celiac (symptomatic)
f. Patients at risk of celiac disease (asymptomatic - relatives of celiac, diabetes, Fe Diff, infertility, osteoporosis, short stature)
g. Other (fill in box)

Objective 2:

1. Does this refer **specifically** to the prevalence or incidence of **celiac disease**? Please remember we are only really interested in the incidence / prevalence of celiac in population X or disease X **NOT** vice verse.
    a. **Yes**.
    b. **No. Exclude**
    c. **No.** But this referrers to an association between celiac and another disease to state in background /discussion

2. Is this a review article?
    a. Yes (keep for references)
    b. No

3. Does the prevalence or incidence refer to:
    a. Classical celiac
    b. Atypical celiac (i.e., Fe diff, infertility, short stature, osteoporosis)
    c. Asymptomatic celiac
    d. Other or (fill in box)

4. What is the patient population that was tested?
    a. Unselected – general population (e.g., blood donors, routine physical etc)
    b. Patients with suspected celiac
    c. Relatives of celiac patients
    d. Iron deficiency
    e. Osteoporosis
    f. Short stature
    g. Infertility
    h. Other (fill in box)

5. What was the screening test(s) used?
    a. Biopsy
    b. Anti-htTG
    c. Anti-endomysial antibody (EMA)
    d. Anti-gliadin antibody (AGA)
    e. HLA DQ2/DQ8
    f. Other (fill in box)

6. What is the country/region of origin of the study (fill in box)?

Objective 3:

1. Does this refer **specifically** to an association between **celiac and GI lymphoma**?
    a. **Yes**.
    b. **No. Exclude**

2. Is this a review article?
    a. Yes (keep for references)
    b. No

3. Does this give **data** on the risk of developing GI lymphoma in celiac?
    a. Yes
    b. No

4. What is the country/region of origin of the study? (fill in box)

5. What celiac population was evaluated?
    a. Classical celiac
    b. Atypical celiac (i.e., fe dif, infertility, short stature, osteoporosis)
    c. Asymptomatic celiac
    d. Other (fill in box)

Objective 4:

1. Does this refer **specifically** to expected **consequences of testing for celiac disease?**
    a. **Yes**.
    b. **No. Exclude**

2. Is this a review article?
    a. Yes (keep for references)
    b. No

3. What consequences were assessed:
    a. False-positive results
    b. Follow-up testing
    c. Invasive procedures (biopsy)
    d. Costs
    e. Cases diagnosed
    f. Patients complying with treatment
    g. Response to treatment
    h. Clinical outcome (reduced risk of complication etc)
    i. Other (fill in box)

4. What is the country/region of origin of the study? (fill in box)

5. What is the patient population that was tested?
    a. Unselected – general population (e.g., blood donors, routine physical, etc.)
    b. Patients with symptoms suggestive of celiac.
    c. Asymptomatic at risk populations (relatives of celiac patients, iron deficiency, osteoporosis, infertility short, stature)
    d. Other (fill in box).

Objective 5:

1. Does this **specifically** refer to identifying or assessing an intervention(s) for promoting or monitoring adherence to a gluten free diet?
    a. **Yes**.
    b. **No. Exclude**

2. Is this a review article?
    a. Yes (keep for references)
    b. No

3. Does this refer to:
    a. Promoting adherence
    b. Monitoring adherence
    c. Both

4. What intervention was assessed? (If **monitoring adherence**)
    a. Biopsy
    b. Antibody testing

5. What intervention was used? (If **promoting adherence**) – fill in box

Level 3 Screening

Celiac 1: Sensitivity and specificity of screening tests:

Inclusion criteria: (a **No** answer to any of the below excludes the article)

1. For serology - the study publication date is 1990 or more recent (biopsy studies can be earlier)
    a. Yes (include)
    b. No (exclude)

2. For AGA - the studies uses a standardized commercial ELISA kit (or this study is testing such a kit or technique)
    a. Yes (include)
    b. No (exclude)

3. For EMA – the substrate is monkey esophagus or human umbilical cord
    a. Yes (include)
    b. No (exclude)

4. For tTG – study uses ELISA with the substrate for tTG being guinea pig or human recombinant tTG.
    a. Yes (include)
    b. No (exclude)

5. For any serology and HLA studies – the control group(s) are appropriate and controls evaluated with the reference test (i.e., biopsy)?
    a. Yes (include)
    b. No (exclude)

6. The paper allows for the extraction of the sensitivity or specificity of the test in question (AGA, EMA, tTG, HLA DQ2/8, biopsy)?
    a. Yes (include)
    b. No (exclude)

7. Was the diagnosis of celiac disease appropriate in the celiac disease group
    a. Yes (include)
    b. No (exclude)

Celiac 2: Prevalence and incidence of celiac disease:

Inclusion Criteria: (a **No** answer to any of the below excludes the article)

1. The country of origin must be **Western Europe, North America, Australia, New Zealand**.
    a. Yes (include)
    b. No (exclude)

2. The publication date was >= 1990 if serology was used to screen (but can be earlier for biopsy)
    a. Yes (include)
    b. No (exclude)

3. The screening test was biopsy, standardized ELISA AGA, EMA (monkey esophagus or human umbilical cord), tTG (guinea pig, or human recombinant)
    a. Yes (include)
    b. No (exclude)

4. The screened population must belong to one of these groups:
        a. Unselected – General population (e.g. Blood donors, routine physical etc)
        b. Patients with suspected celiac
        c. Relatives of celiac patients
        d. Iron deficiency
        e. Osteoporosis
        f. Diabetes

        a. Yes (include)
        b. No (exclude)

Celiac 3: Prevalence/incidence of lymphoma in celiac disease

1. Does this study specifically give the **incidence**, **prevalence** or a **measure of risk** of GI lymphoma (Includes malignant histiocytosis) in a population of celiac patients? (Note: we are not interested in other cancers, and we are not interested in how many lymphoma patients have celiac disease)

   OR

   Does this study discuss ulcerative jejuno-ileitis or refractory sprue as a precursor or marker for GI lymphoma in patients with celiac disease?

   a. Yes (include lymphoma)
   b. Yes (include jejuno-ileitis/refractory sprue)
   c. No (exclude)

Celiac 4: Consequences of testing for celiac disease

1. Does this paper report a consequence of testing for celiac listed below: (note: false positive, and negative results, follow-up testing and need for invasive testing is obtained from Celiac 1 objective)
   a. **Costs**
   b. Patients complying with treatment
   c. **Response to treatment** – i.e., **clinical outcome** (reduced risk of complication – osteoporosis, lymphoma, anemia, symptoms)

      i. Yes (include)
      ii. No (exclude)

2. Did the population include one of the following (note: nothing more than listed):
   a. Patients with symptoms suggestive of celiac disease
   b. Asymptomatic, at-risk populations (affected family members, patients with type 1 diabetes, osteoporosis, Fe Diff)
   c. General population

      i. Yes (include)
      ii. No (exclude)

Note: a **No** to either question excludes the study

Celiac 5: Monitoring or promoting adherence to a GFD

If a monitoring question:

Does this paper report monitoring adherence based on **serology** (standardized ELISA **AGA publication date >= 1990**, **EMA** (monkey esophagus or human umbilical cord), **tTG** (guinea pig, or human recombinant) or **biopsy**?

Note: If this is a study of sensitivity or specificity – it must include actual extractable follow-up data (like drop in titre or improvement in biopsy)

OR

If a promoting adherence question:

Does this paper report on an intervention that was used to promote adherence to Gluten free diet?

a. Yes (include promoting)
b. Yes (include Monitoring)
c. No (exclude)

Data Abstraction Forms

Celiac 1: Serology

1. Paper #:
2. Title:
3. Author/year:
4. Reference:
5. Reviewer:

6. Publication type:
    a. Journal
    b. Conference abstract
    c. Other:

7. Is this a duplicate publication (state refid of duplicate):

8. Study type:
    a. Relevant clinical population: (cases and controls defined from the population based on the results of the test under study)
    b. Case Control: (groups are predefined and may come from different populations):
    c. Other: (list)

9. Country:

10. Racial Groups and % if different from country: *list in box*

11. Group demographics

	Celiac		Control			
	Group 1	Group 2	group1	group 2	group 3	group 4
Group name						
Age groups						
Mean age						
Age range						
% female						
Gluten intake						

12. Type of population (applies to 8 a): (*from level 2 database)*
    a. Unselected – general population (e.g., blood donors, routine physical, etc.)
    b. Patients with suspected celiac
    c. Relatives of celiac patients
    d. Diabetes
    e. Iron deficiency
    f. Osteoporosis
    g. Other (fill in box): *not part of extraction – background / discussion only*

13. Case and control group types (applies to 8 b):
    a. Celiac group 1
        i. Untreated
            1. Classic
            2. Silent celiac
            3. Atypical celiac
            4. Other
        ii. Treated On GFD
        iii. Refractory (implies on GFD) / ulcerative jejuno-ileitis
        iv. Other/can't tell: text box

    b. Celiac group 2 – if applicable
        i. Untreated
            1. Classic
            2. Silent celiac
            3. Atypical celiac
            4. Other
        ii. Treated on GFD
        iii. Refractory (implies on GFD) / ulcerative jejuno-ileitis
        iv. Other/can't tell: text box

Note: Control groups must have had a negative biopsy otherwise should have been excluded at level 3

    c. Control group 1:
        i. Unselected – general population (e.g., blood donors, routine physical, etc.)
        ii. Patients with suspected celiac
        iii. Relatives of celiac patients
        iv. Diabetes
        v. Iron deficiency
        vi. Osteoporosis
        vii. Other disease controls
        viii. Other (fill in box):

    d. Control group 2 (if applicable):
        i. Unselected – general population (e.g., blood donors, routine physical, etc.)
        ii. Patients with suspected celiac
        iii. Relatives of celiac patients
        iv. Diabetes
        v. Iron deficiency
        vi. Osteoporosis
        vii. Other disease controls
        viii. Other (fill in box):

e. Control group 3 (if applicable):
    i. Unselected – general population (e.g., blood donors, routine physical, etc)
    ii. Patients with suspected celiac
    iii. Relatives of celiac patients
    iv. Diabetes
    v. Iron deficiency
    vi. Osteoporosis
    vii. Other disease controls
    viii. Other (fill in box):

f. Control group 4 (if applicable):

    i. Unselected – general population (e.g., blood donors, routine physical, etc.)
    ii. Patients with suspected celiac
    iii. Relatives of celiac patients
    iv. Diabetes
    v. Iron deficiency
    vi. Osteoporosis
    vii. Other disease controls
    viii. Other (fill in box):

14. Reference test(s) for cases (i.e., how was celiac diagnosed):
    a. Biopsy (required):
        i. endoscopic
        ii. capsule
            1. list type: text box
        iii. how many samples taken (text box)
    b. Serology (check as many as applicable)
        i. AGA (date >1990)
        ii. EMA
        iii. tTG
    c. Comments:

15. What test was conducted first
    a. Biopsy
    b. Serology
    c. Simultaneous
    d. Mixed
    e. Unsure/other: comment in box

16. Reference test for control (s) 1-----2-----3-----4
    a. Include biopsy (required)
    b. Otherwise excluded

17. Detail biopsy criteria used to define celiac (ESPGAN, Marsh, Rostami) and state what grades were used (i.e., Marsh I and above? etc.)

18. Was IgA deficiency assessed (if applicable):
    a. Yes
    b. No
    c. N/A
    d. Comments: text box)

19. Overall number:

20. Number of:
    a. Cases 1
    b. Cases 2 (if applicable):
    c. Control group 1:
    d. Control group 2 (if applicable):
    e. Control group 3 (if applicable):
    f. Control group 4 (if applicable):

21. Intervention: (***may be up to 8+ tests studied – distinguish IgG from IgA***)

				Results (4x4 table)				
Test name	Methodology	Cut-off (criteria)	Group	a	b	c	d	

22. Stated results if raw data not given:

Test name	Sensitivity	Specificity	PPV	NPV	Prevalence

23. Comments regarding study: test box

Notes for reference:

Test name can be:

1) Anti-htTG
2) Anti-endomysial antibody (EMA)
3) Anti-gliadin antibody (AGA)

		BIOPSY (gold standard) positive	BIOPSY (gold standard) negative
TEST: _____	positive	a	b
	negative	c	d

Calculate automatically

- Prevalence: (a+c)/(a+b+c+d)
- Sensitivity: (a/(a+c));
- Specificity: d/(b+d);
- PPV: a/(a+b)
- NPV: d/(c+d)

Celiac 1: HLA

1. Paper #:
2. Title:
3. Author/year:
4. Reference:
5. Reviewer:

6. Publication type:
    a. Journal
    b. Conference abstract
    c. Other:

7. Is this a duplicate publication (state refid of duplicate):

8. Country:

9. Racial groups and % if different from country: *list in box*

10. Age groups (ped, adult, both):

11. Mean age:

12. Range of age:

13. Percent female:

14. Study type:
    d. Relevant clinical population: (cases and controls defined from the population based on the results of the test under study)
    e. Case control: (groups are predefined and may come from different populations:
    f. Cross-sectional screening study
    g. Other: (list)

15. Type of population (applies to 14 a, c):
    a. Unselected – general population (e.g., blood donors, routine physical etc)
    b. Patients with suspected celiac
    c. Relatives of celiac patients
    d. Diabetes
    e. Iron deficiency
    f. Osteoporosis
    g. Other (fill in box): *not part of extraction – background / discussion only*

16. Case and control group types (applies to 14b):
    h. Celiac group 1
        i. Untreated
            1. Classic
            2. Silent celiac
            3. Atypical celiac
            4. Other
        ii. Treated on GFD
        iii. Refractory (implies on GFD) / ulcerative jejuno-ileitis
        iv. Other/can't tell: text box Celiac group 2

    i. Celiac group 2 – if applicable
        i. Untreated
            1. Classic
            2. Silent celiac
            3. Atypical celiac
            4. Other
        ii. Treated on GFD
        iii. Refractory (implies on GFD) / ulcerative jejuno-ileitis
        iv. Other/can't tell: text box

Note: Control groups must have had a negative biopsy otherwise should have been excluded at level 3

j. Control group 1:
    i. Unselected – general population (e.g., blood donors, routine physical, etc.)
    ii. Patients with suspected celiac
    iii. Relatives of celiac patients
    iv. Diabetes
    v. Iron deficiency
    vi. Osteoporosis
    vii. Other disease controls
    viii. Other (fill in box):

k. Control group 2 (if applicable):
    i. Unselected – general population (e.g., blood donors, routine physical, etc.)
    ii. Patients with suspected celiac
    iii. Relatives of celiac patients
    iv. Diabetes
    v. Iron deficiency
    vi. Osteoporosis
    vii. Other disease controls
    viii. Other (fill in box):

l. Control group 3 (if applicable):
    i. Unselected – general population (e.g., blood donors, routine physical, etc.)
    ii. Patients with suspected celiac
    iii. Relatives of celiac patients
    iv. Diabetes
    v. Iron deficiency
    vi. Osteoporosis
    vii. Other disease controls
    viii. Other (fill in box):

m. Control group 4 (if applicable):

    i. Unselected – general population (e.g., blood donors, routine physical, etc.)
    ii. Patients with suspected celiac
    iii. Relatives of celiac patients
    iv. Diabetes
    v. Iron deficiency
    vi. Osteoporosis
    vii. Other disease controls
    viii. Other (fill in box):

17. Reference test for cases
    n. Biopsy:
        i. endoscopic
        ii. capsule
            1. list type: text box

    o. Serology (check as many as applicable)
        i. AGA (date >1990)
        ii. EMA
        iii. tTG

18. Reference test for control 1-----2------3-----4
    p. Biopsy
    q. Serology (list)
    r. Can't tell

19. Overall number:

20. Number of:
    s. Cases 1
    t. Cases 2 (if applicable):
    u. Control group 1:
    v. Control group 2 (if applicable):
    w. Control group 3 (if applicable):
    x. Control group 4 (if applicable):

21. HLA tested

				Results (4x4 table)				
Test name	Methodology	Cut-off (criteria)	Group	a	b	c	d	

22. Stated results if raw data not given:

Test name	Sensitivity	Specificity	PPV	NPV	Prevalence

23. Narrative result if data not extractable: text box

24. Comments regarding study: text box

Celiac 2: Prevalence and Incidence of Celiac Disease

1. Paper #:
2. Title:
3. Author/year:
4. Reference:
5. Reviewer:

Patient population:

6. Publication type:
    a. Journal
    b. Conference abstract
    c. Etc (your list)

7. Is this a duplicated: list refid

8. Study type:
    a. Cross-sectional prevalence
    b. Cohort
    c. Case control
    d. Incidence study
    e. Other: (list)

9. Country:

10. Racial groups and % if different from country: *list in box*

11. Type of patients screened:
    a. Unselected – general population (e.g., blood donors, routine physical etc)
    b. Patients with suspected celiac
    c. Relatives of celiac patients
    d. Diabetes
    e. Iron deficiency
    f. Osteoporosis
    g. Other (fill in box): *not part of extraction – background / discussion only*

12. Age groups (ped, adult or both): *from level 2 database*

13. Mean age:

14. Range of age:

15. Percent female:

Intervention:

				Results	
Test name	Methodology	# screened	Cases detected	Incidence (time period)	Prevalence

Note: distinguish IgG from IgA. Also the screen may be single test, or combination of tests. So list each strategy used as a “test name”

16. State control reference and methodology of incidence study: (fill in box)

17. Confirmatory test
    a. None
    b. Biopsy
    c. Other serology
    d. Other: fill in box

18. Was IgA deficiency assessed (if applicable):
    a. Yes
    b. No
    c. N/A
    d. Comments: (text box)

19. Comments about study: text box

Celiac 3: Lymphoma

1. Paper #:
2. Title:
3. Author/year:
4. Reference:
5. Reviewer:

6. Publication type:
    a. Journal
    b. Conference abstract
    c. Etc (your list)

7. Study type:
    a. Cross-sectional prevalence
    b. Case series
    c. Cohort
        i. Prospective
        ii. Retrospective
    d. Case control
    e. Other: (list)

8. Country: *from level 2 database*

9. Racial groups and % if different from country: *list in box*

10. Study population type(s) – this is the population of "lymphoma" in case control **OR** the overall population in a screening/prevalence study and cohort studies: Check multiple if study included different populations

    a. Classic celiac:
        1) Treated celiac
        2) Untreated celiac
        3) Non-compliant
        4) Unclear about treatment
    b. Asymptomatic (silent celiac)
    c. Atypical celiac (found on basis of
    d. Latent celiac (normal histology)
    e. Refractory celiac
    f. Ulcerative jejuno-ileitis
    g. Other celiac complications
    h. Patients on Immunosuppression:
    i. Other:

11. How were celiac patients identified (text box)

12. How were cases of lymphoma identified? (i.e., registry, administrative database, etc.) text box

13. Group demographics

	Overall population study type a, b, c	Case group	Control group
Age groups			
Mean age			
Age range			
% female			
Disease duration			

14. For **case control** study:
    a. Control population type (those without lymphoma)
        i. Unselected general population
        ii. Other disease controls: state disease(s)
        iii. A celiac population
            1. Classic celiac:
                a. Treated celiac
                b. Untreated celiac
                c. Non-compliant
                d. Unclear about treatment
            2. Asymptomatic (silent celiac)
            3. Atypical celiac (found on basis of
            4. Latent celiac (normal histology)
            5. Refractory celiac
            6. Ulcerative jejuno-ileitis
            7. Other celiac complications
        iv. Patients on immunosuppression:
    b. # of cases:
    c. # of controls:
    d. Risk factor used to calculate odds ratio
        i. Celiac itself
            1. classic
            2. refractory
            3. ulcerative jejuno-ileitis
        ii. Compliance with diet
        iii. Disease duration
        iv. Other: state in text box
    e. Raw data (if possible)

Table for case control study

Risk factor	Lymphoma present	Lymphoma absent
Present		
Absent		

15. For **cohort study**
    f. Length of F/U of cohort:
    g. Timeline:
        iii. Prospective cohort
        iv. Retrospective cohort:

    h. Risk factor used to calculate Relative Risk if one population used
        v. Celiac itself
            1. classic
            2. atypical
            3. silent
            4. refractory
            5. ulcerative jejuno-ileitis
        vi. Compliance with diet
        vii. Disease duration
        viii. Other: state in text box

    i. If a celiac cohort was compared to another population to obtain another risk estimate (i.e., standardized mortality or morbidity ratios). Describe control population.

        ix. Unselected general population
        x. Other disease controls: state disease(s)
        xi. A celiac population
            1. Classic celiac:
                a. Treated celiac
                b. Untreated celiac
                c. Non-compliant
                d. Unclear about treatment
            2. Asymptomatic (silent celiac)
            3. Atypical celiac (found on basis of
            4. Latent celiac (normal histology)
            5. Refractory celiac
            6. Ulcerative jejuno-ileitis
            7. Other celiac complications
        xii. Patients on immunosuppression
        xiii. Other: (fill in text box)

    j. Overall number in cohort:
        xiv. Number of cases identified:
    k. Number in control population (if applicable)
        xv. Number of lymphomas identified
        xvi. Raw data if available:

16. Table for classic cohort study

Risk factor	Lymphoma present	Lymphoma absent
Present		
Absent		

17. For cross sectional studies: study population in Q #10
    l. Overall number screened:
    m. Number of cases identified:

18. Results (text boxes)
    n. Lymphoma type
    o. Prevalence of lymphoma in celiac:
    p. Incidence of lymphoma in celiac:
    q. Odds ratio (95% confidence interval)
    r. Relative risk (95% confidence interval):
    s. Standardized mortality ratio
    t. Standardized morbidity ratio
    u. Other risk estimate:

19. Study comments:

Celiac 4: Consequences of Testing

1. Paper #:
2. Title:
3. Author/year:
4. Reference:
5. Reviewer:

Patient population:

6. Publication type:
    a. Journal
    b. Conference abstract
    c. Etc (your list)

7. Study type:
    a. Diagnostic test
    b. Cross-sectional prevalence
    c. Cohort
    d. Case control
    e. Other: (list)

8. Country: *from level 2 database*

9. Racial groups and % if different from country: *list in box*

10. Type of patients tested (*from level 2 database)*
    a. Unselected – general population (e.g., blood donors, routine physical, etc.)
    b. Patients with suspected celiac
    c. Relatives of celiac patients
    d. Diabetes
    e. Iron deficiency
    f. Osteoporosis
    g. Other (fill in box):

11. Type of celiac patients identified:
    a. Classic celiac
    b. Asymptomatic
    c. Atypical celiac
        i. Fe deficiency
        ii. Osteoporosis
        iii. Other
    d. Complicated celiac
        i. Refractory
        ii. Jejuno-ileitis
        iii. Lymphoma)

e. Other

12. Intervention:
    a. Test(s) used to identify celiac patients:
        i. Biopsy
        ii. AGA ELISA (publication date >1990)
        iii. AMA
        iv. tTG:

13. Was IgA deficiency assessed (if applicable):
    a. Yes
    b. No
    c. N/A
    d. Comments: text box)

14. Length of F/U:

15. How were patients followed (if applicable): test box

16. Outcomes:

Outcome	**Result**	**Notes**
Costs		
Cases diagnosed		
Patients complying with treatment		
Response to treatment		
Clinical outcome (reduced risk of complication, etc)		
Other (fill in box)		

17. Comments regarding study: Fill in text box

Celiac 5: Promoting and Monitoring Adherence to Gluten-Free Diet

1. Paper #:
2. Title:
3. Author/year:
4. Reference:
5. Reviewer:

Patient population:

6. Publication type:
    a. Journal
    b. Conference abstract
    c. Etc (your list)

7. Study type:
    a. Diagnostic test
    b. Cross-sectional prevalence
    c. Cohort
    d. Case control
    e. Other: (list)

8. Country: *from level 2 database*

9. Racial groups and % if different from country: *list in box*

10. Age groups (ped, adult, both): *from level 2 database*

11. Mean age:

12. Range of age:

13. Percent female:

14. Disease duration: state in box

15. Type of celiac studied
    a. Classic celiac
    b. Asymptomatic celiac
    c. Atypical celiac (Fe deficiency, osteoporosis, etc.)
    d. Refractory celiac
    e. Ulcerative jejuno-ileitis
    f. IgA deficient celiac
    g. Other:

16. Does this refer to:

a. Promoting adherence
b. Monitoring adherence
c. Both

17. What intervention was assessed? (if **monitoring adherence**)

	Result (normalization of biopsy or drop in antibody titres - list result details)
Biopsy	
Antibody testing (state test used)	
Other: _____	

18. Did this study determine the sensitivity/specificity of the intervention during follow-up or with different histologic grades:
    a. No
    b. Yes: detail in text box

19. Was IgA deficiency assessed (if applicable):
    a. Yes
    b. No
    c. N/A
    d. Comments: text box

20. What intervention was used? (if **promoting adherence**) – fill in box

Intervention	Result

Comments about the study: fill in text box

Appendix D. Quality Assessment Forms

QUADAS Checklist

Item		Yes	No	Unclear
1.	Was the spectrum of patients representative of the patients who will receive the test in practice?			
2.	Were selection criteria clearly described?			
3.	Is the reference standard likely to correctly classify the target condition?			
4.	Is the time period between reference standard and index test short enough to be reasonably sure that the target condition did not change between the two tests?			
5.	Did the whole sample or a random selection of the sample, receive verification using a reference standard of diagnosis?			
6.	Did patients receive the same reference standard regardless of the index test result?			
7.	Was the reference standard independent of the index test (i.e. the index test did not form part of the reference standard)?			
8a.	Was the execution of the index test described in sufficient detail to permit replication of the test?			
8b.	Was the execution of the reference standard described in sufficient detail to permit its replication?			
9a.	Were the index test results interpreted without knowledge of the results of the reference standard?			
9b.	Were the reference standard results interpreted without knowledge of the results of the index test?			
10.	Were the same clinical data available when test results were interpreted as would be available when the test is used in practice?			
11.	Were uninterpretable/ intermediate test results reported?			
12.	Were withdrawals from the study explained?			

Cross-Sectional/Prevalence Study Quality

Item	Yes	No	Unclear
1) Define the source of information (survey, record review)			
2) List inclusion and exclusion criteria for exposed and unexposed subjects (cases and controls) or refer to previous publications			
3) Indicate time period used for identifying patients			
4) Indicate whether or not subjects were consecutive if not population-based			
5) Indicate if evaluators of subjective components of study were masked to other aspects of the status of the participants			
6) Describe any assessments undertaken for quality assurance purposes (e.g., test/retest of primary outcome measurements)			
7) Explain any patient exclusions from analysis			
8) Describe how confounding was assessed and/or controlled.			
9) If applicable, explain how missing data were handled in the analysis			
10) Summarize patient response rates and completeness of data collection			
11) Clarify what follow-up, if any, was expected and the percentage of patients for which incomplete data or follow-up was obtained			

Newcastle–Ottawa Quality Assessment Scale: Cohort Studies

Note: a study can be awarded a maximum of one star for each numbered item within the selection. A maximum of two stars can be given for comparability and selection.

1) Representativeness of the exposed cohort
   a) truly representative of the average _____ (describe) in the community ☸
   b) somewhat representative of the average _____ in the community ☸
   c) selected group of users e.g. nurses, volunteers
   d) no description of the derivation of the cohort
2) Selection of the non exposed cohort
   a) drawn from the same community as the exposed cohort ☸
   b) drawn from a different source
   c) no description of the derivation of the non exposed cohort
3) Ascertainment of exposure
   a) secure record (e.g. surgical records) ☸
   b) structured interview ☸
   c) written self report
   d) no description
4) Demonstration that outcome of interest was not present at start of study
   a) yes ☸
   b) no

Comparability
1) Comparability of cohorts on the basis of the design or analysis
   a) study controls for _____ (select the most important factor) ☸
   b) study controls for any additional factor ☸ (This criteria could be modified to indicate specific control for a second important factor.)

Outcome
1) Assessment of outcome
   a) independent blind assessment ☸
   b) record linkage ☸
   c) self report
   d) no description
2) Was follow-up long enough for outcomes to occur
   a) yes (select an adequate follow up period for outcome of interest) ☸
   b) no
3) Adequacy of follow up of cohorts
   a) complete follow up - all subjects accounted for ☸
   b) subjects lost to follow up unlikely to introduce bias - small number lost - > ____ % (select an adequate %) follow up, or description provided of those lost) ☸
   c) follow up rate < ____% (select an adequate %) and no description of those lost
   d) no statement

Newcastle–Ottawa Quality Assessment Scale: Case Control Studies

Note: a study can be awarded a maximum of one star for each numbered item within the selection and exposure categories. A maximum of two stars can be given for comparability.

Selection

1) Is the case definition adequate?
   a) yes, with independent validation ☸
   b) yes, e.g. record linkage or based on self reports
   c) no description
2) Representativeness of the cases
   a) consecutive or obviously representative series of cases ☸
   b) potential for selection biases or not stated
3) Selection of Controls
   a) community controls ☸
   b) hospital controls
   c) no description
4) Definition of Controls
   a) no history of disease (endpoint) ☸
   b) no description of source

Comparability

1) Comparability of cases and controls on the basis of the design or analysis
   a) study controls for _____ (Select the most important factor.) ☸
   b) study controls for any additional factor ☸ (This criteria could be modified to indicate specific control for a second important factor.)

Exposure

1) Ascertainment of exposure
   a) secure record (e.g. surgical records) ☸
   b) structured interview where blind to case/control status ☸
   c) interview not blinded to case/control status
   d) written self report or medical record only
   e) no description
2) Same method of ascertainment for cases and controls
   a) yes ☸
   b) no
3) Non-Response rate
   a) same rate for both groups ☸
   b) non respondents described
   c) rate different and no designation

Appendix E. Summary ROC Curves

Summary ROC curves as calculated by the methods of Moses and Shapiro (meta-analysis text). For all figures below, the middle curve is the summary ROC, the other curves are upper and lower 96% CI, and the vertical line is the point were sensitivity = specificity; dots are individual studies

Figure 1. Summary ROC HLA average-risk with 95% CIs

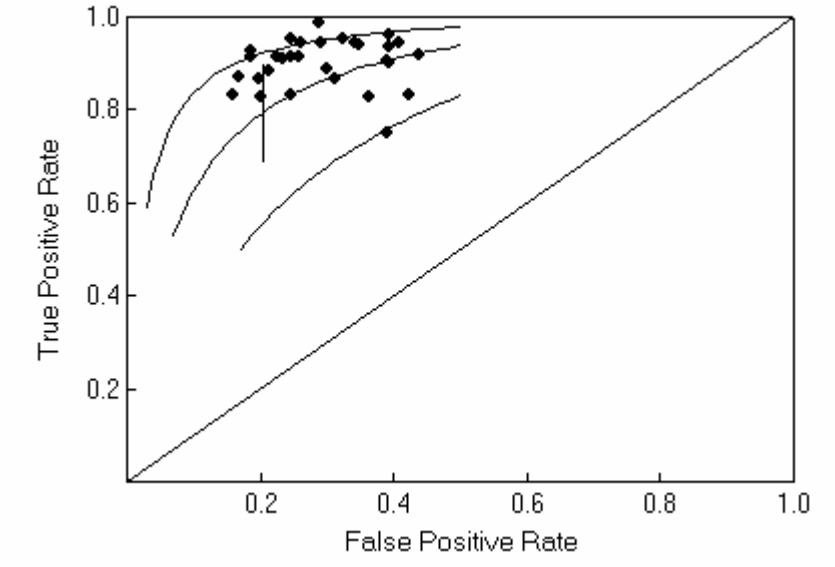


Figure 2. Summary ROC IgA-AGA–adults

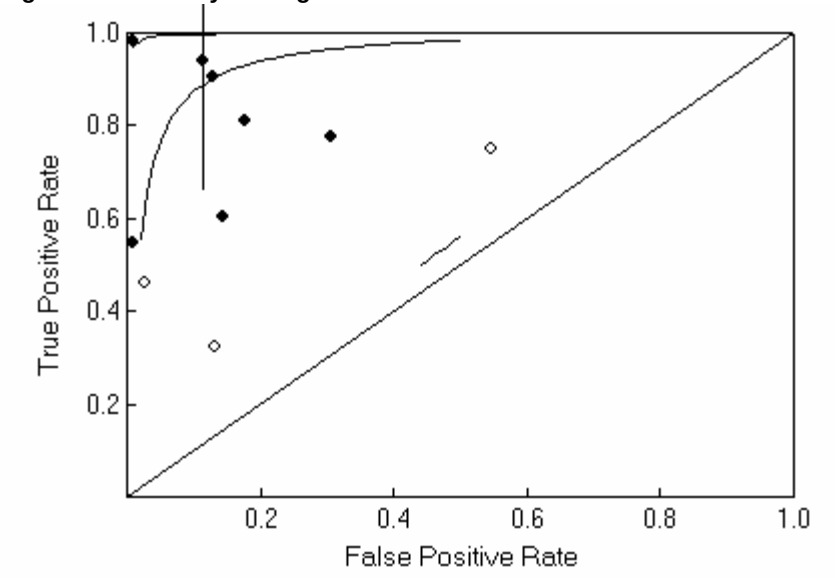


Figure 3. Summary ROC IgG-AGA–adults

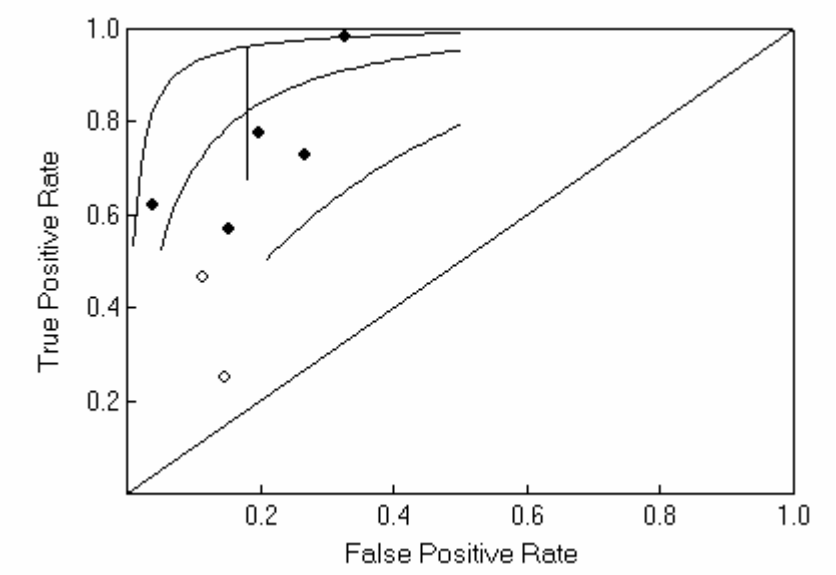


Figure 4. Summary ROC IgA-EMA-EM–adults

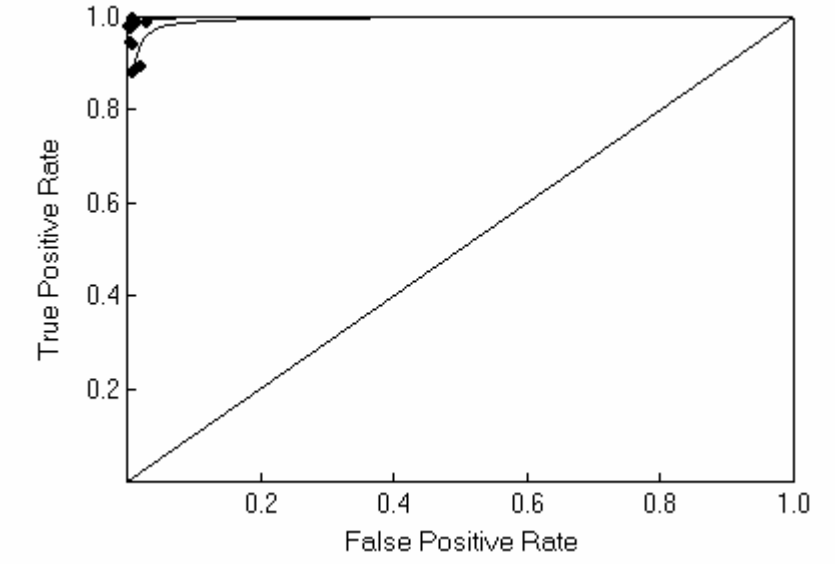


Figure 5. Summary ROC IgA-EMA-HU–adults

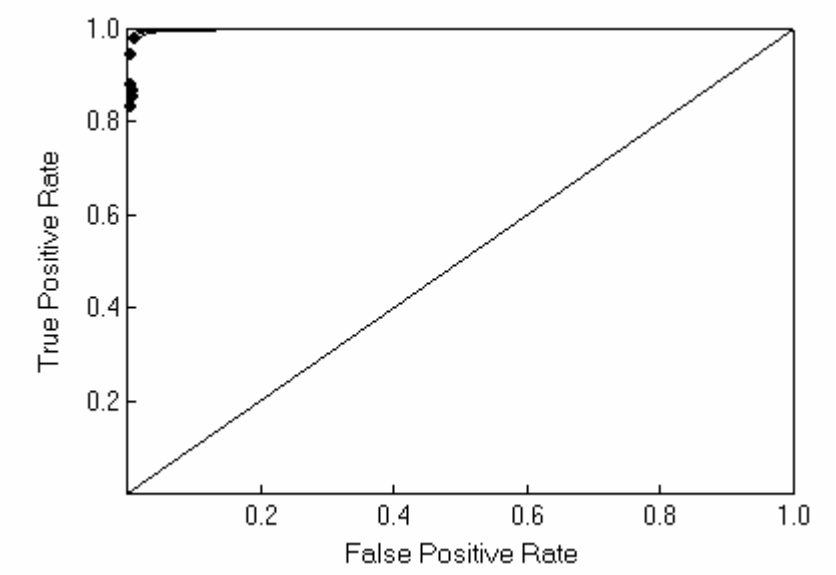


Figure 6. Summary ROC IgA-tTG-GP–adults

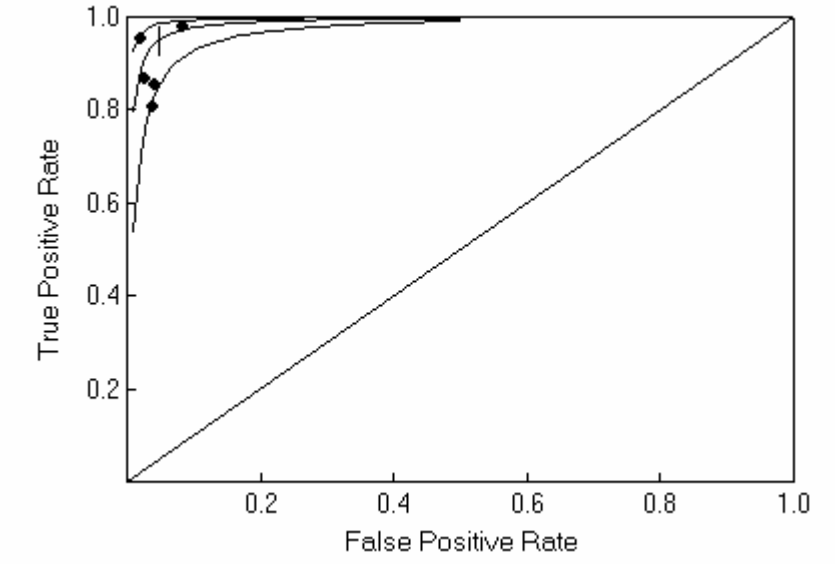


Figure 7. Summary ROC IgA-tTG-HR–adults

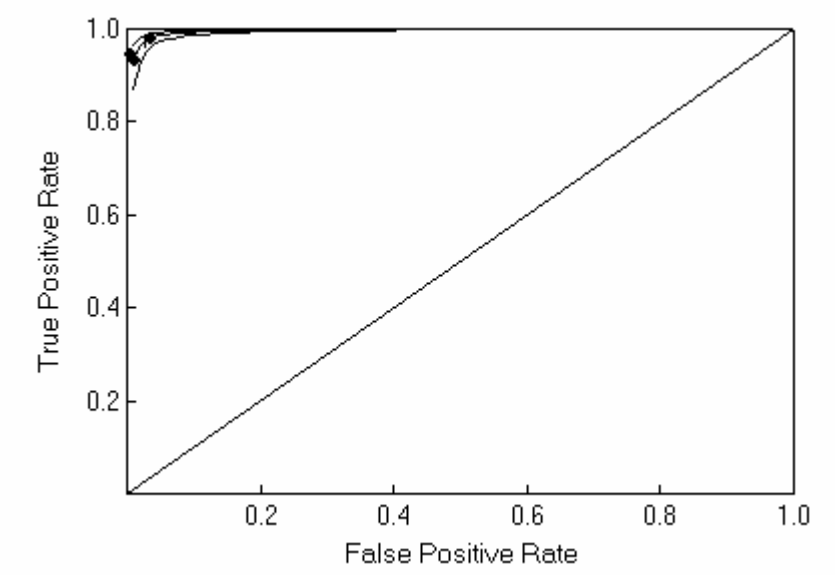


Figure 8. Summary ROC IgA-AGA–child

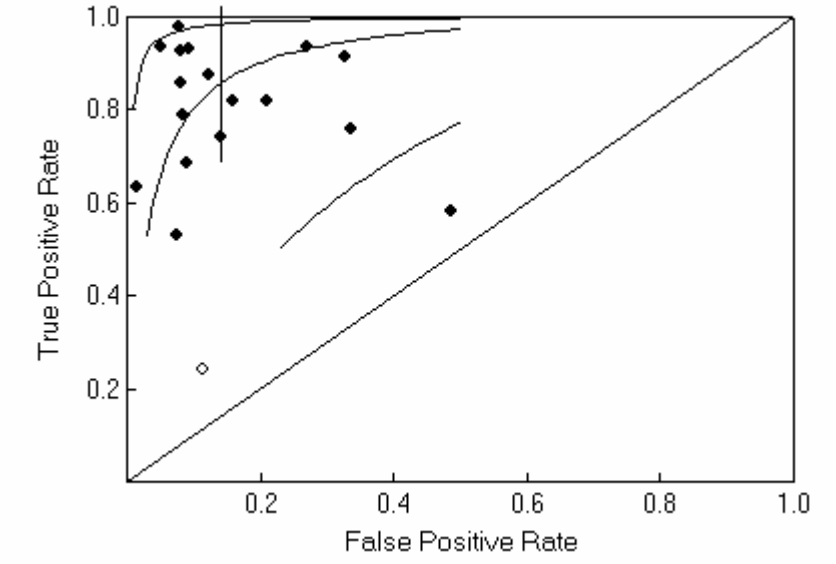


Figure 9. Summary ROC IgG-AGA–child

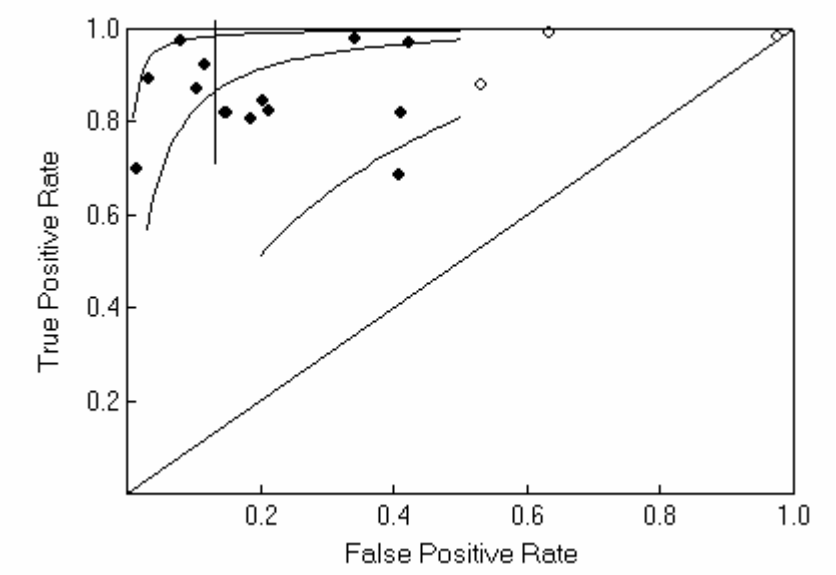


Figure 10. Summary ROC IgA-EMA-ME–child

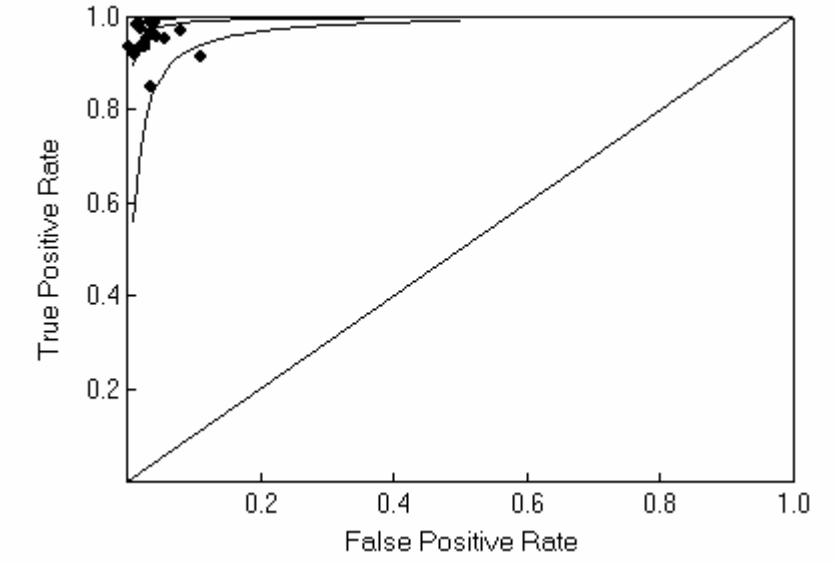


Figure 11. Summary ROC IgA-EMA-HU–child

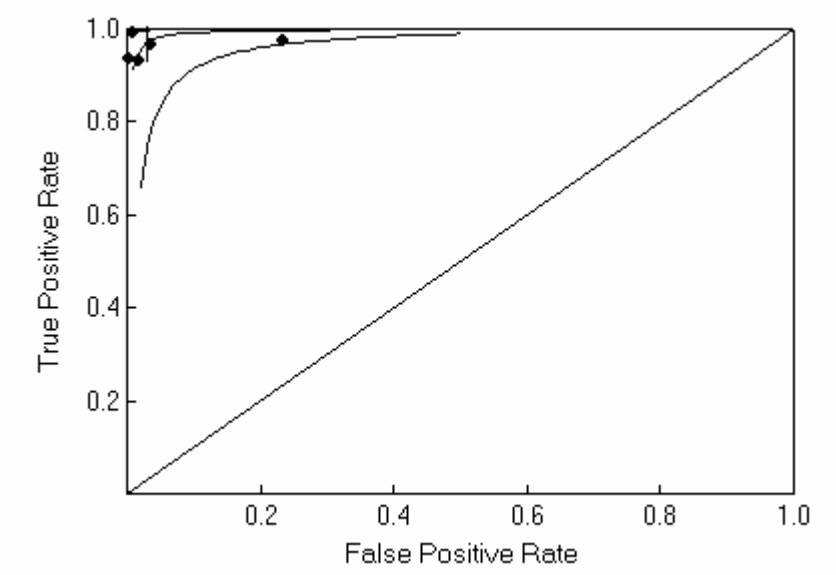


Figure 12. Summary ROC IgA-tTG-GP–child

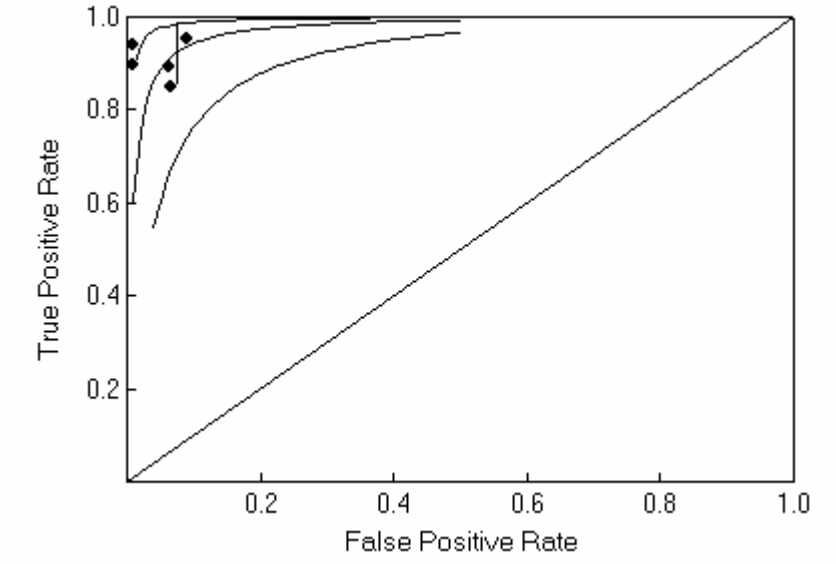


Figure 13. Summary ROC IgA-tTG-HR–child

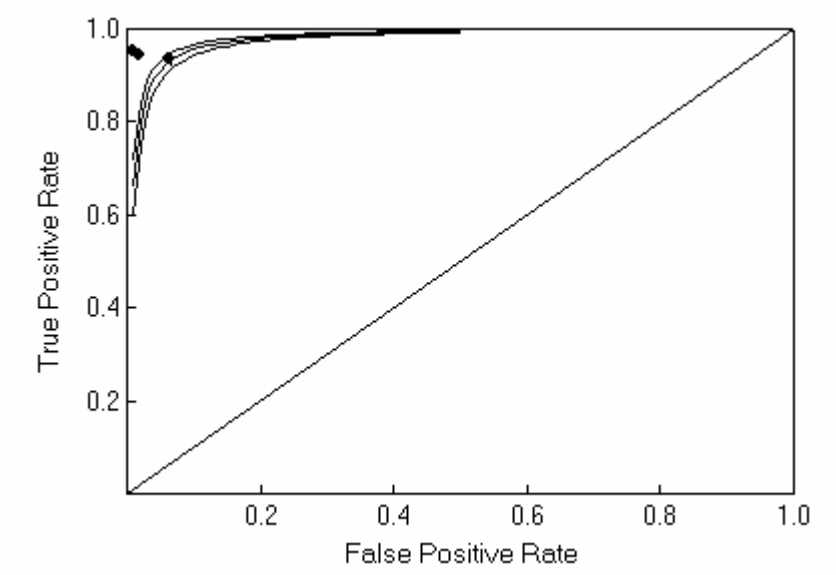


Figure 14. Summary ROC IgA-AGA–mixed-age populations

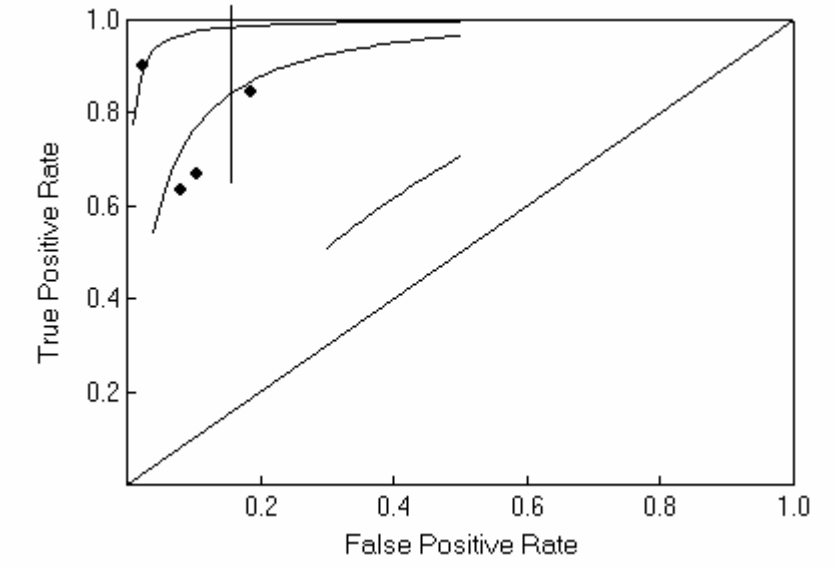


Figure 15. Summary ROC IgG-AGA–mixed-age populations

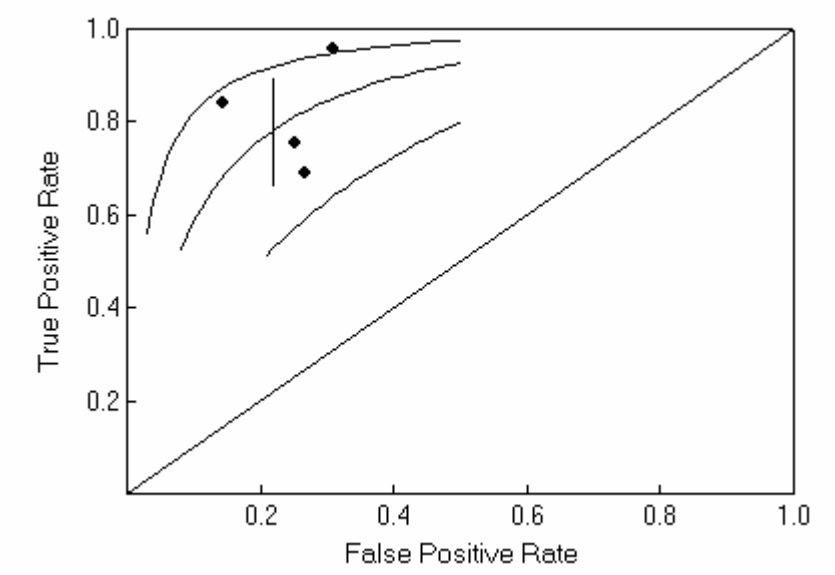


Figure 16. Summary ROC IgA-EMA-ME–mixed-age populations

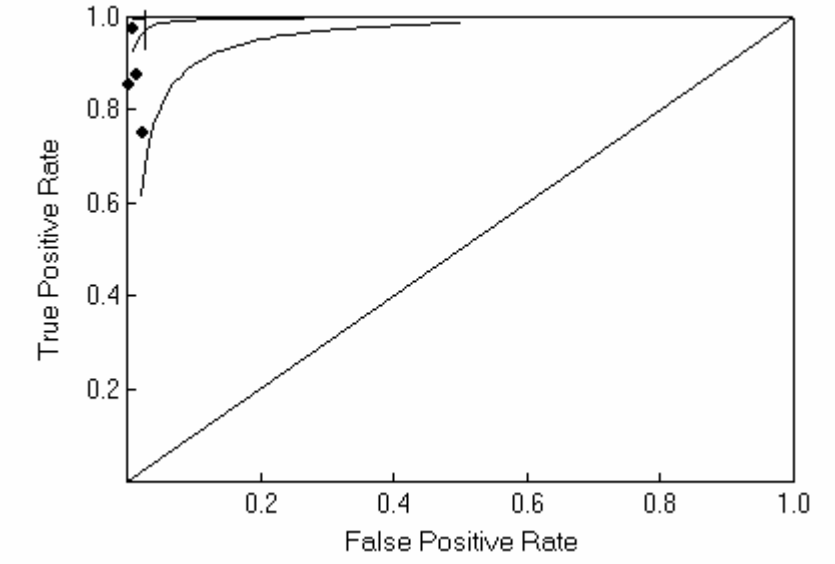
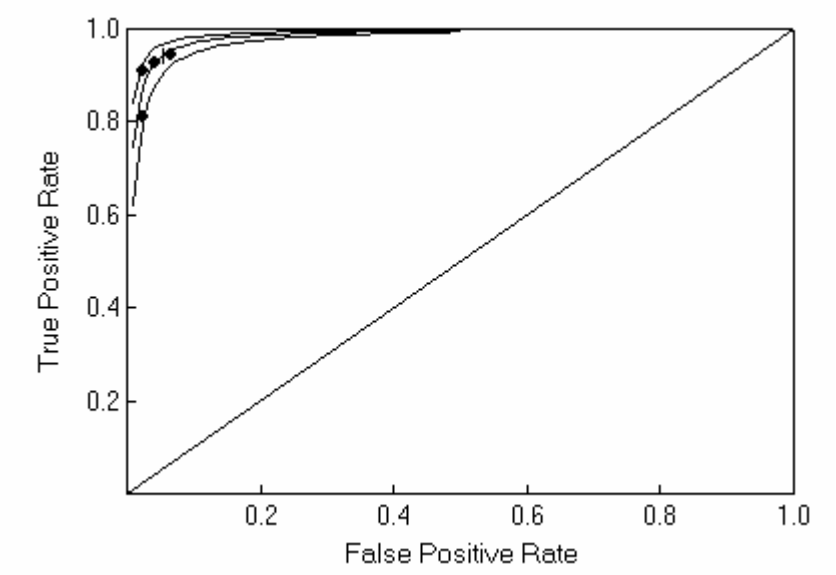


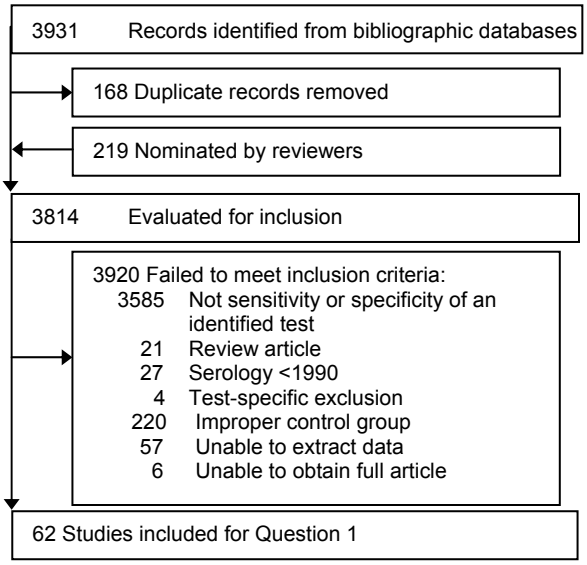
Figure 17. Summary ROC IgA-tTG-GP–mixed-age populations



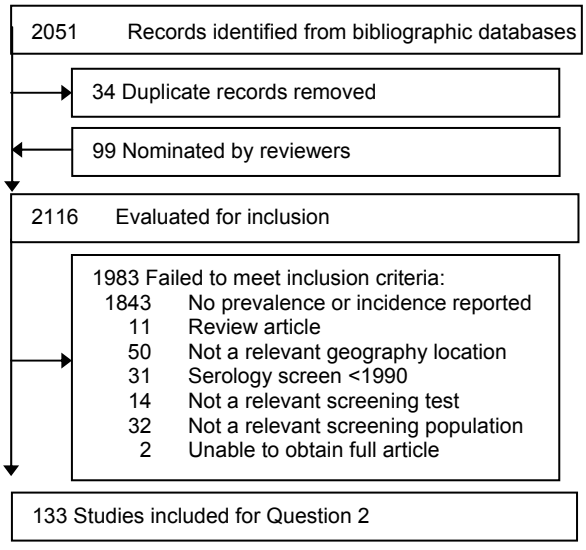
Appendix F. Modified QUOROM Flow Chart

Modified QUOROM Flow Charts

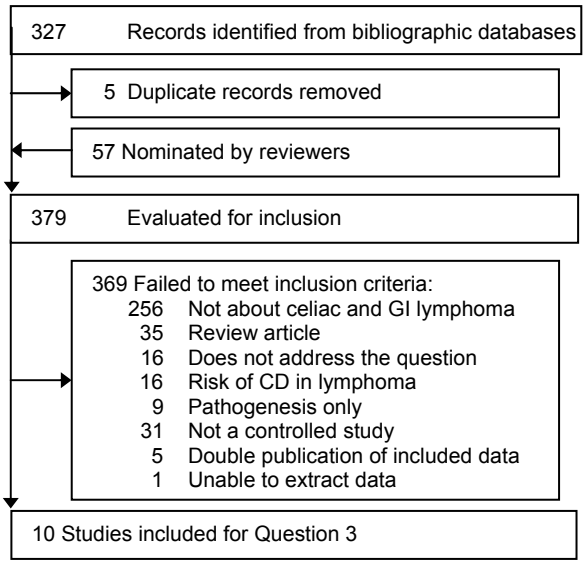
Objective 1 – Sensitivity and Specificity of Tests for CD



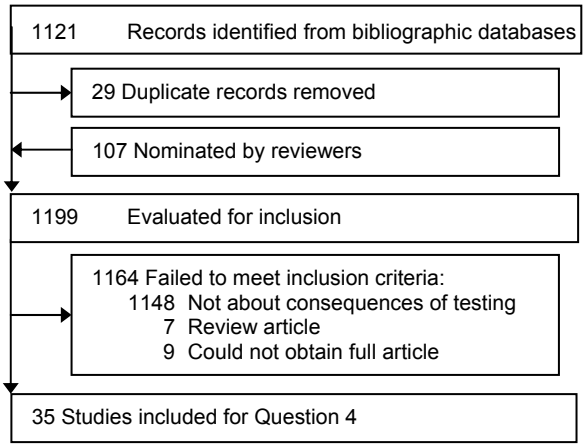
Objective 2 – Prevalence and Incidence of CD



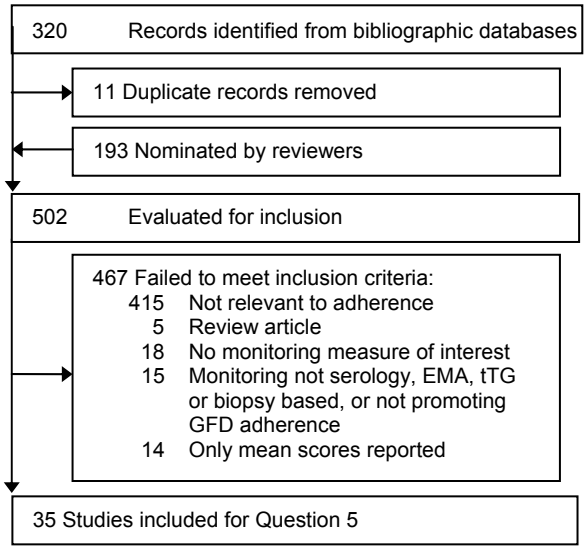
Objective 3 – Celiac Associated Lymphoma



Objective 4 – Expected Consequences of Testing for CD



Objective 5 – Promoting or Monitoring Adherence to a GFD



Appendix G. Raw Pooled Data

All raw pooled data by antibody test and study types showing number of studies and total patient numbers.

AGA - ELISA														
Population	Pooled Number		IgA					Pooled Number		IgG				
	Studies	Patients	Sens	Spec	PPV	NPV	Prev	Studies	Patients	Sens	Spec	PPV	NPV	Prev
Case Control														
Adults	2	336	57.9	92.6	93.2	55.7	0.40	1	157	78.0	80.7	87.6	67.6	56.7
Children	5	412	76.6	79.9	81.1	75.2	0.5	5	412	84.4	60.3	70.5	77.5	0.5
Both	1	343	84.6	81.6	75.2	88.9	0.4	1	343	69.1	73.4	63.1	78.4	0.4
Relevant CP														
Adults	8	946	71.0	81.1	61.7	86.8	0.35	6	652	65.6	85.7	70.7	82.7	31.9
Children	14	1382	80.1	89.9	78.7	90.7	0.3	12	1189	89.3	83.6	68.1	95.2	0.3
Both	3	737	68.2	92.7	91.5	71.5	0.5	3	737	83.8	79.5	82.6	80.9	0.5

EMA – Monkey Esophagus IF														
Study Population	Pooled Number		IgA					Pooled Number		IgG				
	Studies	Patients	Sens	Spec	PPV	NPV	Prev	Studies	Patients	Sens	Spec	PPV	NPV	Prev
Case Control														
Adults	6	706	97.3	100.0	100.0	97.1	50.7	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Children	12	1038	96.5	98.7	98.6	96.6	0.5	1*	153	100	100	100	100	0.1
Both	1	131	75.3	98.3	98.2	76.0	0.6	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Relevant CP														
Adults	5	692	95.0	99.4	98.1	98.5	22.4	1	89	39.3	98.4	91.7	77.9	0.14
Children	6	868	93.2	95.4	88.3	97.4	0.3	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Both	3	737	87.9	99.7	99.7	87.6	0.5	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Note: *this study was conducted in CD patients known to be IgA-EMA negative, and 50% had IgA deficiency														

EMA – Human Umbilical Cord - IF														
Population	Pooled Number		IgA					Pooled Number		IgG				
	Studies	Patients	Sens	Spec	PPV	NPV	Prev	Studies	Patients	Sens	Spec	PPV	NPV	Prev
Case Control														
Adults	5	578	90.3	100.0	100.0	93.7	37.2	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Children	4	375	96.6	100.0	100.0	97.1	0.5	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Both	2	428	92.5	99.6	99.5	93.8	0.5	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Relevant CP														
Adults	1	92	87.5	100.0	100.0	98.8	7.6	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Children	2	172	70.5	86.7	64.6	89.5	0.3	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Both	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a

tTG - Guinea Pig Liver - ELISA														
Population	Pooled Number		IgA					Pooled Number		IgG				
	Studies	Patients	Sens	Spec	PPV	NPV	Prev	Studies	Patients	Sens	Spec	PPV	NPV	Prev
Case Control														
Adults	3	368	83.4	97.1	97.0	84.2	45.1	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Children	3	270	92.3	99.2	99.2	92.0	0.5	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Both	3	559	91.2	94.7	94.3	91.8	0.5	1	85	61.5	100.0	100.0	44.4	0.8
Relevant CP														
Adults	2	327	100.0	94.2	66.7	100.0	15.6	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Children	2	176	95.1	93.0	87.9	97.3	0.3	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Both	1	111	91.7	98.4	97.8	93.9	0.4	1	111	22.9	98.4	91.7	62.6	0.4

tTG – Human Recombinant - ELISA														
Population	Pooled Number		IgA					Pooled Number		IgG				
	Studies	Patients	Sens	Spec	PPV	NPV	Prev	Studies	Patients	Sens	Spec	PPV	NPV	Prev
Case Control														
Adults	1	63	95.2	100.0	100.0	97.7	31.7	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Children	2	115	95.3	98.0	98.4	94.3	0.6	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Both	1	85	90.8	100.0	100.0	76.9	0.8	1	85	67.7	100.0	100.0	48.8	0.8
Relevant CP														
Adults	2	299	100.0	97.8	84.2	100.0	12.7	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Children	1	101	96.2	100.0	100.0	96.1	0.5	n/a	n/a	n/a	n/a	n/a	n/a	n/a
Both	1	426	90.0	94.9	96.2	87.0	0.6	n/a	n/a	n/a	n/a	n/a	n/a	n/a

Appendix H. Biopsy Results

Relationship of Serology to Histology

CD clearly exists in patients with histological grades milder than Marsh IIIa, and given that the sensitivity of biopsy is improved by using a lower grade as a cut-off, an important question arises—what test is most sensitive for detecting CD with mild histologic changes, biopsy or serology?

Fasano,[1] in a large American prevalence study of CD in at risk and not at risk populations, found that only 34% of biopsied EMA-positive subjects had subtotal or total VA (modified Marsh IIIb or IIIc). In this study, no EMA-positive patient had a Marsh 1 lesion, 26% of EMA-positive patients had a Marsh II lesion, and 40% had a Marsh IIIa lesion. All newly-diagnosed, EMA-positive CD patients with (n=98) or without (n=114) biopsy had HLA DQ2, DQ8 or both, as apposed to 59% of EMA-negative subjects (n=92). The results of this study once again suggest that applying a criterion of subtotal or total VA would miss 66% of CD patients. The absence of Marsh I lesions in EMA-screened subjects is not surprising (discussed below), given the lower sensitivity of this test in lower-grade histologic lesions of CD, suggesting that the CD may have been unrecognized in some EMA-negative subjects. Unfortunately, HLA was not evaluated in all subjects and assessment of the correlation with serology in the population at large or systematically with biopsy grade was not reported.

Rostami et al.[2] evaluated the diagnostic value of IgA EMA and AGA in 101 untreated CD patients. The diagnosis of CD was made on the basis of "appropriate histopathological features" (Marsh IIIa or greater) and clinical improvement on a gluten-free diet (GFD). Sixteen first-degree relatives with minor histologic abnormalities (Marsh I-II) were used as controls. Sixteen patients were excluded for not meeting diagnostic criteria, IgA deficiency, or undergoing serology while on GFD.

Rostami et al.[2]	Marsh I-II (controls)	Marsh IIIa	Marsh IIIb	Marsh IIIc
Biopsy	16	29 (%)	23 (%)	17 (%)
AGA	3 (21%)	9 (31%)	16 (70%)	14 (82%)
EMA	0	9 (31%)	16 (70%)	17 (100%)

The combination of the two tests showed an overall sensitivity of 76%. Unfortunately, the authors, as is commonly done, considered Marsh I-II as controls; it is unclear if any of these, particularly those who were AGA positive, actually have CD. As will be described below, there is a subset of patients with Marsh I-II who are serology negative who have CD. In any case, this study demonstrates an important finding, i.e., that the sensitivity of the studied serological markers varies with the severity of the histologic grade. Alarmingly, the sensitivity even for CD patients with Marsh IIIa lesions is close to 30%. This is partially at odds with the results of the Fasano study[1] where only 34% of the identified patients were found to have Marsh IIIb or greater

grade lesions, with the rest having grade II to IIIa lesions. In both studies, no EMA-positive Marsh I lesions were found. The Fasano study, being a population-based screening study, obviously did not biopsy all screened patients. This begs the question of how many grade IIIb or less patients with CD were missed based on the findings of Rostami and Tursi (detailed below).

Tursi et al.[3] assessed the relationship of the histologic grade of 119 consecutive adult patients with CD defined by characteristic duodenal biopsy and "permanent gluten-sensitive enteropathy." The following table summarizes the main findings.

Tursi et al.[3]	Marsh I	Marsh II	Marsh IIIa	Marsh IIIb	Marsh IIIc
Biopsy	13 (11%)	24 (20%)	27 (23%)	31 (26%)	24 (20%)
tTG positive	1 (8%)	8 (33%)	15 (56%)	26 (84%)	23 (96%)
Mean tTG level UA/mL	7.3	18.5	n/a	36	74.95

In this study, 69% of CD patients had VA (Marsh IIIa or greater). The frequency of tTG-positivity (sensitivity) and mean tTG levels were greatest with the highest Marsh grade and dropped steadily with milder histologic grades, reaching a low of only 8%-positivity in CD patients with Marsh I lesions. Since these patients all have "permanent gluten-sensitive enteropathy," it is clear that tTG would have missed 76% of this cohort of CD patients with Marsh I or II lesions who were picked up by biopsy.

Tursi et al.,[4] also assessed 123 adult patients (possibly the same patients cohort from the above study) with either subclinical (equivalent to atypical in this review) or silent CD. All patients were biopsied and CD was diagnosed on the basis of "permanent-gluten sensitive enteropathy", and histology was classified with the modified Marsh criteria. The subclinical group included patients with associated CD conditions such as iron deficiency but without GI symptoms, while silent CD patients were asymptomatic patients screened in at risk groups such as first-degree relatives or type 1 diabetes. EMA was positive in 77/96 (80.8%) of subclinical CD cases and 17/27 (63.0%) of silent CD cases. EMA was negative in patients with Marsh I lesions. Once again, assuming that all these patients with "permanent gluten-sensitive enteropathy" are truly CD patients, then EMA would miss 19% of subclinical CD patients, and 37% of silent CD that were picked-up by biopsy.

In what appears to be a partial duplicate publication, Tursi et al.[5] demonstrated similar results with AGA, and EMA in 115 patients with subclinical or silent CD.

Patients with subclinical CD

Tursi et al[5]	Marsh I	Marsh II	Marsh IIIa	Marsh IIIb	Marsh IIIc
Biopsy	2	10	18	25	30
AGA pos.	0	3 (30%)	14 (77%)	21 (84%)	27 (90%)
EMA pos	0	4 (40%)	16 (88.9%)	23 (92%)	29 (96.7%)

Patients with silent CD

Tursi et al[5]	Marsh I	Marsh II	Marsh IIIa	Marsh IIIb	Marsh IIIc
Biopsy	2	2	5	6	9
AGA pos.	0	0	3 (60%)	4 (66.7%)	7 (77.8%)
EMA pos	0	0	4 (80.0%)	5 (83.3%)	8 (88.9%)

As before, in this group of CD patients, serology would miss patients that would be picked up by biopsy.

Demir et al.[6] (Celiac 4) studied the presentation and clinical features of 104 newly diagnosed Turkish children. EMA and biopsy correlation was available for 72 children. Similar to what was described above, EMA was positive in 92% of children with Marsh III compared with 66.6% of children with Marsh I-II.

Kotze et al.[7,8] assessed 47 symptomatic subjects with CD with intestinal biopsy, tTG and EMA antibodies. Forty were suspected of having CD (9 were children) and the investigations were performed together, while seven were biopsy-diagnosed CD who were already on a GFD. Both EMA and tTG antibodies were negative in these seven patients. The findings of the 40 suspected CD patients are presented in the following table.

Kotze et al[7,8]							
Normal biopsy	7	8	2		1 (child)		
Partial VA			1		1		
Total VA			1	3	7	8 (child)	8
Mean tTG titer	8.14	11.87	41.5	181.7	356.3	307.4	432.8
Mean EMA titer	neg	1/2.5	1/5	1/10	1/20	1/40	1/80
Notes: Titers of EMA and tTg antibodies of 1/2.5 and 20 U, respectively VA= VA							

The authors used an older histology grading system, and did not systematically report on the overall number of "normal biopsies" with raised IELs. They also did not report on the number of subjects with the Marsh II hyperplastic lesion, nor did they distinguish between Marsh IIIa and IIIb (lumped as "partial VA"). Nonetheless, the authors report that in the eight subjects with positive-EMA antibodies (>1/2.5) yet negative for tTG antibodies (<20), the mucosa showed normal villous structure but raised levels of IELs. These eight subjects responded to a GFD and were considered to have CD. The correlation between the two serological tests was high (Pearson's Chi square [the large R is 'accountable' variance]; r=0.797). However, the same finding as in the previous studies is repeated again. CD occurred in eight patients with negative tTG antibodies, and the titres of both EMA and tTG antibodies correlated with histologic grade, once again suggesting that serology alone would miss CD patients who would be picked-up by biopsy. This is a very recent study and it would shed a great deal of light on the false-positive and negative-rate of biopsy if the authors would publish a follow-up study on: (1) the status of the three subjects who were positive for EMA and tTG antibodies yet had "normal" biopsies (IEL status not reported); (2) the seven subjects who were negative for all tests; and (3) the histologic and clinical response to GFD in those who were diagnosed with CD.

Hoffenberg et al.[9] studied a group of children at risk of CD who were part of a large prospective study of the genetic and environmental factors associated with autoimmune diseases. For the CD portion, newborns were screened for the presence of HLA DR3/3, DR3/4, or DR3/x as markers for DQ2. In another group, at risk children with type I diabetes, first-degree relatives of type 1 diabetics, and first-degree relatives of CD patients, were studied. Thirty anti-tTG positive subjects among these screened patients were enrolled in the study (14 diabetics, 11 first-degree relatives, and five HLA DR3). All 30 children underwent Marsh biopsy grading. No relationship was found between Marsh grade and the genetic risk factor leading to screening. A significant correlation was found between Marsh grade and anti-tTG (r=0.57, p<0.01). The calculated mean anti-tTG titers are presented in parentheses in the table below.

Biopsy results of 30 tTG-positive children

Hoffenberg et al[9]	Marsh 0	Marsh I	Marsh II	Marsh III
Biopsy	5 (16.7%)	4 (13.3%)	2 (6.7%)	19 (63%)
Mean tTG level UA/mL	< 0.6	< 0.6	< 0.6	> 0.6 (0.70)

Unlike the other studies presented in this series, this study selected patients at risk of CD who were anti-tTG positive. Unfortunately, this makes direct comparisons difficult, but in essence this study supports the notion of a greater sensitivity of tTG in high-grade histologic lesions through the finding that high-grade lesions are associated with higher anti-tTG titres.

In a small case control study assessing the diagnostic value of EMA, Sategna-Guidetti,[10] also found that in patients with documented CD, EMA positivity correlated with the severity of the histologic grade. In this study, EMA was falsely negative in 50% of CD patients without VA.

Other Histological Features

Several other histological features have been studied in an attempt to improve the accuracy of biopsy in the diagnosis of CD. Some of these features include: assessment of small bowel mucosal mast cells,[11-14] mucosal fat,[15] and endocrine cell hyperplasia.[16] Discussion of these features is beyond the scope of this review.

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Appendix I. Evidence Tables

List of abbreviations used in the evidence tables

Ab=antibody AGA=anti-gliadin antibodies ARA=antireticulin antibodies Bx=biopsy CD=celiac disease CI=confidence interval CO=control Dx=diagnosis EGD=esophagogastroduodenoscopy EIA=enzyme immunoassay EMA=anti-endomysium antibodies F=female GC=gluten challenge GCD=gluten-containing diet GERD=gastroesophageal reflux disease GFD=gluten-free diet GI=gastrointestinal GP=guinea pig Hb-hemoglobin HLA=human leukocyte antigens HU=human umbilical cord HUVEC= human umbilical vein endothelial cells JAB=human jejunal antibodies IBS=irritable bowel syndrome IDA=iron deficiency anemia IDDM=insulin-dependent diabetes mellitus IF=immunofluorescence M=male ME=monkey esophagus mos=months	n=number of patients n/a=not applicable NHL=non-Hodgkin's lymphoma n/r=not reported OR=odds ratio pt=patient RR=relative risk SD=standard deviation TGA=anti-thyroglobulin tTG=anti-tissue transglutaminase UTCD=untreated celiac disease VA=villous atrophy y=year

Celiac 1: Sensitivity and Specificity of Tests for CD

Serology

Evidence Table 1: Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Altuntas, 1998 Turkey	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** • endoscopic biopsy **First test:** • serology test **Controls biopsied:** • yes **Biopsy criteria:** • subtotal or total VA, crypt hyperplasia, increased IEL **Checked IgA def.** • n/r **Studied tests** • IgA-AGA • IgG-AGA **Methodology:** • ELISA **Cut-off:** • levels between 25 and 50 RU/mL were accepted as weakly positive and levels >50 RU/mL as strongly positive	**Celiac Group 1** • 26 short-statured children with probable CD on biopsy • mean age: n/r • % F: n/r	**Group 1:** • 21 short-statured children without CD on biopsy; • median age: n/r • % F: n/r	**celiac 1 vs control 1** IgA AGA	6	20	2	19	23	90	75	48
				celiac 1 vs control 1 IgG AGA	26	0	21	0	100	0	55	0

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Artan, 1998, Turkey	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** • biopsy **First test:** • simultaneous **Controls biopsied:** • yes **Biopsy criteria:** • ESPGAN **Checked IgA def.** • n/r **Studied tests** IgA-AGA; IgG-AGA; IgA and IgG; IgA or IgG **Methodology:** • ELISA **Cut-off:** • >25 arbitrary units (did not adjust for age)	**Celiac Group 1** • 24 children with CD by ESPGAN, out of 63 suspected CD pts • age: n/r • % F: n/r	**Group 1:** • 39 of 63 with normal intestinal villous structure. • Age: n/r • % F: n/r	celiac 1 vs control 1-IgA-AGA celiac 1 vs control 1-IgG-AGA IgA AND IgG IgA OR IgG	14 20 12 20	10 4 12 4	19 16; 13; 25;	20; 23 26 14	58 83 50 83	51 59 67 36	42.4 55.6 48 44	66.7 85.2 68.4 77.8

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Ascher, 1996 Sweden	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** • n/r **First test:** • biopsy and serum tests obtained simultaneously **Controls biopsied:** • yes **Biopsy criteria:** • ESPGAN **Checked IgA def.** • Yes **Studied tests** • EIA IgA; EIA IgG; DIG-ELISA IgA; DIG-ELISA IgG; ARA; Human JAB; Rat JAB; EMA **Methodology:** for EIA IgA and IgG: ELISA; for ARA: IF; for EMA, JAB: immunohistochemical method **Cut-off:** EIA IgA and IgG: 35 AU (arbitrary units) for children <5 y; and 20 AU for children >5 y; DIG-ELISA: IgA values >13 mm and IgG values >16 mm were considered positive in children <5 y; IgA values <11 mm and IgG values >14 mm - in children >5 y	**Celiac Group 1** • 55 pts with biopsy proven CD • mean age: n/r; <5 y n=22; >5 y n=33 • % female: n/r **Celiac Group 2** • 36 out of initial 55 pts who were treated with GFD of 1 y; • mean age: n/r; <5 y n=21; >5 y n=15 • % female: n/r **Celiac Group 3** • 21 pts on a gluten-challenge diet of 3-6 mos • mean age: n/r; <5 y n=18; >5 y n=3; • % female: n/r NB: group 2: biopsy proven CD on a GFD of > 1 y; group 3: biopsy proven CD on a gluten challenge of 3-6 mos duration	**Group 1:** • 65 disease controls with biopsy proven normal intestinal mucosa; • mean age: n/r; <5 y n=18; >5 y n=47 • % female: n/r	group 1 vs control, EIA IgA	50	5	1	64	90.9	98.5		
				group 1 vs control, EIA IgG	53	2	20	45	96.4	69.2		
				group 1 vs control, DIG-ELISA IgA	50	5	2	63	90.9	96.9		
				group 1 vs control, DIS-ELISA IgA	48	7	9	56	87.3	86.2		
				group 1 vs control ARA	48	6	18	47	88.9	72.3		
				group 1 vs control, Human JAB	55	0	18	47	100	72.3		
				group 1 vs control, RAT JAB	55	0	11	54	100	83.1		
				group 1 vs control, EMA	54	1	0	65	98.2	100		
				group 3 vs control, EIA IgA	20	1	1	64	95.5	98.5		
				group 3 vs control, EIA IgG	19	2	20	45	90.5	69.2		
				group 3 vs control, DIG-ELISA IgA	17	4	2	63	80.9	96.9		
				group 3 vs control, DIS-ELISA IgA	15	5	9	56	75	86.1		
				group 3 vs control, ARA	18	3	18	47	85.7	72.3		
				group 3 vs control, Human JAB	21	0	18	47	100	72.3		
				group 3 vs control, RAT JAB	18	3	11	54	85.7	83		
				group 3 vs control, EMA	19	2	0	65	90.5	100		

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Ascher, 1990 Sweden	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** • Watson capsule **First test:** • biopsy and serum tests obtained simultaneously **Controls biopsied:** • yes **Biopsy criteria:** ESPGAN **Checked IgA def.** Yes **Studied tests** • Pharmacia Gluten IgA-AGA measured by enzyme immunoassay (PG IgA-EIA) **Methodology:** • ELISA **Cut-off:** • 35 AU (arbitrary units)	**Celiac Group 1** • 36 pts with CD out of 130 consecutive group of children who had a small intestinal biopsy due to symptoms suggestive of CD; out of 36 pts with CD, 28 have been verified according to ESPGAN criteria; • mean age: n/r • % female: n/r **Celiac Group 2** • children with CD according to ESPGAN's criteria, differing with regard to gluten content of diet: first biopsy on a gluten-containing diet (n=29); at the second biopsy after 1 y on GFD (n=45); at the third biopsy after gluten-challenge (n=45); • mean age: n/r • % F: n/r **Celiac Group 3** • children with an initial abnormal mucosa that normalized on a GFD but did not relapse after gluten challenge during 3-31 mos • mean age: n/r • % F: n/r	**Group 1:** • 92 children taken from consecutive group of 130 children who did not have CD; 12 had other food intolerance, 1 had cystic fibrosis and 79 had no intestinal disorder	group 1 vs control PG IgA-EIA	35	1	7	85	97	92	83.3	98.8

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results									
					4X4 table								
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Bahia, 2001 Brazil	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/a **Population Type:** • suspected CD **Reference test:** • Carey capsule **First test:** • n/r **Controls biopsied:** • yes **Biopsy criteria:** • severe VA **Checked IgA def.** • n/r **Studied tests** • IgA and IgG-AGA **Methodology:** • ELISA **Cut-off:** • mean+2 SD for a group of 20 normal children: 0.022 for IgA (mean=0.0065, SD=0.0076); and 0.103 for IgG (mean=0.0393, SD=0.032)	**Celiac Group 1** • 22 pts with CD • age (mos): mean/SD: 30.6+/-28.8; • % F: 54.5 • median w/range: 19.3 (6.0-135.6) • % female: 54.5 **Celiac Group 2** • 61 pts with other enteropathies (OE); • age (mos): mean/SD: 43.3+-38.1; • median w/range: 27.9 (4.8-156.5); • % F: 59	**Group 1:** • 46 controls with biopsy-proven non-CD enteropathies (CO) • age (mos): mean/SD: 96.9+/-48.5; median w/range: 92.6 (12.2-170.8); • % F: 54.5	IgA group 1 vs CO IgA group 1 vs OE+CO IgG group 1 vs CO IgG group 1 vs OE+CO	21 21 20 20	1 1 2 2	2 9 1 12	46 98 45 95	95.5 95.5 90.9 90.9	95.6 91.6 97.8 88.7	when prevalence is 1:500 PPV for IgA is 4.8%; in prevalence of 1:1000 PPV=2.0; in prevalence of 1:2000, PPV=1.1	in any prevalence of CD NPV for IgA is 99.9%

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results									
					4X4 table								
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Bardela, 2001 Italy	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/a **Population Type:** • suspected CD **Reference test:** • endoscopic biospy **First test:** • n/r **Controls biopsied:** • yes **Biopsy criteria:** • Marsh, no grade reported **Checked IgA def.** • Yes and excluded **Studied tests** • IgA-AGA; IgA-EMA; IgA-tTGA **Methodology:** • ELISA for AGA; ELISA for tTGA GP liver; IF for EMA ME **Cut-off:** • AGA: 12 AU/mL; tTGA: >10 AU/mL; EMA: antibody titre > 1:10	**Celiac Group 1** • 40 untreated biopsy-proven CD pts • age (y): mean 38, range 16-77 • % F: 72.5 **Celiac Group 2** • 195 treated CD pts; biopsy proven on GFD • age (y): mean 38, range 16-79 • % F: 70.2	**Group 1:** • 110 biopsy proven non-CD disease controls (CO): inflammatory bowel disease (n=22); IBS (n=29); peptic ulcer (n=7); diverticular disease (n=6); pancreatitis (n=5); non- ulcer dyspepsia(n=14); anemia not due to malabsorption (n=7); reflux esophagitis (n=3); atrophic gastritis (n=2); acute appendicitis (n=1) • age (y): mean 41, range 14-80 • % F: 75.7;	AGA group 1 vs CO EMA group 1 vs CO tTGA group 1 vs CO	38 40 40	2 0 0	12 3 2	98 107 108	95 100 100	89 97.2 98.2	when expected prevalence is 0.5%, PPV for AGA is 4.2%; for EMA - 15.7% and for tTGA - 21.8%; when expected prevalence of CD is 50%, PPV for AGA is 89.7%, for EMA - 97.4% and for tTGA - 98.2%	when expected prevalence is 0.5%, NPV for AGA is 99.9%; for EMA - 100% and for tTGA - 100%; when expected prevalence of CD is 50%, NPV for AGA is 94.7%, for EMA - 100% and for tTGA - 100%

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Berger, 1996 Switzerland	**Publication type:** • journal **Study design:** • case-control **Ethnicity:** • n/a **Population Type:** • suspected CD **Reference test:** • endoscopic biopsy **First test:** • n/r **Controls biopsied:** • yes **Biopsy criteria:** •ESPGAN revised with complete VA **Checked IgA def.** • n/r **Studied tests** •5 different AGA assays: Eurospital; Labodia; Pharmacia; FIST-IF; Granditsch; IgA EMA **Methodology:** • for AGA - ELISA; for EMA - IF **Cut-off:** For IgG Eurospital: index30-50; in Pharmacia units: 30-50 AU; Labodia: 30 U/mL; in Pharmacia units-18; Pharmacia: 20-100 AU; FIST: titer 1:20; in Pharmacia units-13 AU; Granditsch: 0.350 OD; in Pharmacia units: 70 AU; for IgA Eurospital: index 8-20; in Pharmacia units: 53-132 AU; Labodia: 15 U/mL; in Pharmacia units-28; Pharmacia: 20-35 AU; FIST: titer 1:20; in Pharmacia units-34 AU; Granditsch: 0.250 OD; in Pharmacia units: 38 AU	**Celiac Group 1** • 67 biopsy proven CD pts • age: n/r • % F: n/r	**Group 1 (CO):** •54 biopsy proven non-CD pts: transient GI problems (n=39), ulcerative colitis (n=2), Crohn's disease (n=8), duodenal ulcer (n=3), short stature (n=2) •age: n/r •% F: n/r	**CD vs CO** Eurospital IgG FIST IgG Granditsch IgG Labodia IgG Pharmacia IgG Eurospital IgA FIST IgA Granditsch IgA Labodia IgA Pharmacia IgA;	 55 56 55 61 46 41 44 53 58 51	 12 11 12 6 21 26 23 14 9 16	 27 53 46 42 22 3 49 23 35 18	 27 1 8 12 32 51 5 31 19 36	 82 84 82 91 69 61 66 79 87 76	 50 2 15 22 59 94 9 57 35 67	 67 51 54 59 68 93 47 70 62 74	 69 8 36 57 53 57 17 59 58 59

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Biagi, 2001 Italy	**Publication type:** • journal **Study design:** • case-control **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** • Carey capsule **First test:** • n/r **Controls biopsied:** • yes **Biopsy criteria:** • partial VA or greater **Checked IgA def.** • Only in EMA (neg) pts in CD group **Studied tests** • IgA-tTG • IgA-EMA **Methodology:** • TTG-GP; EMA -ME **Cut-off:** • on the basis of ROC analysis performed on the preliminary results (group of 30 controls) results >0.65 OD (optical density) were considered positive; <0.35 OD - negative and 0.35-0.65 OD - borderline	**Celiac Group 1** • 56 pts with biopsy-proven CD • age (y): mean/SD: 39.2+-19.2; range 4-79 • % F: 76	**Group 1:** • 52 disease controls (biopsy-proven non-SD enteropathies) with irritable bowel disease (n=29); Crohn's disease (n=9); gastric lymphoma (n=7); Whipple disease (n=3); giardiasis (n=2); systemic mastocytosis (n=1); IgE-mediated food sensitivity • mean age (y): 40.4+-19.9; range 14-79; • % F:	IgA TTG group 1 vs CO when considering borderline results as positive	55	1	8	44	98.2	84.6	87.3	97.7
				IgA-tTG group 1 vs CO when considering borderline results as negative	49	7	1	51	87.5	98.1	98	87.9
				IgA EMA group 1 vs CO	53	3	0	52	94.6	100	100	94.5

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Bode, 1993 Denmark	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** • n/r **First test:** • biopsy **Controls biopsied:** • yes **Biopsy criteria:** •typical biopsy and response to a GFD (likely ESPGAN) **Checked IgA def.** • yes **Studied tests** • serum IgG-AGA • serum IgA-AGA **Methodology:** •DIG-ELISA - diffusion in gel ELISA **Cut-off:** • 1) old limits: positive test for IgA AGA was defined as >10.5 mm and/or IgG level >14 mm; 2) new limits for children: for IgA >10 mm and for IgG >13 mm	**Celiac Group 1** • 14 of 233 consecutive children with untreated CD • median age for the 233: 2.75; range 0.33-15.5) • 117 males; 74 females **Celiac Group 2a** •T-CD - group 2a) 47 children with CD on GFD •age (y): median 11.83, range 1-17.92 •% F: 61.7 **Celiac Group 2b** • 14 children with known CD on gluten challenge	**Group 1:** • 177 children with non-CD diseases: postenteritis diarrhea (n=43), short stature (n=25), diarrhea (n=25), failure to thrive (n=22), food allergy (n=11), dissaccharide intolerance (n=8), dietary problems (n=8), giardiasis (n=4), ulcerative colitis (n=3), recurrent abdominal pain (3), constipation (n=2), recurrent infections (n=2), acute gastroenteritis (n=1), Crohn's disease (n=1), other GI diseases (n=4), other non-GI diseases (n=15) **Group 2:** • 8 children not CD/GFD and gluten challenge • age (y): median 2.5, range 1.17-7.5 • % F: 37.5	group 1 vs control 1, serum AGA-IgA; in brackets data according to new cut off group 1 vs control 1, serum AGA-IgG; in brackets data according to new cut off limits group 1 vs control 1, serum AGA-IgA/IgG; in brackets data according to new cut-off limits					64 (79) 71 (93) 86 (100)	99 (98) 99 (98) 99 (97)	90 (79) 100 (76) 92 (70)	97 (98) 98 (99) 99 (100)

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Bode, 1994 Denmark	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** • n/r **First test:** • n/r **Controls biopsied:** • yes **Biopsy criteria:** • Crypt hyperplasia, VA and increase inflammatory cells **Checked IgA def.** • n/r **Studied tests** • serum IgG-AGA • serum IgA-AGA **Methodology:** •DIG-ELISA - diffusion in gel ELISA **Cut-off:** • positive test for IgA AGA was >10.5 mm; for IgG - >14 mm; borderline levels for IgA was between 9.5 mm and 10.5 mm; and for IgG - between 13 mm and 14 mm	**Celiac Group 1** • 100 consecutive adult pts admitted for a small intestinal biopsy on suspicion of CD • age (y): median 51, range 17-81 • % F: 64; 13 CD pts **Celiac Group 2** • 118 adult pts with increased or borderline (gliadin antibodies) AGA • age (y): median 48, range 19-95 • % F: 72; • 55 children pts with increased or borderline (gliadin antibodies) AGA • age (y): median 4, range 7 mos-17 • % F: 56	**Group 1:** • 87 out of 100 suspected of CD who did not have CD by biopsy	group 1 IgA-AGA					46	98	75	92
				group 1 IgG-AGA					62	97	73	94
				group 1 IgA/IgG-AGA					77	95	71	97

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Bonamico, 2001 Italy	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** • biopsy **First test:** • biopsy **Controls biopsied:** • yes **Biopsy criteria:** •ESPGAN (severe VA and crypt hyperplasia) **Checked IgA def.** • Yes **Studied tests** • tTG-HR (RIA) • tTG-GP (ELISA) • IF-EMA **Methodology:** • tTG-HR • tTG-GP • EMA-ME IF **Cut-off:** • RIA anti-tTG Ab ≥0.05 index as selected on ROC plot analysis; ELISA anti-tTG Ab ≥7 AU; IF-EMA - n/r (appearance of fluorescence)	**Celiac Group 1** • 62 pts; untreated with biopsy-proven celiac CD • median age 7, age range 1-23 • % F: n/r **Celiac Group 2** • 78 pts; GFD treated, at least 6 mos • median age, age range • % F: n/r **Celiac Group 3** • 14 pts; on gluten-challenge at least 3 mos • median age, age range • % F: n/r	**Group 1:** • 56 disease controls; chronic diarrhea, short stature, recurrent abdominal pain; age and sex-matched controls • median age, age range • % F: n/r	RIA group 1 vs control	62	0	0	56	100	100	100	100
				RIA group 2 vs control	34	44	0	56	43.5	100	100	56
				RIA group 3 vs control	14	0	0	14	100	100	100	100
				ELISA group 1 vs control	56	6	0	56	90.3	100	100	90.3
				ELISA group 2 vs control	7	71	0	56	9.8	100	100	44
				ELISA group 3 vs control	11	3	0	56	78.5	100	100	94.9
				IF-EMA group 1 vs control	59	3	1	55	95.1	98.2	98.3	94.8
				IF-EMA group 2 vs control	9	69	1	55	11.5	98.2	90	44.3
				IF-EMA group 3 vs control	13	1	1	55	92.8	98.2	92.8	98.2

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Bottaro, 1997 Italy	**Publication type:** • journal **Study design:** • case-control **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** • n/r **First test:** • n/r **Controls biopsied:** • yes **Biopsy criteria:** •ESPGAN **Checked IgA def.** • Yes **Studied tests** serum AGA-IgG serum AGA-IgA serum EMA-IgA serum ARA-IgA **Methodology:** •for AGA ELISA; for AGA-IgA IF; for EMA-IgA IF using ME or HU **Cut-off:** • AGA: for IgA 10% and for IgG 25%, resulting the mean+-SD of values obtained from children proven normal	**Celiac Group 1** • 50 children with biopsy proven CD • age: median 2.5 y, range 7 mos - 15 y • % F: 68	**Group 1:** • 25 control group of children (CO) • age: median 3.0 y, range 9 mos - 14 y • % F: 52	group 1 vs control 1, serum AGA IgA	46	4	8	17	92	68	85.2	80.9;
				group 1 vs control 1, serum AGA IgG	50	0	16	9	100	36	75.7	100
				group 1 vs control 1, serum EMA-IgA, HUC	47	3	0	25	94	100	100	89.2
				group 1 vs control 1, serum EMA-IgA, ME	48	2	1	24	96	96	97.9	92.3
				group 1 vs control 1, serum ARA IgA								

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Carroccio 2002 Italy	**Publication type:** • journal **Study design:** • case-control **Ethnicity:** • n/a **Population Type:** • suspected CD **Reference test:** children: Crosby capsule; adults: endoscopic biopsy **First test:** • n/r **Controls biopsied:** • yes **Biopsy criteria:** Marsh; CD was diagnosed as enlarged crypts and/or VA - with normalization on GFD **Checked IgA def.** • Yes **Studied tests** Serum AGA-IgG; Serum AGA-IgA; Serum EMA; Culture EMA; Culture EMA+gliadin **Methodology:** ELISA for AGA; indirect IF on ME for serum EMA; biopsy specimen incubation in a culture medium with gliadin peptide and further IF on ME **Cut-off:** AGA-results expressed as a % of reference serum: 20% was upper normal limit for IgG and 10% for IgA antibodies; EMA-semi-quantified as follows: 0=not detectable; 1=positive at dilutions between 1/5 and 1/20; 2=positive between 1/40 and 1/80; 3=positive at 1/100; 4=positive at 1/200; 5=positive at >1/200; EMA in culture-same as serum EMA and for IgG 25%, resulting the mean+-SD of values obtained from children proven normal	**Celiac Group 1** • 91 pts with biopsy proven CD • age (y): median 2; range 7 mos - 84 y • % F: 56 **Celiac Group 2** • 21 treated CD pts on a GFD with normal intestinal architecture • age (y): median 5, range 3-51 • % F: 62	**Group 1:** • 100 subjects with a normal intestinal morphology or diseases other than CD (biopsy proven) • age (y): median 21 y, range 9 mos-76 y • % F: 56 **Group 2:** • 22 disease controls (biopsy-proven non-CD) with GERD-like symptoms • age (y): median 33, range 4-60 • % F: 54.5	group 1 vs control 1, serum AGA IgG	69	22	25	75	76	75	73.4	77.3
				group 1 vs control 1, serum AGA IgA	61	30	10	90	67	90	86	75
				group 1 vs control 1, serum EMA	80	11	1	99	88	99	98.7	90
				group 1 vs control 1, culture EMA	82	9	0	100	90	100	100	91.7
				group 1 vs control 1, culture EMA+gliadin	87	4	0	100	96	100	100	96
				group 2 vs control 2, culture EMA+gliadin	10	11	0	22	47.6	100	100	66.7

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Carroccio 1993 Italy	**Publication type:** • journal **Study design:** • case-control **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** • Biopsy Watson capsule **First test:** • biopsy **Controls biopsied:** • yes **Biopsy criteria:** •Biopsies confirmed at diagnosis, on GFD, and rechallenge (severity grade-not reported) **Checked IgA def.** • n/r **Studied tests** IgA-AGA IgG-AGA IgA-EMA **Methodology:** •ELISA; IF likely ME **Cut-off:** • AGA mean +2 SD	**Celiac Group 1** • infants with CD on gluten diet; biopsy proven • median age 2.6; range 0.8-10 • 21 males; 22 females	**Group 1:** • 60 infants disease controls; biopsy proven non-CD • median age 1.2; range 0.9-9 • 32 males; 28 females	celiac 1 vs control	31 40 45	14 5 0	5 32 2	55 28 58	68 88.9 100	91.7 46.7 96.7	86.1 55.6 95.7	79.7 84.8 100

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Carroccio 2002 Italy	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** gastroduodenoscopy and biopsy **First test:** • simultaneously **Controls biopsied:** • yes **Biopsy criteria:** •Ferguson and Murray; partial or total VA **Checked IgA def.** yes (none of CD pts showed IgA deficiency **Studied tests** EMA-IgA; anti-GP-tTG IgA (GP used); anti-h-tTG IgA (HR) **Methodology:** for EMA - IF ME; for tTG–ELISA **Cut-off:** anti-h-tTG IgA-results were expressed as a % of the positive control serum. Normal values were taken as <7%, which represented a value >2 SD above the mean of 850 healthy individuals; anti-gp-tTG IgA-values >95th percentile of a control group were considered positive;	**Celiac Group 1** • 24 consecutive pts with untreated biopsy proven CD • age (y): median 30, range 18-80 • % F: 58	**Group 1:** • 183 consecutive pts with biopsy proven non-CD disorders: IBS (n=70), esophagatis (n=45), peptic ulcers (n=41), Crohn's disease (n=15), Food intolerance (10), chronic liver disease (n=6), gastric cancer (n=2), right colon cancer (n=2), collagenous colitis (n=1), intestinal bacterial overgrowth syndrome (n=1), psoriasis (n=1) • age (y): median 46, range 17-84 • % F: 51	CD vs CO: EMA	24	0	0	183	100	100	100	100
				CD vs CO: anti-GP-tTG IgA	24	0	15	168	100	92	60	100
				CD vs CO: anti-h-tTG IgA	24	0	6	177	100	97	80	100

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Cataldo, 2000 Italy	**Publication type:** • journal **Study design:** • case-control **Ethnicity:** • n/a **Population Type:** • IgA deficient adults and children **Reference test:** • n/r **First test:** • biopsy **Controls biopsied:** • yes **Biopsy criteria:** Original & revised criteria? **Checked IgA def.** • Yes **Studied tests** IgG-EMA IgA-EMA IgG-tTG IgA-tTG IgG-AGA IgA-AGA **Methodology:** •IgG-EMA, ME; IgG-tTG, human serum albumin **Cut-off:** • n/a	**Celiac Group 1** • 20 untreated CD (biopsy proven) with IgAD • age/gender: n/a **Celiac Group 2** • 11 untreated CD without IgAD • age/gender: n/a	**Group 1:** • 10 healthy IgAD controls on GD (healthy controls (not biopsied - not used) • age: n/a • gender: adults and children **Group 2** •25 healthy controls on a GD, first degree relatives of CD pts, adult or paediatric pts with GI diseases i.e. mild protein intolerance, pstenteritis syndrome, Crohn's disease, ulcerative colitis, or giardiasis •age/gender: n/a	Celiac 1 vs control 1	20 0 20 0 20 0	0 20 0 20 0 20	0 0 2 0 0 0	10 10 8 10 10 10	100 0 100 0 100 0	100 100 80 100 100 100	100 0 90.1 0 100 0	100 33.3 100 33.3 100 33.3

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Chan, 2001 Canada	**Publication type:** • journal **Study design:** • case-control **Ethnicity:** • n/a **Population Type:** • suspected CD **Reference test:** • Carey capsule or 4 to 6 duodenal biopsies at endoscopy **First test:** • biopsy **Controls biopsied:** • yes **Biopsy criteria:** Villous atrophy, crypt hyperplasia, increase lymphocytes **Checked IgA def.** • Yes **Studied tests** IgA-EMA; IgA-tTG **Methodology:** •EMA-ME; IgA tTG ELISA GP liver **Cut-off:** • EMA:≥1:10; tTG: >20	**Celiac Group 1** • 9 untreated CD (biopsy + from a group of 77 children • age: 2 mos -16 y **Celiac Group 2** • 12 suspected CD with DM from a group of 16 DMs • age: 3-18Y **Celiac Group 3** • 2 IgAD pts, one biopsy positive, (excluded in the analysis)	**Group 1:** • 62 disease controls with negative biopsy with abdominal pain, diarrhea, failure to thrive/short stature, family history of CD, Crohn's disease, vomiting, abdominal distension, ulcerative colitis, autoimmune thyroiditis/short stature, trisomy, (from a total of 77) • age 2 mos to 16 y **Group 2** •2 DM from a group of 16 DMs	celiac 1 vs control 1	8 8	1 1	2 4	64 62	89 89	97 94	80 67	98 98

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Chartrand 1997 Canada	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/a **Population Type:** • suspected CD **Reference test:** • Biopsy - endoscopic **First test:** • biopsy **Controls biopsied:** • yes **Biopsy criteria:** ESPGAN - with flat mucosal biopsy **Checked IgA def.** • yes - 2 of false negatives were IgA def **Studied tests** IgA–AGA IgG-AGA IgG or IgA **Methodology:** •ELISA **Cut-off:** • 0.25 for IgA, 0.3 for IgG (optical density)	**Celiac Group 1** • 30 of 176 children suspected of CD • mean age: 5.2; range 0.5-18; • % F: n/r	**Group 1:** •146 with suspected CD - biopsy excluded •mean age: n/r •% F: n/r	Celiac 1 vs Control	24 25 28	6 5 2	12 31 42	134 115 104	80 83 93	92 79 71	67 45 43	96 96 98

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Chirdo 1999 Argentina	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/a **Population Type:** • suspected CD **Reference test:** • n/r **First test:** • biopsy **Controls biopsied:** • yes **Biopsy criteria:** Total or subtotal VA **Checked IgA def.** • n/a **Studied tests** IgG commercial gliadin; IgG ethanolic extract; IgG ω-gliadins ; IgA commercial gliadin; IgA ethanolic extract; IgA IgG ω-Gliadins **Methodology:** •ELISA **Cut-off:** • n/r	**Celiac Group 1** • 28 pts, biopsy proven untreated • mean age: 7 y; range 13 mos-14 y • % F: 15 (54%)	**Group 1:** •31 disease controls; chronic diarrhea, short stature, abdominal distension •mean age: 5.4 y, 2-12 y •13 F, 18 M	Celiac 1 vs Control	24 23 25 21 18 24	4 5 3 7 10 4	6 8 5 4 3 1	25 23 26 27 28 30	85.7 82.1 89.3 75 64.3 85.7	80.6 74.2 83.9 87.1 90.3 96.8	80 74 83 84 86 96	86 82 90 80 74 88

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Chirdo 2000 Argentina	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** Intestinal biopsy **First test:** • biopsy **Controls biopsied:** • only group 3 **Biopsy criteria:** ESPGAN **Checked IgA def.** • Yes **Studied tests** IgA w-AGA; IgG w-AGA; not a commercial kit (not used) IgA-EMA (used) **Methodology:** •IgA-EMA-ME IF **Cut-off:** • cut-off value for each antigen by using the same set of samples. Five control samples used as reference to normalize the day-to-day variation	**Celiac Group 1** • 105 untreated CD pts (biopsy proven) • mean age: 6.5 y; range 16 mos-15 y • % F: n/r	**Group 1:** • 45 healthy controls • mean age 8.5 y; range 3-14 y **Group 2:** • 36 healthy blood donors • mean age 36, range 22-45 y **Group 3:** • 46 biopsy negative disease control; presenting with short stature, chronic diarrhoea, parasitic infection • mean age 5.2 y; range 1.5-14 y this group used for analysis **Group 4:** • 27 disease controls with Crohn's disease, ulcerative colitis, or Helicobacter pylori infection	Celiac 1 vs control 3	94 100 97	11 5 8	5 9 0	41 37 46	89.5 95.2 92.4	89.1 80.4 100	94.9 91.7 100	78.8 88.1 85.2

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Dahele 2001 Scotland	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/a **Population Type:** • suspected CD **Reference test:** duodenal biopsy **First test:** • biopsy **Controls biopsied:** • yes **Biopsy criteria:** Included partial VA or greater **Checked IgA def.** • Yes **Studied tests** IgA AGA; IgG AGA; IgA EMA; IgA tTG **Methodology:** • IgA AGA, ELISA; IgG-AGA, ELISA; IgA-EMA, HU; IgA-tTG ELISA GP liver **Cut-off:** • for IgA & IgG AGA: ≥30 unit/mL & ≥45 units/mL, respectively	**Celiac Group 1** • 53 untreated CD pts (biopsy proven) • median age 51 y; range 22-77 y • 39 F; 14 M	**Group 1:** • 65 control pts • median age: 45 y; range 17-90 y • 46 F; 19 M	Celiac 1 vs Control 1	34 44 40 35;	19 8 13 18	10 14 0 3	55 51 65 62	64 83 75 66	85 78 100 92	77 76 100 92	74 86 83 78

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Dahele 2001 Scotland NB: update of Dahele et al., Dig Dis Sci; 2001;46: 214	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/a **Population Type:** • suspected CD **Reference test:** duodenal biopsy **First test:** • biopsy **Controls biopsied:** • yes **Biopsy criteria:** Included 6 with IEL, rest partial VA or greater **Checked IgA def.** • yes - 2 IgA deficient excluded (114/116 CD used) **Studied tests** IgA AGA; IgA AEM; IgA tTG **Methodology:** • IgA-AGA, ELISA; IgG-AGA, ELISA; IgA-EMA, HU; IgA tTG ELISA GP liver **Cut-off:** • for IgA & IgG AGA: ≥30 unit/mL & ≥45 units/mL, respectively	**Celiac Group 1** • 116 untreated CD pts (biopsy proven) • median age: 47 y; range 15-78 y • 74 F; 42 M	**Group 1:** • 65 control pts (suspected CD pts with normal biopsy) • median age 45 y; range 16-90 y • 45 F; 20 M	Celiac 1 vs Control 1	69 99 92	45 15 22	9 0 2	56 65 63	61 87 81	86 100 97	88.5 100 97.9	42.7 81.3 74.1

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Di Leo 1999 Italy	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** biopsy **First test:** • biopsy **Controls biopsied:** • yes **Biopsy criteria:** ESPGAN **Checked IgA def.** • yes **Studied tests** EIA-IgA; EIA-IgG; DIG-ELISA IgA; DIG-ELISA IgG; ARA; human JAB-IgA; Rat JAB; EMA-IgA **Methodology:** •for EIA-IgA and IgG-ELISA; for ARA-IF; for EMA, JAB-immunohistochemical method •**Cut-off:** EIA IgA and IgG: 35 AU (arbitrary units) for children <5 y; and 20 AU for children >5 y; DIG-ELISA: IgA values >13 mm and IgG values >16 mm were considered positive in children <5 y; IgA values <11 mm and IgG values >14 mm - in children >5 y	**Celiac Group 1** • 45 untreated pts (biopsy proven untreated) • mean age: 6.11 y; range 9 mos-17 y • 32 F, 13 M **Celiac Group 2** • 18 GDF pts (celiac on GFD) • mean age: 13.2 y; range 3 y 8 mos-16 y 6 mos • 13 F, 5 M	**Group 1:** • 67 healthy controls • mean age: 8 y. 9 mos; range 1 y. 4 mos-16 y. 9 mos • 43 F, 24 M	group 1 vs control, EIA IgA	50	5	1	64	90.9	98.5		
				group 1 vs control, EIA IgG	53	2	20	45	96.4	69.2		
				group 1 vs control, DIG-ELISA IgA	50	5	2	63	90.9	96.9		
				group 1 vs control, DIS-ELISA IgA	48	7	9	56	87.3	86.2		
				group 1 vs control, ARA	48	6	18	47	88.9	72.3		
				group 1 vs control, Human JAB	55	0	18	47	100	72.3		
				group 1 vs control, RAT JAB	55	0	11	54	100	83.1		
				group 1 vs control, EMA	54	1	0	65	98.2	100		
				group 3 vs control, EIA IgA	20	1	1	64	95.5	98.5		
				group 3 vs control, EIA IgG	19	2	20	45	90.5	69.2		
				group 3 vs control, DIG-ELISA IgA	17	4	2	63	80.9	96.9		
				group 3 vs control, DIS-ELISA IgA	15	5	9	56	75	86.1		
				group 3 vs control, ARA	18	3	18	47	85.7	72.3		
				group 3 vs control, Human JAB	21	0	18	47	100	72.3		
				group 3 vs control, RAT JAB	18	3	11	54	85.7	83		
				group 3 vs control, EMA	19	2	0	65	90.5	100		

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Dickey 2001 Northern Ireland	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** • n/a **First test:** • biopsy **Controls biopsied:** • yes **Biopsy criteria:** VA **Checked IgA def.** • no **Studied tests** EMA (excluded); tTGA; EMA and/or tTGA **Methodology:** •EMA=ME; tTGa, ELISA GP liver **Cut-off:** >30 as positive	**Celiac Group 1** • 73 untreated biopsy-proven CD pts • age 13-72 y • 45 F, 28 M	**Group 1:** 58 disease controls; diarrhea, anaemia, family history of celiac and others	Celiac 1 vs Control 1	55 68	18 5	1 2	57 56	75.3 93.2	98.3 96.6	98.2 97.1	76.0 91.8

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Faith-Magnusson 1994 Sweden	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/a **Population Type:** • suspected CD **Reference test:** • Watson capsule or Stortz **First test:** • biopsy **Controls biopsied:** • yes **Biopsy criteria:** ESPGAN+ Alexander grading IV, grade III to IV callenge **Checked IgA def.** • no **Studied tests** DIG - ELISA combined IgA & IgG; ELISA combined IgA & IgG **Methodology:** •ELISA; DIG-ELISA **Cut-off:** DIG ELISA (mm) combined IgA+IgG ≥ 6 and /or; ≥10; ≥12; ≥14; ≥16; ELISA (mm) for combined IgA+IgG ≥0.25; and; ≥0.8; ≥0.9; ≥1.0; ≥1.1	**Celiac Group 1** • 116 pts; biopsied twice; median time on GFD pre 2nd biopsy 13 mos (10-24 mos) • age @ 1st biopsy median 13 mos, range 0.7-16.7 y, age @ 2nd biopsy 29 mos range 1.6-17.8 y • 73 F; 43 M	**Group 1:** • 199 disease controls; poor weight gain; diarrhea; stature • median age 22 mos range 0.7-16.8 y • 105 F; 94 M	Celiac 1 vs Control 1	n/r	n/r	n/r	n/r	98.1 95.2 91.4 88.6 93.3 88.7 88.7 88.7	27.8 39.5 70.5 92.3 64.6 75.8 82.3 93.5		

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Gilbert 2000 Canada	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/a **Population Type:** • n/r **Reference test:** • biopsy **First test:** • biopsy **Controls biopsied:** • yes **Biopsy criteria:** mild, moderate, severe VA **Checked IgA def.** • n/r **Studied tests** IgA-EMA; IgA-tTG **Methodology:** •EMA-HU •tTG- HR **Cut-off:** EMA:≥1:5; tTG: >400u	**Celiac Group 1** • 21 CD adults • age: n/r • % F: n/r	**Group 1:** • biopsied disease controls with CD excluded • age: n/r • % F: n/r	Celiac 1 vs Control	21 20	0 1	0 0	42 42	100 95.2	100 100		

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Gonczi 1991 Australia	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/r **Population Type:** • suspected celiac; single institution 1977-1983; mean age 3.97 (range 1 mos-16 y) **Reference test:** • biopsy technique not reported (likely capsule) **First test:** • biopsy **Controls biopsied:** • yes **Biopsy criteria:** ESPGAN no details on biopsy findings **Checked IgA def.** • yes - 1 CD child **Studied tests** IgA AGA; IgG-AGA **Methodology:** •ELISA mean + 2 SD; IgA 25 AU; IgG 46 **Cut-off:** 20.2%	**Celiac Group 1** • 20 children untreated biopsy-proven CD • age: n/r • % F: n/r **Celiac Group 2** • 25 untreated adults biopsy-proven CD • age: n/r • % F: n/r	**Group 1:** • 79 children biopsy-negative controls w sx • age: n/r • % F: n/r **Group 2:** • 34 adult disease controls • age: n/r • % F: n/r	celiac 1 vs control 1-IgA-AGA	19	1	6	73	95	92.4	76	98.6
				celiac 1 vs control 1-IgG – AGA	20	0	6	73	100	92.4	76.9	100
				celiac 1 vs control 1-IgA+IgG	20	0	1	78	100	98.7	95.2	98.7
				celiac 2 vs control 2-IgA-AGA	23	2	4	30	92	88.2	85.2	93.8
				celiac 2 vs control 2-IgG – AGA	25	0	11	23	100	69.7	69.4	100
				celiac 2 vs control 2-IgA+IgG	25	0	1	33	100;	97.1	96.2	100

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Hallstrom 1989 Findland	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/r **Population Type:** • n/a **Reference test:** • biopsy **First test:** • biopsy **Controls biopsied:** • yes **Biopsy criteria:** Flat mucosa **Checked IgA def.** • yes **Studied tests** IgA - EMA **Methodology:** •EMA-ME **Cut-off:** n/r	**Celiac Group 1** • 32 untreated adult CD pts • mean age 36 y; range 18-63 y) • % F: n/r **Celiac Group 2** • 18 children with untreated CD • mean age 1; range 2-16 • % F: n/r	**Group 1:** • 24 non-CD children by biopsy, with various abdominal symptoms • mean age 7.5 y; range 1-15 • % F: n/r	celiac 1 vs control 1	29	3	0	24	90.6	100	100	88.9
				celiac 2 vs control 1	18	0	0	24	100	100	100	100

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Hansson 2000 Sweden	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/r **Population Type:** • n/a **Reference test:** • n/r **First test:** • serology test **Controls biopsied:** • no **Biopsy criteria:** ESPGAN **Checked IgA def.** • no **Studied tests** IgA EMA; IgA serum; IgG AGA; IgA tTG; human erythrocyte tTG; tTG GP liver **Methodology:** •IgA-EMA-ME; tTG, ELISA (human) **Cut-off:** n/r	**Celiac Group 1** • 22 untreated pts (biopsy proven CD) • median age 3 y; range 1-16 y • 14 F, 8 M	**Group 1:** • 23 disease controls (note: no biopsy for the 5/23 control group); GI symptoms; inflammatory bowel disease; cow's milk protein intolerance; food intolerance; miscellaneous GI disorders 17 confirmed biopsy negatives • median age: 6 y, range 1-16 y • 9 F, 13 M	Celiac 1 vs Control 1	21	1	0	23	95.5	100	100	95.8
					21	1	6	17	95.5	73.9	77.8	94.4
					18	4	4	19	81.8	82.6	81.8	82.6
					21	1	1	22	95.5	95.7	95.5	95.7
					22	0	0	23	100	100	100	100
					20	2	1	22	90.9	95.7	95.2	91.7

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Iltanen 1999 Finland	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/r **Population Type:** • 78 children with suspected CD **Reference test:** • biopsy **First test:** • biopsy **Controls biopsied:** • yes **Biopsy criteria:** ESPGAN - CD confirmed at follow-up **Checked IgA def.** • n/r **Studied tests** IgA - EMA **Methodology:** •HU - IF **Cut-off:** >=1:5	**Celiac Group 1** • 23 CD children out of 78 evaluated for suspected CD • median age: overall group 6.5; range 0.5-15.8 • % F: n/r	**Group 1:** • 57 biopsy negative children who were suspected of CD • age: n/r • % F: n/r	celiac 1 vs control 1	20	0	13	44	100	77.1	60.1	100

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Kaukinen, 2000 Finland	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/r **Population Type:** • pts suspected of CD with self reported symptoms upon gluten ingestion **Reference test:** • biopsy **First test:** • simultaneous **Controls biopsied:** • yes **Biopsy criteria:** Villous height to crypt ration <2.0; IEL and HLA also tested **Checked IgA def.** • n/r **Studied tests** IgA EMA; IgA htTg; IgA AGA; IgG AGA **Methodology:** •IF-EMA-HU; Human recombinant TTG ELISA (from Enova Diagnostics web site); AGA ELISA **Cut-off:** ≥1:5; ≥20 U; IgA-AGA ≥0.2 EU/mL; IgG-AGA ≥10 EU/mL	**Celiac Group 1** • 93 pts with self-reported suffering from GI symptoms upon gluten ingestion • mean age: 39 y; range 17-73 y; 9 CD pts • % F: 70; 23 M	**Group 1:** • 84 of 93 with negative biopsy • age: n/r • % F: n/r	celiac 1 vs control 1	7 8	1 0	0 0	84 84	88.9 100 83 17	100 100 45 86	100 100	98.9 100

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Kolho, 1997 Finland	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/r **Population Type:** • suspected CD; or reason to exclude CD **Reference test:** • Biopsy either ingestible capsule technique or endoscopically **First test:** • biopsy **Controls biopsied:** • yes **Biopsy criteria:** revised ESPGAN **Checked IgA def.** • yes – in pts with neg serology **Studied tests** EMA (HU); EMA (ME) **Methodology:** •EMA-ME •EMA-HU **Cut-off:** cut off level was 20% for class IgA and IgG antibodies	**Celiac Group 1** • 53 pts newly diagnosed CD (biopsy proven) • mean age: 6.46 y (range 0.77-19.7 y) • gender: n/r **Celiac Group 2** • 22 pts with CD in remission (CD on GFD) • age (range 0.77-19.7 y) • gender: n/r **Celiac Group 3** • 13 CD pts gluten challenge • age range: 0.77-19.7 y • gender: n/r	**Group 1:** • 48 pts children/adolescents GI complaints; disturbed growth or elevated AGA titres (normal biopsy) • median age 5.39 y (age range 0.63-13.3 y) • % F: n/r **Group 2:** • 20 pts with cow's milk sensitivity enteropathy n/r **Group 3:** • 23 pts with inflammatory bowel disease n/r **Group 4:** • 23 pts with diabetes mellitus n/r	CD untreated and on gluten challenge (group 1) vs control groups 1-4 (combined in the study) CD gluten challenged vs control EMA	50 50 13 13	3 3 0 0	0 0 0 0	114 114 13 13	95 95 100 100	100 100 100 100	100 100 100 100	97 97 100 100

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Kumar, 1989 USA, Israel	**Publication type:** • journal **Study design:** • relevant clinical population and control cases **Ethnicity:** • n/r **Population Type:** • suspected CD; asymptomatic family members **Reference test:** • Crosby-Kugler capsule **First test:** • biopsy **Controls biopsied:** • yes **Biopsy criteria:** ESPGAN + Townley **Checked IgA def.** • no **Studied tests** IgA-EMA **Methodology:** •IgA EMA, IF studies; ME **Cut-off:** n/r	**Celiac Group 1** • 38 children (biopsy proven); untreated CD • age: n/r • gender n/r **Celiac Group 2** • 37 pts; GFD treated. • age: n/r • gender: n/r **Celiac Group 3** • 30 suspected CD; on gluten diet **Celiac Group 4** • 30 suspected cd on GFD	**Group 1:** • 106 asymptomatic family members • age & gender: n/r **Group 2:** • 52 children with chronic diarrhea; 30 ulcerative colitis; 65 Crohn's disease; 21 liver disease; 34 recurrent abdominal pain • age & gender: n/r **Group 3:** • 87 healthy subjects • age & gender: n/r	CD untreated and suspected untreated and 8 biopsy + family members vs Control 1 (106-8), 2, 3	73	3	11	376	96	89	87	96.7

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Ladinser, 1994 Italy	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/r **Population Type:** • n/r **Reference test:** • endoscopic biopsy; IgA EMA **First test:** • biopsy **Controls biopsied:** • yes **Biopsy criteria:** revised ESPGAN **Checked IgA def.** • IgA deficiency excluded by serum testing **Studied tests** IgA EMA **Methodology:** • IgA-EMA, IF studies HU smooth muscle; IgA-EMA, IF studies ME **Cut-off:** n/r	**Celiac Group 1** • 20 biopsy-proven CD pts • mean age: 35 y (range 5-68) • % F: 67%; 20 on gluten-containing diet **Celiac Group 2** • 10 pts GFD (subset of original 30). • age: n/r • gender: n/r	**Group 1:** • 35 pts (CD on GFD) with positive AGA serum samples no histological abnormalities • Mean age: 40 y (range 3-65) • % F: 57% **Group 2:** • 40 non-celiac pts without GI disorder with AGA below average range (IgA<5 and IgG <15) • mean age: 37 y (range 5-65) • % F: 50%	20 active untreated cases vs control group; IgA-EMA (HU) 20 active untreated cases vs control group IgA-EMA (ME)	20 18	0 2	0 0	75 75	100 90	100 100	100 100	100 98

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Lerner, 1994 USA, Israel NB: duplicate of Pacht et al., Isr J Med Sci 1995;31:218 (used data from Lerner et al, 1994 since more complete and larger control group)	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/r **Population Type:** • n/r **Reference test:** • Crosby capsule **First test:** • simultaneous Biopsy & serology **Controls biopsied:** • yes **Biopsy criteria:** criteria of Townley modified by Ingkaran **Checked IgA def.** • no **Studied tests** IgA -AGA; IgG-AGA; IgA-ARA (rat kidney); IgA-ARA (mouse kidney); IgA-EMA ME **Methodology:** AGA=ELISA; ARA=IF rat & mouse kidney; EMA=IF ME **Cut-off:** ELISA 2 SD above the mean of normal value considered positive	**Celiac Group 1** • 34 biopsy proven CD • mean age: 9.6 (range 1.7-17 y) • male:female: 0.6:1.0	**Group 1:** • 9 pts abnormal biopsy pathological control grade II-IV atrophy; (3) Giardia lamblia (3) protracted diarrhea (1) Crohn's (1) HSP (1) intestinal lymphangectasis (1) hypogammaglobulinaemia • mean age 9.3 y (range 2-16 y) • male:female 1.2:1 **Group 2:** • 32 pts with normal intestinal morphology (GCD) • mean age: 8.4 y (range 1-16 y) • male:female 0.9:1	CD1 vs CO IgA-AGA CD1 vs CO IgG-AGA CD1 vs CO IgA-ARA-r CD1vs CO IgA-ARA-m CD1 vs CO IgA-EMA;					52 88 65 53 97	94 92 100 100 98	87 88 100 100 97	74 92 77 71 98

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Lindberg, 1985 Sweden NB: duplicate Jardas et al., Pol J Immunol 1994;19:49	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/r **Population Type:** • suspected malabsorption **Reference test:** • biopsy duodenojejunal flexure Watson pediatric capsule EMA **First test:** • biopsy & serology simultaneous (sera collected ± 2 weeks of biopsy) **Controls biopsied:** • yes **Biopsy criteria:** ESPGAN and Alexander grades; reported sensitivity and specificity for pts with severely damaged mucosa **Checked IgA def.** • yes **Studied tests** IgA-AGA; IgG-AGA; combined IgA/IgG **Methodology:** •DIG-ELISA **Cut-off:** 11 mm IgA; 14mm IgG combined	**Celiac Group 1** • 25 pts untreated CD; 29 pts probable CD • mean age: 12 months (range 7-132 mos) • gender: n/r • 58 pts in total used (with severely damaged mucosa) **Celiac Group 2** • 32 pts CD treated GFD (2nd biopsy) • mean age: 30 mos (range 18-168 mos) • gender: n/r **Celiac Group 3** • 37pts confirmed CD challenged period with gluten • mean age: 36 mos (range 16-192 mos) • gender: n/r	**Group 1:** • 121 pts with other GI disorders i.e., unspecified diarrhea, cow's milk protein intolerance, multiple food allergies, post infectious diarrhea, diarrhea caused by Yersinia enterocolitica • mean age 14 mos (7-130 mos) • gender: n/r **Group 2:** • 23 pts short stature no GI symptoms • mean age: 48 mos (range 12-180 mos) • gender: n/r NB: the study combined control 1 and control 2 (132 pts)	Author reports groups as: CD severely damaged intestinal mucosa vs control IgA AGA CD severely damaged intestinal mucosa vs control IgG AGA CD severely damaged intestinal mucosa vs control IgA/IgG AGA					88 93 97	88 89 83		

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Lindquist, 1994 Sweden	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/r **Population Type:** • suspected CD **Reference test:** • paediatric Watson capsule **First test:** • Simultaneous biopsy & serology **Controls biopsied:** • yes **Biopsy criteria:** ESPGAN; subtotal or partial VA **Checked IgA def.** • yes **Studied tests** IgA-EMA IgA-AGA **Methodology:** IgA-EMA, indirect IF & ME; IgA-AGA, DIG-ELISA **Cut-off:** IgA-AGA >11mm regarded as positive	**Celiac Group 1** • 42 confirmed CD pts (meets ESPGAN criteria) • mean age: 2.7 y (range 5 mos-14.4 y) • % F: 48 **Celiac Group 2** • 10 suspected CD pts with characteristic biopsy but repeat biopsy on GFD was pending • mean age: 2.7 y (range 5 mos-14.4 y) • % F: 48	**Group 1:** • 25 pts with other pathology (CD excluded) • mean age: 2.7 y (range 5 mos-14.4 y) • % F: 48	Celiac 1 & 2 vs control 1 EMA	51	1	3	38	98.1	92.7	94.4	97.5
				Celiac 1 & 2 vs control 1 IgA	45	7	3	39	86.5	92.7	93.7	85

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Maki, 1991 Finland	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/r **Population Type:** • suspected celiac **Reference test:** • paediatric Watson capsule **First test:** • Simultaneous Biopsy & serology **Controls biopsied:** • yes **Biopsy criteria:** ESPGAN; subtotal or partial VA **Checked IgA def.** • yes **Studied tests** IgA-EMA IgA-AGA **Methodology:** IgA-EMA, indirect IF & ME; IgA-AGA, DIG-ELISA **Cut-off:** IgA-AGA > 11mm regarded as positive	**Celiac Group 1** • 42 confirmed CD pts (meets ESPGAN criteria) • mean age: 2.7 y (range 5 mos-14.4 y) • % F: 48 **Celiac Group 2** • 10 suspected CD with characteristic biopsy but repeat biopsy on GFD was pending • mean age: 2.7 y (range 5 mos-14.4 y) • % F: 48	**Group 1:** • 25 pts with other pathology (CD excluded) • mean age: 2.7 y (range 5 mos-14.4 y) • % F: 48	Celiac 1 & 2 vs control 1 EMA	51	1	3	38	98.1	92.7	94.4	97.5
				Celiac 1 & 2 vs control 1 IgA	45	7	3	39	86.5	92.7	93.7	85

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
McMillan, 1991 Ireland	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/r **Population Type:** • suspected celiac **Reference test:** • Crosby capsule "muzzle loaded" **First test:** • simultaneous Biopsy & serology **Controls biopsied:** • yes **Biopsy criteria:** revised ESPGAN **Checked IgA def.** • yes **Studied tests** IgG-AGA; IgA-AGA; IgG-AGA; IgA-AGA; IgG-EMA; IgA-EMA; IgA jejunum; IgG jejunum **Methodology:** • AGA by indirect IF; AGA by ELISA; EMA by IF, ME; jejunum, IF with sections of black hooded rat jejunum as antigen **Cut-off:** titre of one in 20 or greater were considered positive	**Celiac Group 1** • 23 biopsy proven CD + 5 probable CD = 28 pts • Adults age (y): 16-89 • % F: n/r	**Group 1:** • 7 pts 2 did not respond to GFD 4 had ulcerative colitis (1 of those pts had partial VA); 1 had IBS • mean age: n/r • % F: n/r **Group 2:** • 61 normal results of jejunal biopsies	Celiac group 1 vs control group 2	21 21 16 28 11 25 15 21	7 7 12 0 17 3 13 7	4 0 9 0 1 0 0 0	57 61 52 61 60 61 61 61	75 75 57 100 39 89.2 54 75	93.4 100 85 100 98.3 100 92 100	84 100 64 100 92 100 75 100	89 90 81 100 78 95.3 81 90

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Meini, 1996 Italy	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/r **Population Type:** • 65/85 IgA-def pts seen in single immunology clinic 1989-93 (76.5%) **Reference test:** • Watson capsule **First test:** • biopsy **Controls biopsied:** • yes **Biopsy criteria:** partial VA or total VA **Checked IgA def.** • yes **Studied tests** IgA-AGA; IgG-AGA **Methodology:** ELISA **Cut-off:** 25 AU/dL	**Celiac Group 1** • 5 pts; untreated; IgA-deficient • mean age: 8.8 y; range 7-11 y • % F: 80	**Group 1:** • 60 biopsy-negative IgA-deficient	celiac 1 vs control 1	0 5	5 0	0 11	55 44	0 100	100 80	- 31.2	91.7 100

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Picarelli, 2000 Italy	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/r **Population Type:** • suspected CD **Reference test:** • EMA; biopsy (EGD) 2 biopsies **First test:** • EMA serology **Controls biopsied:** • yes **Biopsy criteria:** ESPGAN **Checked IgA def.** • yes 9 of 18 IgA deficient **Studied tests** IgG-EMA; IgA-AGA; IgG-AGA **Methodology:** EMA, indirect IF ME; AGA, in house ELISA **Cut-off:** 0.9 for AGA-IgA and 1.1 for AGA-IgG	**Celiac Group 1** • 30 EMA neg suspected CD; 18 confirmed cases CD • mean age: 10.6 y (age range 2-16 y) • % F: 60	**Group 1:** • 60 disease control (other GI diseases) • mean age: 11.3 y (age range 4-16) • % F: 55 **Group 2:** • 63 healthy children • mean age 10.5 y (age range 3-16 y) • % F: 52 - this group not biopsied so not used **Group 3:** • 12 suspected CD pts biopsy negative	celiac 1 vs control 1 and EMA	18	0	0	135	100	100	100	100
				celiac 1 vs control 3 for IgA-AGA	4	14	4	8	22.2	66.7	50	36.3
				IgG-AGA	6	12	5	7	33.3	58.3	54.5	36.8;

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Poddar, 2002 India	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/r **Population Type:** • suspected CD **Reference test:** • biopsy - endoscopic **First test:** • biopsy **Controls biopsied:** • yes **Biopsy criteria:** ESPGAN (VA and unequivocal response to GFD) **Checked IgA def.** • n/r **Studied tests** IgA–AGA **Methodology:** ELISA **Cut-off:** 5U/mL	**Celiac Group 1** • 50 CD children of 100 biopsied for suspected CD • mean age: 6.3; range 2.5-12 y • % F: n/r	**Group 1:** • 44 biopsy-negative controls who were suspected of CD	celiac 1 vs control 1	47	3	4	43	94	91.5	92	93.5

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Rich, 1990 USA	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** pediatric suction biopsy technique under fluoroscopic control **First test:** • n/r **Controls biopsied:** • yes **Biopsy criteria:** n/r - state "severe" lesion **Checked IgA def.** • n/r **Studied tests** xylose; IgA-AGA; IgG-AGA **Methodology:** D-xylose absorption test; for AGA - ELISA **Cut-off:** xylose ≤25 mg/dL was considered abnormal; AGA - Ab levels >2 SD above the mean of the reference group of normal subjects were considered to be abnormal	**Celiac Group 1** • 15 biopsy-proven CD pts out of 60 consecutive group of children suspicious of having CD • age: n/r • % F: n/r	**Group 1:** • 45 non-CD biopsies out of 60 consecutive group of children suspicious of having CD • age: n/r • % F: n/r	celiac vs CO: Xylose celiac vs CO: IgG AGA celiac vs CO: IgA AGA	14 15 8	1 0 7	24 19 3	21 26 42	93 100 53	47 58 93	36.8 44 72.7	84 100 85.7

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Russo, 1999 Canada	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** endoscopic biopsy **First test:** • n/r **Controls biopsied:** • yes **Biopsy criteria:** ESPGAN **Checked IgA def.** yes (1 pts with CD were found to be IgA deficient) **Studied tests** AGA-IgA; AGA-IgG; EMA ME; EMA HU **Methodology:** for AGA-IgA and IgG ELISA; for EMA - IF on either ME, or HU **Cut-off:** for AGA-gA cut-off was 0.25 EU and for AGA-IgG 0.3 EU; for EMA positive results were considered when a characteristic honeycomb pattern was observed around the smooth muscle	**Celiac Group 1** • 24 pediatric pts with CD (biopsy proven) diagnosed by evaluating consecutive group of 95 children with suspected CD • age: mean 3.5 y, range 7 mos-11 y • % F: 50	**Group 1:** • 71 children with biopsy proved non-CD enteropathies or normal mucosa • age: n/r • % F: n/r	celiac vs control IgG-AGA celiac vs control IgA-AGA; celiac vs control EMA ME celiac vs control EMA HU parallel series					83.3 83.3 75 45.8 100 100	84.5 85.9 88.7 95.8 73 93	64.5 66.7 69.2 78.6 57 86	93.8 93.8 91.3 84 82 100

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Salmaso, 2001 Italy	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/a **Population Type:** consecutive CD Dx 1996-99 **Reference test:** • no info on biopsy **First test:** • biopsy? **Controls biopsied:** • yes **Biopsy criteria:** grades 1-1V Marsh with response to GFD **Checked IgA def.** yes; IgA deficients were excluded **Studied tests** IgA-EMA; IgA-tTG **Methodology:** UC EMA; GP-tTG antigen **Cut-off:** tTG=20 AU	**Celiac Group 1** • 23 adult untreated CD pts • median age: 50; range 27-96 • % F: 56.5 **Celiac Group 2** • 59 children with untreated CD • median age: 8.2 y; range 2-14 • % F: 37.3	**Group 1:** 58 adult age-matched biopsy-negative controls **Group 2:** 48 children age-matched biopsy-neg controls	celiac 1 vs control 1 celiac 2 vs control 2	20 20 59 56	3 3 0 3	0 2 0 0	58 56 48 48	87 87 100 95	100 97 100 100	100 90.9 100 100	95.1 94.9 100 94.1

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Sategna-Guidetti, 1995 Italy	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** endoscopic biopsy **First test:** • n/r **Controls biopsied:** yes, except pts with ulcerative colitis **Biopsy criteria:** Roy-Choudhury criteria; partial or total VA **Checked IgA def.** yes **Studied tests** AGA-IgA; AGA-IgG; Ig AGA total Ig; EMA IgA (ME); JAB IgA **Methodology:** for AGA-IgA and IgG: ELISA; for Ig-AGA, EMA, JAB: IF **Cut-off:** for AGA-IgA and IgG: mean±2 SD of control absorbance index values; for Ig AGA: endpoint still generating a pertubular and a periglomerular reticular pattern; for EMA: results considered when a characteristic honeycomb pattern was observed around the smooth muscle; for JAB: IF at a dilution of 1:5 in phosphate-buffered saline	**Celiac Group 1** • 100 untreated biopsy-proven CD pts • age (y): mean/median n/r; range 14-79 • % F: 71	**Group 1:** • 52 healthy volunteers recruiting among medical and nursing staff • **Group 2:** • 57 pts with non-CD conditions: Crohn's disease (n=38), ulcerative colitis (n=5), lymphoma (n=7), Whipple's disease (n=1), irritable bowel disease (n=6) • age: n/r	celiac vs CO EMA	100	0	0	109	100	100	100	100
				celiac vs CO JAB	100	0	0	109	100	100	100	100
				celiac vs CO IgA-AGA	55	45	0	109	55	100	100	70.8
				celiac vs CO IgG-AGA	78	22	18	91	78	83.5	81.3	80.5
				celiac vs CO Ig-AGA	92	8	5	104	92	95	96	90

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Sblaterro, 2000 Italy	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** n/r **First test:** • biopsy **Controls biopsied:** ESPGHAN **Biopsy criteria:** ESPGHAN **Checked IgA def.** yes **Studied tests** EMA-IgA; GP-tTG IgG and IgA; HR tTG **Methodology:** for tTG: ELISA; for EMA: IF **Cut-off:** normal values for IgA HR-tTG were taken as <13% and for IgG HR-tTG <30%; normal values for IgA GP-tTG were taken as <7% and <16%	**Celiac Group 1** • 65 pts with biopsy proven CD • age (y): median 12, range 2-60 • % F: 52.3	**Group 1:** • 150 healthy donors and 20 pts with Crohn's disease (biopsy proven non-CD enteropathies or normal mucosa) • age: mean/media n/r; range 12-60 y • % F: 44	celiac vs CO EMA	60	5	0	170	93	100	100	97
				celiac vs CO IgG GP-tTG	40	25	3	167	61	98	93	87
				celiac vs CO IgA GP-tTG	53	12	3	167	84	98	94.6	88
				IgG/IgA GP-tTG	58	7	6	164	90	96	91	98
				celiac vs CO IgG HR-tTG	44	21	7	163	67.6	96	86.3	88.6
				celiac vs CO IgA HR-tTG	59	6	1	169	91.5	99	98.3	96.6
				IgG/IgA HR-tTG	64	1	8	162	98.5	95	88.9	99.4

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Sulkanen, Collin, et al. 1998 Finland N.B: used adult group of Sulkanen, Collin, et al., 1998; more complete data located in Sulkanen, Halttunen, et al. 1998	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** n/r **First test:** • n/r **Controls biopsied:** • yes **Biopsy criteria:** subtotal or severe partial VA, crypt hyperplasia, increased IEL **Checked IgA def.** yes (4 pts with CD and 1 CO had IgA deficiency) **Studied tests** HU IgA; HU IgG; ARA IgA and IgG; AGA IgA and IgG **Methodology:** for AGA ELISA; for ARA IF; for HU IF using HU **Cut-off:** HU-Ab positivity included a specific honey-comb IF around the smooth-muscle fibres; AGA IgA - cut off level-0.2 EU/mL; AGA IgG cut off level - 10.0 EU/mL; ARA test was considered positive when the characteristic R1-type ARA pattern was found	**Celiac Group 1** •92 consecutive adult pts with biopsy-proven CD •age (y): median 43; range 20-72 • % F: 71.7	**Group 1:** • 95 disease controls with biopsy proven non-CD enteropathies: 52 undergoing gastroscopic exam for dyspepsia; 14 with Crohn's disease, 29 with ulcerative colitis • age (y): median 44; range 16-76	celiac vs control IgA-HU	78	12	0	95	84.8	100	100	87
				celiac vs control IgG-HU	11	81	0	95	12	100	100	54
				celiac vs control IgA-ARA	72	20	0	95	78.2	100	100	82.6
				celiac vs control IgG-ARA	12	80	2	93	13	98	85.7	53.7
				celiac vs control IgA-AGA	74	18	13	82	80.4	86.3	85	82
				celiac vs control IgG-AGA	35	57	3	92	38	96.8	92	61.7

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Sulkanen, Halttunen, et al. 1998 Finland	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/a **Population type:** • n/a **Reference test:** Watson capsule **First test:** • n/r **Controls biopsied:** • yes **Biopsy criteria:** ESPGAN **Checked IgA def.** yes (n=14) **Studied tests** IgA tTG; IgA EMA; IgA-ARA; IgA-AGA; IgG-AGA **Methodology:** tTG-source n/r ELISA; IgA-EMA-HU; IgA and IgG-AGA-ELISA; IgA ARA-IF) **Cut-off:** for IgA tTG cut off level-10 AU	**Celiac Group 1** • 136 consecutive pts with untreated CD (biopsy proven) • age (y): median 10.7, range 0.8-69.3 • % F: n/r **Celiac Group 2** • 38 pts with CD on GFD of a median duration 48 mos • age: median, range n/r • % F: n/r **Celiac Group 3** • 18 pts on a gluten-challenge • age: median, range n/r • % F: n/r • 11 pts after reintroduction of a GFD (group 4) • age (y): median, range n/r • % F: n/r	**Group 1:** • 154 disease CO with suspicion of CD but biopsy proven non-CD • age (y): median 10, range 0.8-76.0 • % F: n/r	CD group1 vs CO IgA-tTG	129	7	13	194	95	93.7	90.8	96.5
				CD group1 vs CO IgA-EMA	126	10	1	206	92.6	99.5	99.2	94.9
				CD group1 vs CO IgA-ARA	125	11	8	199	92	96.1	94	94.7
				CD group1 vs CO IgA-AGA	115	21	38	169	84.5	81.6	75.2	89
				CD group1 vs CO all IgG-AGA	94	42	55	152	69	73.4	63	78.3

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Tesei, 2003 Argentina	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** endoscopic biopsy **First test:** Simultaneously **Controls biopsied:** yes **Biopsy criteria:** Marsh 2 to 4 - with confirmation **Checked IgA def.** yes (11 pts were diagnosed as IgA deficient having dermatitis herpetiformis) **Studied tests** HU-anti-tTG IgA-HR; EMA-IgA; AGA-IgA; AGA-IgG **Methodology:** for HU-anti-tTG, ELISA; for EMA, ME IF; for AGA, ELISA **Cut-off:** for HU-anti-tTG - 7 AU/mL; for AGA - 20 AU/mL; for EMA - florescence at dilution of 1:5	**Celiac Group 1** • 250 consecutive pts with biopsy proven CD • age (y): mean 39, range 13-79 • % F: 74	**Group 1:** • 176 biopsy proven non-CD disease controls: chronic diarrhea (n=78), chronic microcitic anemia (n=33), first-degree relatives of index cases (n=32), healthy individuals (n=11), malabsorption (n=4), miscellaneous (n=12), undernutrition (n=6) • age (y): mean 40, range 17-83 • % F: 75	CD vs CO: HU-anti-tTG IgA CD vs CO: EMA CD vs CO: AGA IgA CD vs CO: AGA IgG	225 214 159 210	25 36 91 40	9 0 14 25	167 176 162 151	91 86 64 84	96 100 92 86	97 100 92 89	87 83 64 79

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Troncone, 1999 Italy	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** n/r **First test:** • n/r **Controls biopsied:** • yes **Biopsy criteria:** ESPGAN **Checked IgA def.** yes (none of celiac or CO suffered from IgA deficiency) **Studied tests** anti-tTG IgA; anti-tTG IgG; IgA EMA **Methodology:** for tTG-GP, ELISA; for EMA, ME IF **Cut-off:** for anti-tTG - at the 97th percentile of the control group (9% of the reference serum for IgA and 60% - for IgG); for EMA - as the highest dilution giving a positive result (a thin fluorescent network around the smooth muscle fibres)	**Celiac Group 1** • 48 biopsy proven CD pts • age (y): mean 5.7, range 0.9-20 • % F: 58 **Celiac Group 2** • 33 pts who were on GFD of at least 2 y • age (y): mean 7.3, range 4.3-18 • % F: 60.6 **Celiac Group 3** • 10 pts from group 2 who were reintroduced to gluten-containing diet	**Group 1:** • 63 biopsy proven non-CD disease controls: chronic diarrhea (n=16), failure to thrive (n=14), Crohn's disease (n=10), cow's milk protein allergy (n=5), GERD (n=5), recurrent abdominal pain (n=4), sideropenic anemia (n=4), hepatatis C (n=3), Cystic fibrosis (n=2) • age (y): mean 4.2, range 1.1-17 • % F: 49	CD vs CO: anti-tTG IgA CD vs CO: anti-tTG IgG	44 11	4 37	1 1	62 62	91.7 23	98 98	98 92	94 63

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Valdimarsson, 1996 Sweden	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** Watson capsule in 91 pts; fiberoptic endoscopy - in 65 pts **First test:** • biopsy **Controls biopsied:** • yes **Biopsy criteria:** Alexander's classification; partial or subtotal VA **Checked IgA def.** yes (5 had IgA deficiency) **Studied tests** IgA AGA; IgA EMA **Methodology:** for AGA, ELISA; for EMA, IF ME **Cut-off:** for AGA-IgA, 30 units (>92nd percentile in healthy blood donors); for AGA-IgG, 20 units (>91.3th percentile in healthy blood donors); for IgA EMA, characteristic honeycomb pattern was observed around the smooth muscle	**Celiac Group 1** • 19 pts with CD (biopsy-proven) out of 156 pts referred for symptoms suspicious for CD • age: n/r • % F: n/r	**Group 1:** • 137 pts with biopsy proven non-CD; 5 had IgA deficiency and 7-dermatitis herpetiformis • age: n/r • NB: pts with IgA deficiency and dermatitis herpetiformis were excluded from the analysis of sensitivity, specificity, so that CO group was composed of 125 pts	celiac vs CO IgA EMA celiac vs CO IgA AGA	14 15	5 4	0 38	125 87	74 79	100 70	100 28	96 96

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Valentini, 1994 Italy	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** multipurpose biopsy tube, endoscopic **First test:** • simultaneously **Controls biopsied:** • yes **Biopsy criteria:** partial VA or greater **Checked IgA def.** n/r **Studied tests** IgA-AGA; IgG-AGA; IgA-EMA ME **Methodology:** for IgA/IgG AGA, ELISA; for IgA-EMA, IF **Cut-off:** lower limit of AGA positive was 1 EU, based on the mean+-2 SD of the results obtained in a large series of healthy adults; IgA-EMA identified by their reticulin-like staining of the smooth muscle; sera containing antibody at a titre of 1:5 or greater were considered to be positive	**Celiac Group 1** • 100 consecutive adult untreated pts with CD (biopsy proven) • age (y): mean 37.4+-15.4, range 17-79 • % F: 77 **Celiac Group 2** • 33 pts out of 100 group 1 who were on GFD of at least 6 mos (mean 9.9 mos, range 6-12 mos);	**Group 1:** • 30 disease controls (CO) with biopsy proven non-CD conditions: ulcerative colitis (n=8), gastric lymphoma (n=8), Crohn's disease (n=5), Whipple disease (n=3), IBS (n=3), giardiasis (n=2), Graves disease (n=1) • age (y): mean 40+-13, range 18-60 • % F: 47	celiac vs CO IgA EMA	99	1	0	30	99	100	100	96.7
				celiac vs CO IgA/IgG AGA	92	8	3	27	92	90	96.8	77.1

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Vitoria, 2001 Italy	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** n/r **First test:** • n/r **Controls biopsied:** • yes **Biopsy criteria:** subtotal VA **Checked IgA def.** n/r **Studied tests** IgA tTG-ab; IgA EMA **Methodology:** tTG-HR, ELISA; IgA-EMA, IF ME **Cut-off:** for tTG, values of 9 U/mL and more were considered positive; for EMA, reticular pattern of fluorescence in the muscular mucosa at a dilution of serum 1:5 were considered positive	**Celiac Group 1** • 42 biopsy proven CD pts • age: mean 4.2±4.2 y • % F: n/r	**Group 1:** • 28 biopsy proven non-CD disorders • age: mean 6.1+-5.2 y • % F: n/r	celiac vs CO: tTG celiac vs CO: EMA	40 42	2 0	0 0	28 28	95 100	100 100	100 100	93 100

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Vogelsang, 1995 Austria	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/r **Population Type:** • suspected CD **Reference test:** biopsy **First test:** • endoscopic biopsy distal duodenum or Baumgartner-Classen capsule from duodenojejunal flexure **Controls biopsied:** • yes **Biopsy criteria:** modified ESPGAN; flat mucosa; crypt hyperplasia raised IELs **Checked IgA def.** yes; no cases found **Studied tests** IgA-AGA; IgG-AGA; IgA-EMA **Methodology:** ELISA; >95th percentile; ME **Cut-off:** AGA 0.2 AU/mL	**Celiac Group 1** • 49 untreated biopsy-proven CD • age: median 35; range 17-75	**Group 1:** • 53 biopsy-neg controls; median age 31; range 15-79; Crohn's (17); IBS (16); lactase def (7); fibroma (1); duodenitis (1); gastroparesis (3); gastritis (2); chron pancr (1); UC (1); coll colitis (1)	celiac 1 vs control 1	40 36 49	9 13 0	9 14 0	44 39 53	81.6 73.5 100	83 73.6 100	81.6 72 100	83 75 100

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Volta, 1995 Italy	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** n/r **First test:** • n/r **Controls biopsied:** • only disease CO **Biopsy criteria:** Roy-Choudhury criteria **Checked IgA def.** n/r **Studied tests** IgA-EMA on ME; IgA-EMA on HU **Methodology:** IF **Cut-off:** staining of the endomysium around the smooth muscle was considered as a positive result	**Celiac Group 1** • 60 untreated pts with biopsy-proven CD • age (y): median 41, range 14-68 • % F: 70 **Celiac Group 2** • 36 pts with CD on GFD of 12 mos duration	**Group 1:** • 100 disease CO (biopsy proven non-CD in disease CO): ulcerative colitis (n=25), Crohn's disease (n=35), various other GI diseases (n=40) • age (y): median 44, range 25-70 • % F: 68 **Group 2:** • 100 healthy CO (not biopsied) • age: median, range n/r • % F: n/r	celiac vs CO IgA-EMA ME celiac vs CO IgA-EMA HU	57 57	3 3	0 0	100 100	95 95	100 100	100 100	97.1 97.1

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Whelan, 1996 Ireland	**Publication type:** • journal **Study design:** • case control **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** n/r **First test:** • n/r **Controls biopsied:** • only disease CO **Biopsy criteria:** subtotal VA **Checked IgA def.** n/r **Studied tests** HUVEC IgA - human umbilical vein endothelial cells; EMA IgA; (ME) ARA IgA (rat liver); only EMA used for analysis **Methodology:** HUVEC - IF; EMA - IF; ARA - IF **Cut-off:** n/a	**Celiac Group 1** • 25 untreated CD (biopsy-proven) – (UTCD) • age: median, range n/r • % F: n/r **Celiac Group 2** • 16 treated CD on GFD – TCD • age: median, range n/r • % F: n/r	**Group 1:** • 20 disease CO (biopsy proven non-CD in disease CO) - 10 pts with ulcerative colitis and 10 - with Crohn's disease • age: median, range n/r • % F: n/r **Group 2:** • 16 CO with normal intestinal mucosa • age: median, range n/r • % F: n/r	UTCD vs CO: HUVEC UTCD vs CO: EMA ME UTCD vs CO: ARA	25 25 21	0 0 4	0 0 0	36 36 36	100 100 84	100 100 100	100 100 100	100 100 90

Evidence Table 1 (cont'd): Included studies of the sensitivity and specificity of serology for CD

Author, Year, Location	Study Design	Study Population	Control Population	Results								
					4X4 table							
				Comparison	a	c	b	d	Sens	Spec	PPV	NPV
Wolters, 2002 Netherlands	**Publication type:** • journal **Study design:** • cohort **Ethnicity:** • n/a **Population Type:** • n/a **Reference test:** endoscopic biopsy **First test:** • n/r **Controls biopsied:** • yes **Biopsy criteria:** subtotal VA with crypt hyperplasia **Checked IgA def.** n/r **Studied tests** IgA-AGA; IgG-AGA; IgA-EMA; IgA-GP-tTG; IgA-HU-tTG **Methodology:** for AGA-ELISA; for EMA–IF ME; for tTG - ELISA **Cut-off:** for AGA: Ab titers were considered positive if IgA-AGA exceeded 4 U/mL and IgG-AGA exceeded 150 U/mL; for EMA - fluorescence in the muscularis mucosa at a serum dilution equal to or greater than 1:5	**Celiac Group 1** • 52 pediatric pts with biopsy proven CD • age: mean 4.0 y, range 1.1-14.4 y • % F: 73	**Group 1:** • 49 disease CO pts with biopsy proven non-CD • age: mean 5.1 y, range 0.8-19.2 y • % F: 41	CD vs CO: IgA AGA	43	9	10	39	83	86	81	81
				CD vs CO: IgG AGA	43	9	7	42	83	80	86	82
				CD vs CO: IgA EMA	48	4	5	44	92	90	90.5	92
				CD vs CO: anti-GP-tTG IgA	50	2	4	45	96	92	92.6	95.7
				CD vs CO: anti-h-tTG IgA	50	2	0	49	96	100	100	96

HLA DQ2/DQ8

Evidence Table 2: Case-control study evidence for the use of HLA as a marker of CD

Author, Year, Location	Study Design	Study Population	Control Population	Results		
				Celiac Group	Control	
					Group 1	Group 2
Iltanen, 1999, Finland	• case control • monoclonal antibodies used to stain jejunal IELs and mucosal HLA-DR; DQA1*0501 and DQB1*0201 alleles determined	• 21 children with biopsy-confirmed CD • CD criteria: ESPGAN • mean age: 6.1 y, range 0.5-16.3 y • % F: n/r • ethnicity: n/r	**Group 1:** • 67 ethnically-, age-matched pts with biopsy-negative CD • ethnicity: n/r	• DQ2: 19 (90.48) • DQ8: n/a • DQ2 or 8: n/a Prevalence of CD: 0.24	• DQ2: 29.85 Sens: 0.90 Spec: 0.70 PPV: 0.49 NPV: 0.96 • DQ8: n/a • DQ2 or 8: n/a	

Evidence Table 2 (cont'd): Case-control study evidence for the use of HLA as a marker of CD

Author, Year, Location	Study Design	Study Population	Control Population	Results		
				Celiac Group	Control	
					Group 1	Group 2
Sacchetti, 1998, Southern Italy	• case control • used PCR to examine prevalence of HLA heterodimer and HLA DRB104 alleles in healthy subjects, in CD-affected children, and in other age-matched subjects affected by confounding disease	• 122 children with biopsy-confirmed CD • CD criteria: ESPGAN • ethnicity: Italian	**Group 1:** • 32 age-matched pts with GI symptoms but negative biopsy for CD • Ethnicity: Italian **Group 2:** • 116 ethnically-matched healthy adult controls	• DQ2: 106 (86.89%) • DQ8: n/a • DQ2 or 8: n/a	• prevalence of CD: 0.79 • DQ2: 6 (18.75%) Sens: 0.87 Spec: 0.81 PPV: 0.95 NPV: 0.62 • DQ8: n/a • DQ2 or 8: n/a	• Prevalence of CD: 0.51 • DQ2: 31 (26.72%) Sens: 0.87 Spec: 0.73 PPV: 0.77 NPV: 0.84 • DQ8: n/a • DQ2 or 8: n/a

Celiac 2: Incidence and Prevalence of CD

Incidence of CD in the General Population

Evidence Table 3. Incidence of CD in the general population

Study, year; country	Group at risk	Case ascertainment	Incidence	Other observations
Bode, 1996 Denmark	**Region:** County of Copenhagen **Period:** 1976-1991 retrospective **Age groups:** all adults>15 **Size:** 480,581 in 1976; 503,283 in 1992	**Institution(s):** sole 3 teaching hospitals + clinic of region **Register(s):** Discharged Dx w ICD 269-0.01; all small bowel biopsy from path depts; case register of all celiacs **Verification of accuracy:** review of case records **Dx Criteria:** at least 1 biopsy; good response to GFD; gluten challenge with re-biopsy if Dx uncertain **% capture:**	**Outcome measures:** cumulative incidence avg incidence rates adjusted for age/sex **Results:** **# cases:** 101 (64F/37M) **Characteristics of cases:** **median age at Dx:** 40.1 y (range 16-81) **crude incidence:** 1.27/100,000 **cumulative incidence:** 19 y: 198/1000 births	**Women:** **Crude incidence:** 1.55/100,000 **Lifetime cumulative incidence:** 55.8/100,000 **Men:** **Crude incidence:** 0.96/100,000 **Lifetime cumulative incidence:** 35.3/100,000 **Prevalence (1992):** 45.9/100,000

Evidence Table 3 (cont'd). Incidence of CD in the general population

Study, year; country	Group at risk	Case ascertainment	Incidence	Other observations
Collin 1997 Finland	**Region:** Tampere City **Period:** 1975-94 **Age groups:** Adult **Size:** 121,000 in 1970; 147,000 in 1994 **Note:** Screening of high-risk groups in effect Systematic small bowel biopsy during gastroscopy	**Institution(s):** Sole 3 centers that perform gastroscopy; The University Hospital. The City Hospital and the local health centre **Register(s):** Membership list of local Coeliac Society **Verification of accuracy:** All case records and specimen of CD pts were verified **Dx Criteria:** Subtotal or severe partial VA and crypt hyperplasia; clinical or histological improvement on GFD **% capture:** NR	**Outcome measures:** Prevalence: All adult CD pts living in Tampere on Dec 31 1994 per 100 000 population. 5-y Incidence: All adult CD pts dx'ed between 1975 and 1994 per 100,000 pop. **Results:** **# cases:** 301 cases (222 F/79 M) **Characteristics of cases:** **Crude incidence (/100,000 pop/yr):** 1975-79: 1.6 1980-84: 6.8 1985-89: 13 1990-94: 17.2 **Cumulative incidence:** NR	**Median age at Dx:** 39 y (range 1-80) **Prevalence in 1994:** 204/100,000 (95% CI 181-231) **Mode of presentation:** Apparent symptoms: 24% Minor symptoms: 37% Screening: 27% Chance at endoscopy: 13% **Causes for increased incidence:** Use of serologic screening Performance of small bowel biopsy on all pts undergoing gastroscopy Ability of all GP to refer pts to gastroscopy

Evidence Table 3 (cont'd). Incidence of CD in the general population

Study, year; country	Group at risk	Case ascertainment	Incidence	Other observations
Corrao, 1996 Italy	**Region:** Provinces of Turin, Cuneo, Brescia, Umbria region, Sardinia region **Period:** 1990-91 prospective vs retrospective **Age groups:** All ages **Size:** Italian population census in 1991 6,339,194 in 1991 (11.0% Italy); 107,048 live births (9.4% Italy)	**Institution(s):** Dx lists of peds, med, GI depts in all hosp; Dx lists leading Italian hosp **Register(s):** National Health Service records; local Italian Coeliac Society **Verification of accuracy:** capture-recapture method **Dx Criteria:** biopsy without challenge in 47.2% ESPGAN in 52.8% **% capture:** 85.6% **Exclusion(s):** pts residing outside Dx area	**Outcome measures:** crude incidence: #new Dx/yr/100,000 pop cumulative incidence: #cases over period/100,000 births **Results:** **# cases:** 270 (181F/89M) **Characteristics of cases:** **mean age at Dx:** children 3.7; adults 34; overall 21 **Crude incidence:** 2.13/100,000/y **Cumulative incidence (/10,000 births):** 2 y: 5.75 5 y: 8.08 10 y: 10.30 15 y: 11.42	**RR according to gender** (95% CI)**:** Male: 1.0 Female: 1.90 (1.48, 2.45) **RR according to age (95% CI):** 0-15: 1.0 16-39:0.33 (0.25-0.44) 40-59:0.21 (0.15-0.30) >60: 0.11 (0.06-0.18)

Evidence Table 3 (cont'd). Incidence of CD in the general population

Study, year; country	Group at risk	Case ascertainment	Incidence	Other observations
Hawkes, 2000 England	**Region:** South Glamorgan **Period:** 1980-95 retrospective **Age groups:** all ages **Size:** Registrar General mid-yr estimates for total pop, pop <16yr, total live births 415,900 in 1995	**Institution(s):** sole 3 teaching hospitals in Cardiff **Register(s):** Local Celiac Society, letters to GP from consultants **Verification of accuracy:** pathology & dietetic records from the 3 Cardiff teaching hospitals **Dx Criteria:** ESPGAN **% capture:** 100%	**Outcome measures:** Crude incidence: age-weighed avg/100,000 pop **Results:** # **cases:** 137 (98F/39M) **Characteristics of cases:** **mean age at Dx:** children 5.3 yrs (range 0.9-14.9); adults 49.1 yrs (range 19.9-88.2) **Crude incidence:** **children (/100,000/yr):** 1981-85: 2.08 1986-90: 2.53 1991-95: 2.15 **adults (/100,000/yr):** 1981-85: 1.32 1986-90: 2.15 1991-95: 3.08 **Cumulative incidence:** NR	

Evidence Table 3 (cont'd). Incidence of CD in the general population

Study, year; country	Group at risk	Screening	Incidence	Other observations	Incidence	Other observations
Hoffenberg 2003 US	**Region:** Denver Colorado **Period:** 1993-99 prospective **Age groups:** Birth to 7 y; 987 for at least 1 y; 386 for at least 5 y **Size:** Sample from 22 346 newborns screened for HLA, i.e. 987 infants from one institution (St Joseph's Hospital). **Ethnicity:** 56% non Hispanic white 30% Hispanic 7% African American 2% Asian American 5% biracial/other **Genotypes:** HLA-DR3/3; DR3/4, DQB1*0302; DR4, DQ8; DR5/7	**Intervention:** HLA type newborn; follow-up of selected genotypes **Follow-up:** CD serology at 9, 15 and 24 months of age, then yearly. If positive tTG on 2 separate occasions, or if positive tTG plus clinical suspicion, evaluation for small bowel biopsy. **CD serology:** EMA IgA 1993-98; tTG IgA 1998-99; Retesting of EMA positive samples with tTG once test available; Total IgA; tTG IgG used on IgA deficients. **Dx criteria:** Either positive tTG plus biopsy (Marsh 2 or greater), or two consecutive tTG positives 6 months apart	**Outcome measures:** Time to the event of evidence of CD; Cumulative probability of being event-free for the entire population; Cumulative incidence of evidence of CD, stratified by genotype (DR3/3; DR3/x, DRx/x); RR evidence of CD by genotype **Results:** **# cases:** 40 tTG pos at least once; 19 with evidence of CD; 10 biopsy-confirmed; 9 with pos tTG at least twice **Characteristics of cases:** 13F/6M Mean age 4.6 (range 2.6-6.5) 84% non-hispanic whites **Crude incidence:** NR **Cumulative incidence at age 5:** 9/1000 births (95% CI 4-20) **Cumulative incidence at age 5 according to genotype:** HLA-DR3/3: 32/1000 (95% CI 10-110) HLA-DR3/x: 34/1000 (95% CI 30-117) HLA-DR3 neg: 3/1000 (95% CI 0-27)	**RR according to gender:** Female: 3.34 (95% CI 1.00-10.9) **RR according to ethnicity:** Non-hispanic whites: 3.33 (0.7-12.5) **RR according to genotype** (Compared to HLA DR3 negatives): HLA-DR3/3: 9.1 (95% CI 1.7-48) HLA-DR3/x: 5.6 (95% CI 1.5-21)		

Evidence Table 3 (cont'd). Incidence of CD in the general population

Study, year; country	Group at risk	Case ascertainment	Incidence	Other observations
Ivarsson, 2003, Sweden Duplicate: Ivarsson Acta Paediatrica 2000;89:165	**Region:** Entire Sweden **Period:** 1973-97 prospective and retrospective **Age groups:** Children • 0-2 y • 2-15 y **Size:** 258,683 in 1973; 623,439 in 1991 **Dx Criteria:** ESPGAN	**Institution(s):** Prospective reporting from 9 departments of pediatrics **Register(s):** Sweden's National Child health program; central register of CD cases in children <15; Statistics Sweden **Verification of accuracy:** local registers **% capture:**	**Outcome measures:** Incidence rate: #new cases/100,000 PYs; cumulative incidence: # cases up to age/1000 birth in same cohort **Results:** # **cases of CD:** 2151 (1340F/811M) **Characteristics of cases:** **Crude incidence (/100,000PY):** 1973-84: 0-2: 65 (57-74) 1987-94: 0-2: 198 (186-210) 1997: 0-2: 51 (36-70) **Cumulative incidence at 2 y:** 1995: 1.7 (95% CI 1.3-2.1)/1000 births	**RR according to age:** 0-2 y: 1.0 2-15: 0.031 (0.022-0.043) **RR according to gender:** M: 1.0 F: 1.9 (1.7-2.1) **RR according to period:** 1973-84: 1.0 1985-95 1.7 (1.2-2.5) 1996-97 6.8 (4.5-10)

Evidence Table 3 (cont'd). Incidence of CD in the general population

Study, year; country	Group at risk	Case Ascertainment	Incidence	Other observations
Jansen, 1993 Netherlands	**Region:** Entire country **Period:** 1975-91 retrospective **Age groups:** all ages **Size:** Central Bureau for statistics 14,892,574 in 1990	**Institution(s):** **Register(s):** Dutch Coeliac Disease Society for 1992 **Verification of accuracy:** **Dx criteria:** at least 1 biopsy **% capture:** 97%	**Outcome measures:** Incidence: # new cases/100,000 pop/yr; Prevalence rate: # cases/100,000 **Results:** **# cases:** 1983 **Characteristics of cases:** **Crude incidence (/100,000/yr):** 1981-88: 0.65 1988-90: 0.8 1991-92: 1.0	**Prevalence** 1990: 7.9/100,000 (95% CI 6.7-9.3) 1991: 11.3/100,000 (95% CI 9.5-1.0) 1992: 12.7/100,000 (95% CI 11.0-14.5)

Evidence Table 3 (cont'd). Incidence of CD in the general population

Study, year; country	Group at risk	Case ascertainment	Incidence	Other observations
Lopez Rodriguez, 2003 Spain	**Region:** Caceres, south-west Spain **Period:** 1981-99 retrospective **Age groups:** Children 0-14 y; Spanish National Statistics Institute **Size:** 65 137 children in 1998; 98 641 in 1986 66 496 in 1996	**Institution(s):** San pedro de Alcantara hospital, Province referral center for biopsy **Register(s):** **Verification of accuracy:** clinical records **Dx Criteria:** ESPGAN **% capture:**	**Outcome measures:** crude incidence: # cases/100,000 pop/yr **Results:** **# cases:** 157 (90F/67M) **Characteristics of cases:** **mean age at Dx:** 1981-90: 37.4+/-47.4 mo 1991-99:43.9+/-43.7 mo **Crude incidence (/100,000/yr):** 1981-90: 6.87 (5.26-8.83) 1991-99: 16.04 (12.99-19.59)	**RR according to period:** 1981-90: 1 1991-99: 2.31 (1.61-3.31) **RR according to age:** 4-15 y: 1.0 0-4 y (1981-90): 18.2 0-4 y (1991-99):42.04 **% with typical clinical presentation:** 1981-90: 83.6% 1991-99: 58.3% **Significant positive correlation between age at Dx and age of introduction of gluten:** 1981-90: r=0.296 , p=0.0211 1991-99: r= 0.293, p=0.0037

Evidence Table 3 (cont'd). Incidence of CD in the general population

Study, year; country	Group at risk	Case ascertainment	Incidence	Other observations
Magazzu, 1994 Sicily	**Region:** Entire country **Period:** 1975-89 retrospective **Age groups:** children **Size:** 69,945 births in 1989	**Institution(s):** All 4 Sicilian centers of pediatric GI in Catania, Messina and Palermo **Register(s):** Registrations from all 62 Sicilian health authorities; pt list of each GI pediatric centers; pt list of gluten-free products consumption **Verification of accuracy:** Chart review of cases; personal interviews; contact of family pediatrician **Dx Criteria:** ESPGAN **% capture:** **Exclusion(s):** no proof of remission on GFD; at least subtotal VA on biopsy; pts not traced back (1.5%)	**Outcome measures:** cumulative incidence rate by birth cohort: #new cases/#live birth in same birth cohort (95%CI); incidence density: # cases/(#birth in cohort/#yrs of follow up)/15 y obs period **Results:** # **cases:** 1074 (607F/467M) **Characteristics of cases:** 99.4% Caucasian **Crude incidence:** **Cumulative incidence:** 1980 birth year: 1.19/1000 child.yr, 95% CI 0.96-1.45 1984 birth yr: 1.16/1000 child.yr, 95% CI 0.92-1.42 1989 birth yr: 0.13 /1000 child.yr, 95% CI 0.06-0.23	

Evidence Table 3 (cont'd). Incidence of CD in the general population

Study, year; country	Group at risk	Case ascertainment	Incidence	Other Observations
Maki, 1990 Finland	**Region:** Tampere City **Period:** 1961-84 **Age groups:** Children **Size:** 131,394 live births 1960-84 **Exclusion(s):** Cases not born in a certain strict area around the city	**Institution(s):** Department of Paediatrics, University Central Hospital of Tampere **Register(s):** nil **Verification of accuracy:** Questionnaire to pts and their parents; hospital medical records; Child health center charts **Dx Criteria:** Small bowel biopsy; ESPGAN in 44% **% capture:** NR	**Outcome measures:** Crude incidence: # cases/100,000pop/yr **Results:** **# cases:** 96 **Characteristics of cases:** **Crude incidence:** 1964-73: 10.12/100,000 PYs 1974-83: 3.46/100,000 PYs 1960-84: 2.28/100,000 PYs **Cumulative incidence:**	Significant correlation between the age at Dx and duration of breastfeeding Decreased incidence in 0-2; increased incidence in 2-15; increased subclinical presentation in children

Evidence Table 3 (cont'd). Incidence of CD in the general population

Study, year; country	Group at risk	Case Ascertainment	Incidence	Other Observations
Talley, 1994 US	**Region:** Olmstead County **Period:** 1960-90 retrospective all ages **Age groups:** **Size:**	**Institution(s):** **Register(s):** Rochester Epidemiology Project **Verification of accuracy:** Medical records reviewed; biopsy reviewed **Dx Criteria:** **% capture:** **Exclusion(s):** living outside territory at time dx	**Outcome measures:** **Results:** **# cases:** 28 (19F/9M) **Characteristics of cases:** **Crude incidence:** Overall: 1.2 1960-69: 0.9 1970-79: 0.7 1980-90: 1.7 **Cumulative incidence:**	0-14:0.4/100,000PYs 15-44:0.7 45-64:2.5 >65 2.1 prevalence 1991: 21.8/100 000 **Median age at Dx:** median 50 (interQ range 35-62)

Evidence Table 3 (cont'd). Incidence of CD in the general population

Study, year; country	Group at risk	Case ascertainment	Incidence	Other Observations
Weile, 1993 Denmark	**Region:** Copenhagen County **Period:** 1960-88 retrospective **Age groups:** children **Size:** 1,972,864 live births **Dx criteria:** Biopsy-proven (90%); clinically suspected CD (10%)	**Institution(s):** **Register(s):** National Central register of Diagnosis 1977-87 Local register of pts admitted for SB biopsy Celiac Patient Society **Verification of accuracy:** Data twice thoroughly evaluated **% capture:**	**Outcome measures:** Cumulative incidence: #cases/1000 birth in birth cohort **Results:** # **cases:** 176 (103F/73M) **Characteristics of cases:** **Crude incidence:** 1988: 0.102/1000 births **Cumulative incidence:** median: 0.089/1000 live births (range 0-0.182) age 5: 0.118/1000 births	

Prevalence and Incidence in the General Population—Different Geographic and Racial/Ethnic Populations

Evidence Table 4: Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Borch, 2000 Sweden Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** biopsy; IgA & IgG-AGA; IgA-EMA ME **Biopsy criteria/description:** Alexander Grade **Confirmatory test:** n/r **Checked IgA def.** n/r	**Ethnicity:** 459 Swedish; 17 Northern Europeans; outside of Northern Europe **Patient type/# screened:** n=2,000 healthy adults invited to participate in endoscopy study of relation of H.pylori to duodenitis; 482 agreed to participate **Demographics:** age range: 34-75 y **Incidence:**	first serology: 96 confirmation serology: 7 biopsy proven: 9			Not all systematically biopsied; only those with suggestive endoscopic features

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Carlsson, 2001 Sweden Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** AGA, cut-off at 25AU; indirect IF EMA, cut-off at titre >5; biopsy using Watson capsule **Biopsy criteria/description:** used Waston capsule; revised ESPGAN? for classification: normal, subnormal (villous length/crypt length<2, increased number of inflammatory cells in the muscosa with or without damage to the surface epithelium and the brush border), or total VA (flat mucosa) **Confirmatory test:** biopsy **Checked IgA def.** n/r	**Ethnicity:** n/a **Patient type/# screened:** unselected; all children born July 1992 through June 1993 **Demographics:** n=690 (out of 1287/3007 children initially contacted, excluding 22 known CD cases) mean age: 32 mos; range: 27-41 mos **Incidence:**	5.1% (35/690) had either EMA or AGA positive; (13/690) EMA alone: by biopsy: 1.6% (11/35/690) serology positives underwent jejunal biopsies, 11 of them became biopsy-confirmed CDs			

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Catassi, 1996, Italy Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** IgA or IgG-AGA **Biopsy criteria/description:** ESPGAN **Confirmatory test:** EMA and biopsy **Checked IgA def.** n/r	**Ethnicity:** n/a **Patient type/# screened:** School age children **Demographics:** n=17,201; age range: 6-15 y **Incidence:**	1289/17201 (7.5%) AGA-IgG or IgA pos; confirmed with EMA=111/17201; biopsy on 98/111 was pos in 75 + 7 who had no biopsy but CD by various investigations			

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Catassi, 2000, Italy Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** IgG-AGA (7 AU); IgA-AGA (15 AU); IgA-EMA indirect IF (1:5 dilution); biopsy **Biopsy criteria/description:** ESPGAN; Marsh; grade I = isolated increase gamma delta IEL count, grade II= increase gamma delta lymphocyte count with shortened villi/crypt hyperplasia, grade III= subtotal VA **Confirmatory test:** biopsy **Checked IgA def.** yes, 6 found	**Ethnicity:** n/a **Patient type/# screened:** general public, students **Demographics:** n=2,096; pedi age range: 11-15 y % F: 49.5 **Incidence:**	0.86% (18/2,096)			

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Collin, 2002 Finland Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** biopsy **Biopsy criteria/description:** ESPGAN **Confirmatory test:** biopsy **Checked IgA def.** n/r	**Ethnicity:** n/a **Patient type/# screened:** 1. GERD group: regurgitation or heartburn; 2. dyspepsia group; 3. suspected CD **Demographics:** n=9,971; adolescent and adult median age: 58 y; range: 12-93 y % F: 63.5 **Incidence:**	GERD: 0.61% (18/2974)	dyspepsia: 0.77% (41/5347)	CD: 5.33% (88/1650)	

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Corazza, 1997 Republic of San Marino Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** indirect IF EMA titre >1:5; biopsy **Biopsy criteria/description:** n/r **Confirmatory test:** biopsy **Checked IgA def.** no, but mentioned as such that it could have caused some misclassification of pts, but the effect should be minimal given the powerful sensitivity and specificity of EMA test	**Ethnicity:** n/a **Patient type/# screened:** random sample stratified for age and sex **Demographics:** n=2,237; adult median age: 44 y; range, 20-87 y % F: 53.2 **Incidence:**	by both EMA and biopsy: 1 in 559 pts, or 1.79 per 1,000 (0.18%)			

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Csizmadia 1999 Netherlands Duplicate: no	**Publication type:** Journal (research letter) **Study design:** cross-sectional prevalence **Test/methodology:** IgA-EMA (methodology n/r) **Biopsy criteria/description:** n/r **Confirmatory test:** small bowel biopsy, subset of 27 had HLA typing for DQ2 **Checked IgA def.** n/r	**Ethnicity:** n/a **Patient type/# screened:** 6,127 children between ages of 2-4 y general population **Demographics:** 6,127 pediatric pts age: range 2-4 y % F: n/r **Incidence:** Time period: between May 1997-June 1998	1.2% (75/6127) by IgA-EMA, 18/75 refused small bowel biopsy; 0.51% (31/75/6127) VA (CD)			26/27 with VA had the allele HLA-DQ2; prevalence 1:198 in children 2-4 y

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Dickey 1992, Ireland Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** IgA-AGA **Biopsy criteria/description:** no biopsy performed **Confirmatory test:** n/r **Checked IgA def.** n/r	**Ethnicity:** n/a **Patient type/# screened:** healthy blood donors **Demographics:** n=443; adults, age range 18-65 y **Incidence:**	By first serology: 5			

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Fasano, 2003, USA Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** EMA-IF; ME or HUC; positive at 1:10; all positive EMA tested with human tTG ELISA positive at 2 SD above mean of healthy controls; HLA DQ2 and DQ8 **Biopsy criteria/description:** **Confirmation test:** htTG; biopsy; also small subset 98 EMA positive and 114 EMA neg had HLA DQ2/8 tested **Checked IgA def.** • n/r	**Ethnicity:** 94% White; 3% Black; 1.5% Hispanic, 1% Asian, 0.5% other **Patient type/# screened:** 9,019 at risk of CD; 4,126 not at risk **Demographics:** at risk: symptoms of CD (1,326 children, 1,909 adults); CD associated disorders; 4,508 1st deg relatives; 1,275 2nd deg relatives; not at risk: 2,000 blood donors (mean age 39 y, range 19-65 y); 1,119 school children (mean age 12.3 y, range 6-18 y); 1,007 adults and children for routine physical (mean age 39 y, and 13.7 y, range 19-71 y and 2-18 y, respectively) **Incidence:**	1) at risk: a) 1st deg relatives-205/4508 (4.55%); children-54/1294 (4.17%); adults-151/3214 (4.70%); b) symptomatic adults-28/1910 (1.47%); children-53/1326 (4.00%) 2) not at risk-1/4126 (0.75%); adults-27/2845 (0.95%); children-4/1281(0.31%); biopsy in EMA pos-Marsh I-0%; II-30/116 (25.9%); IIIa-46/116 (39.7%); IIIb-24/116 (20.7%); IIIc-16/116 (13.8%); HLA DQ2-76/98 (78%); DQ8-16/98 (16%); DQ2 and 8-6/98 (6%); all EMA pos were also tTG pos			116/350 biopsied

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Green, 2000 USA Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** EGD, biopsy **Biopsy criteria/description:** n/r **Confirmatory test:** n/r **Checked IgA def.** n/r	**Ethnicity:** n/a **Patient type/# screened:** n=1,749 adults; suggestive endoscopic features of CD while undergoing routine endoscopy **Demographics:** n/r **Incidence:**	Biopsy proven: 9			Not all systematically biopsied; only those with suggestive endoscopic features

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Grodzinsky, 1996 Sweden Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** IgA-AGA; both IgA- and IgG-AGA **Biopsy criteria/description:** n/r **Confirmatory test:** ? **Checked IgA def.** n/r	**Ethnicity:** n/a **Patient type/# screened:** healthy adult blood donors **Demographics:** n=1,866; median age 38.5 y, range 18-64 y **Incidence:**	By first serology: 124 by confirmation serology: 11 by biopsy: 7			IgA or IgG-AGA confirmed by having both and by biopsy; prevalence by IgA-EMA n/r

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Hovdenak, 1999 Norway Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** IgA and IgG-AGA, ELISA, cut-off levels: for IgA ≥0.35 and for IgG ≥0.90; IgA-EMA, IF, cut-off n/r **Biopsy criteria/description:** endoscopic biopsy; at least 3 biopsy specimens taken; classification as either normal, partial VA or subtotal VA **Confirmatory test:** biopsy **Checked IgA def.** n/r	**Ethnicity:** n/a **Patient type/# screened:** 2,069 healthy blood donors screened for CD; 1st level of screening: measuring of IgA and IgG AGA; 2nd level of screening: measuring IgA-EMA in AGA positives; 3rd level of screening - biopsy in IgA EMA positives **Demographics:** n=2,069; age: median 39 y, range 18-67 y M/F ratio: 1.65 **Incidence:**	Prevalence of CD in the screening group was 1:340 (7/2069); prevalence of test positivity: 83 of 2069 were positive for AGA; EMA was positive in 8 of these 83 pts; biopsy proven CD was diagnosed in 7 of these 8 pts			Biochemical analysis showed iron deficiency in 2 pts, hypocalcemia in 1 pts and low serum zinc in 5 pts; 4 pts had osteoporosis and another 4 had decreased vitamin E serum concentration

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Ivarsson, 1999 Sweden Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA and IgG AGA - ELISA, cut-off n/r; IgA EMA - IF on a ME, cut-off dilution level varied from 1/20 to 1/320; positive if presence of characteristic reticulin-like staining pattern; serum IgA level measurement using routine nephelometric method, level below 0.05 gL was defined as IgA deficient **Biopsy criteria/description:** criteria for the diagnosis of CD was biopsy demonstrating enteropathy grade III to IV according to Alexander **Confirmatory test:** endoscopic biopsy **Checked IgA def.** yes, using routine nephelometric method; 0.2% (4/1892) pts were found to have IgA deficiency	**Ethnicity:** n/a **Patient type/# screened:** 1,894 individuals taken randomly from 1994 WHO MONICA study population and screened for CD **Demographics:** n=1,894 age (y): median 50, range 25-74 % F: 50 **Incidence:**	Prevalence of CD was 5.3 per 1000 (10/1894); prevalence of newly diagnosed CD was at least 4 per 1000 (8/1892) - (1 woman with positive IgA EMA refused biopsy); as for the prevalence of test positivity: IgA and/or IgG AGA - positive in 23% (438/1892); IgA EMA - positive in 0.5% (9/1892); all CD pts had elevated IgA EMA			CD was more common amongst women (7 F and 3 M) and the older age groups, with the highest prevalence in the interval 55-64 y - 50%

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Jager, 2001 Germany Duplicate: no	**Publication type:** Journal **Study design:** Cross-sectional prevalence **Test/methodology:** IDDM-associated antibodies: islet cell antibodies (ICA) - detected by IF, insulin autoantibodies (IAA) - radioimmunoassay, anti-IA-2 antibodies, and anti-GAD65 antibodies (anti-GADA) - both radioligand binding; thyroid disease-associated antibodies: anti-thyroid peroxidase (anti-TPO) and anti-thyroglobulin (anti-TG) - both ELISA; pernicious anemia-associated antibodies: anti-gastric parietal cell antibodies (anti-GPC) - IF; adrenalitis-associated antibodies: anti-adrenal cortex antibodies - IF; celiac disease-associated antibodies: IgA AGA, IgG AGA, IgA-tTG - all ELISA **Biopsy criteria/description:** n/r **Confirmatory test:** none **Checked IgA def.** n/r	**Ethnicity:** n/a **Patient type/# screened:** 197 pts with a new onset of IDDM diagnosed according to WHO criteria; 882 first-degree relatives; 150 healthy controls without a family history of IDDM **Demographics:** 197 pts with IDDM (age (y): median 16, range 5-27, 43% F); 882 first-degree relatives - 485 were parents (age (y): median 43, range 22-59), 382 siblings and 15 offspring of IDDM pts (age (y): median 16, range 2-41) **Incidence:**	Recent-onset IDDM (n=197): IgG AGA - 10.2%; IgA AGA - 7.6%; anti-tTG IgA - 9.7%; at least 1 antibody positive - 16.8%	First-degree relatives (n=882): IgG AGA - 5.6%; IgA AGA - 2.6%; anti-tTG IgA - 3.2%; at least 1 antibody positive- 7.3%	Healthy control subjects (n=150): IgG AGA - 3.2%; IgA AGA - 2.0%; anti-tTG IgA - 2.6%; at least 1 antibody positive- 4.6%	IDDM associated antibodies and thyroid antibodies were significantly more frequent both in recent-onset IDDM group and first-degree relatives, compared to controls ($p<0.05$); the relevance of IgG/IgA AGA and IgA tTG was significantly higher in the group of recent-onset IDDM compared to first degree relatives and controls ($p<0.05$), but the difference between first-degree relatives and controls did not reach statistical significance; the overall frequency of GPC and adrenal antibodies did not differ significantly among the groups; as for coexistence of antibodies, recent-onset IDDM pts presented with 27% of the subjects testing antibody-positive-specific for 2 or more the envisaged disorders compared with 3.1% in the group of first-degree relatives and 0% of control population ($p<0.05$); recent-onset IDDM (n=197): IDDM-associated antibodies: ICA 82.1%; anti-GADA - 76.0%; anti-IA-2 antibodies - 44.4%; IAA - 37.8%; at lease 1 antibody - 93.4%; Thyroid disease-associated antibodies: anti-TPO+-TG antibodies - 18.4%; pernicious anemia-associated antibodies: anti-GPC - 5.6%; adrenalitis-associated antibodies: anti-adrenal cortex antibodies - 1.0%; first-degree relatives (n=882): IDDM-associated antibodies: ICA 4.9%; anti-GADA - 7.6%; anti-IA-2 antibodies - 4.0%; IAA - 3.4%; at lease 1 antibody - 11.6%; Thyroid disease-associated antibodies: anti-TPO+-TG antibodies - 7.8%; pernicious anemia-associated antibodies: anti-GPC -6.0%; adrenalitis-associated antibodies: anti-adrenal cortex antibodies - 1.1%; healthy control subjects (n=150): IDDM-associated antibodies: ICA 1.3%; anti-GADA - 2.6%; anti-IA-2 antibodies - 0.6%; IAA - 0.6%; at lease 1 antibody - 4.0%; Thyroid disease-associated antibodies: anti-TPO+-TG antibodies - 3.2%; pernicious anemia-associated antibodies: anti-GPC -3.2%; adrenalitis-associated antibodies: anti-adrenal cortex antibodies - 0.7%

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Johnston, 1998 UK Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA AGA - ELISA, normal range of 0-99 (97.5th percentile); IgA EMA - IF on a ME, positive was taken at a titre of 1:5 **Biopsy criteria/description:** enteropathy consistent with CD was considered to include severe partial VA, sub-total or total VA **Confirmatory test:** biopsy; Watson-Crosby capsule; biopsy done in 51 out of 87 pts in MONICA 1991 survey pts and in 20 out of 72 pts in MONICA 1983 survey group **Checked IgA def.** n/r	**Ethnicity:** n/a **Patient type/# screened:** 1) MONICA 1991 survey study: 89 subjects tested positive for CD serology taken from 1823 subjects randomly tested for CD (2 pts were excluded from further analysis because they had CD diagnosis prior to follow-up of the screening program, but included in the assessment of prevalence); 2) MONICA 1983 survey study: 72 CD pts out of 102 who consented to a follow-up of 11.2 y (range 11.3-11.9) **Demographics:** MONICA 1991: 89 subjects with positive CD serology; age (y): mean 50.9, range n/r, 49.4% F; age- and sex-matched 89 controls: age (y): mean 51.1, range n/r, 49.4% F MONICA 1983: 72 with a known CD; age (y): mean 58.1, range n/r, 53% F; no controls were included in this survey **Incidence:**	MONICA 1991: 0.82% [15 (2 with a CD prior to screening program and 13 - screening revealed CD pts) out of 1823] biopsy proven CD making a prevalence of 1:122	MONICA 1983: the estimated prevalence for CD from this survey is 4/1206, or 1:301; if the 2 deceased subjects are included prevalence raises to 6/1206, or 1:201		MONICA 1991 survey: comparing the untreated CD group with controls, there were no differences between symptom profile or laboratory parameters: attendances at their General Practitioners for diarrhea, fatigue, anemia or weight loss; MONICA 1983 survey: comparison of standardized mortality rates between serology-positive subjects and the general population showed no significant difference (4 deaths observed from cancer during a follow-up, compared to the 4.28% (95% CI 1.09, 10.24) expected cancer deaths, giving a relative risk of cancer deaths as 0.94 (95% CI 0.3, 2.4); 13 deaths in total observed during follow-up, compared to 14.11 (95% CI 6.92, 22.23) expected deaths, giving relative risk of all deaths as 0.92 (95% CI 0.5, 1.6)

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Kolho, 1998 Finland Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** EMA – IF HUC; methodology n/r **Biopsy criteria/description:** capsule or endoscopic biopsy; ESPGAN criterias; CD3-positive T cell calculation (limit for high cell number was 77 cells/mm; limit for a moderate cell number was 63 cells/mm); gamma/delta T-cell receptor-bearing cell calculation (limit for a high cell number was 8.2 cells/mm) **Confirmatory test:** capsule or endoscopic biopsy **Checked IgA def.** n/r	**Ethnicity:** n/a **Patient type/# screened:** not at risk: 1,070 adults of Finnish ancestry screened for CD **Demographics:** 1,070 adult population with no clinical signs of CD screened at Helsinki University General Hospital during 1996 **Incidence:**	11 out of 1,070 were positive for IgA EMA; 8 out of these 11 were found to have CD on biopsy, giving the prevalence of CD in this group 1:130			In 7 pts agreeing to start GFD, a 2nd biopsy done after 6 mos, revealed villous structural changes to normal in 1 to subtotal atrophy

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Maki, 2003 Finland Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA-tTG, Celikey assay with low-cut off of 5U/mL; IgG-tTG, ELISA; IgA- and IgG-EMA, IF; total serum IgA - nephelometerical determination with serum levels of <0.05 g/L indicative of IgA deficiency; HLA- DR; DQ2 and DQ8 **Biopsy criteria/description:** endoscopic biopsy; the ratio of villous height and crypt depth less than 2 was considered to be indicative of CD **Confirmatory test:** biopsy in children with IgA EMA and IgA-tTG positivity (in 2001) **Checked IgA def.** yes	**Ethnicity:** n/a **Patient type/# screened:** 3,654 schoolchildren **Demographics:** asymptomatic schoolchildren median age: 12 y, range 7-16 y at the time of 1st sampling (1994) **Incidence:**	Prevalence of CD was 1:99 (37/3654); prevalence of test positivity for CD was 56/3654; 10 of these were identified by symptoms, and of remaining 46, 27 had abnormal biopsy. all but two (52) of antibody pos pts had either HLA-DQ2 or the HLA-DQ8 haplotype; Prevalence of combination of antibody positivity and CD-associated HLA haplotype was 1 in 67			Unclear if 10 pts screened with serology were biopsied or not (abstract vs result table)

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Mazzeti, 1992 Italy Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** IgA-AGA; biopsy **Biopsy criteria/description:** atrophic or completely absent villi **Confirmatory test:** n/r **Checked IgA def.** n/r	**Ethnicity:** n/r **Patient type/# screened:** n=3,022; Roman school children **Demographics:** age range: 13-15 y olds **Incidence:**	By first AGA-IgA: 19 biopsy proven: 18			Not all systematically biopsied; only those with suggestive endoscopic features

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Not, 1998, USA Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgG and IgA-AGA - ELISA (goat immunoglobulin) cut-off was above mean ± 2 SD; IgA-EMA (IgA-AGA or IgG-AGA) - indirect IF on either ME or HU, cut-off n/r **Biopsy criteria/description:** n/r **Confirmatory test:** no biopsy in the 96 IgA/IgG pos or 8 EMA positive pts; HLA haplotype typed in 4 EMA-positive and 23 EMA-negative donors; all 4 EMA-positives carried CD-associated alleles: 3 had DQA1*0501 and DQB1*0201 haplotype and 1 - DQA1*03 and DQB1*0302 **Checked IgA def.** yes; none of 86 donors with positive IgG AGA and negative IgA AGA/EMA had IgA deficiency	**Ethnicity:** 1,740 Caucasians (87%), 230 African-American (11.5%), and 30 Asians (1.5%) **Patient type/# screened:** 2,000 healthy blood donors **Demographics:** mean age: 39 y % F: 48 **Incidence:**	Prevalence of test positivity: 4.8% (96/2000) for IgA and/or IgG; 0.4% (8/2000) for EMA			No biopsy performed to diagnose CD; Confirmatory test: HLA DQA1& DQB1

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Pittschieler, 1996 Italy Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA- & IgG-AGA; IgA-EMA; biopsy **Biopsy criteria/description:** partial or total VA **Confirmatory test:** ? **Checked IgA def.** n/r	**Ethnicity:** 2,778 German; 1,837 Italian **Patient type/# screened:** healthy consenting adults **Demographics:** n=4,615; median age (y) 36.6; range 18-82 **Incidence:**	By first serology: 140 By confirmation serology: 9 By biopsy: 9			

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Riestra, 2000 Spain Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** IgG/IgA-AGA-ELISA; values above 25 (children) and 34 (adults) AU for IgA or above 46 (children) and 42 (adults) AU for IgG were considered positive; IgA-EMA, IF, sera manifesting fluorescence at a titre of at least 1:5 was considered positive; The study was conducted as a 1) two step (determination of IgA/IgG AGA, if positive measuring IgA-EMA); and a 2) one-step protocol (measuring IgA-EMA) **Biopsy criteria/description:** endoscopic biopsy; Marsh **Confirmatory test:** EMA- indirect IF ME; titre≥1/5; biopsy in IgA and IgG pos/IgG AGA pos with IgA deficiency or isolated EMA positives; HLA DQ2 **Checked IgA def.** yes; none were found to have IgA deficiency	**Ethnicity:** n/a **Patient type/# screened:** 1,170 randomly selected individuals from general population **Demographics:** age (y): mean 44.9+-20.9; range 2-89; 55.3% F **Incidence:**	overall prevalence of CD was 2.6:1000; in two-step screening prevalence rate was 0.8:1000 (1/1170, 95% CI 0-55%); in one-step screening prevalence was 1.7:1000 (2/1170, 95% CI 0-6.9%); as for test positivity: IgA or IgG AGA was positive in 15% (174/1170); 1/174 confirmed with EMA and biopsy; 1 CD biopsy proven CD was confirmed even if AGA and EMA were negative; HLA-DQ2 allele was found in 2/3 new CD pts			

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Rostami, 1999 Netherlands Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** IgA-EMA, IF; IgA-nephelometry; methodology n/r **Biopsy criteria/description:** EPSGAN; Marsh **Confirmatory test:** endoscopic biopsy; endoscopically guided capsule (Fujinon) **Checked IgA def.** yes; none were found to have IgA deficiency	**Ethnicity:** n/a **Patient type/# screened:** 1,000 healthy blood donors **Demographics:** n/r	Prevalence of CD in a healthy donors was 1 in 330 (3/1000); as for the prevalence of test positivity: 3/1000 were positive for EMA; biopsy in all of these 3 confirmed CD: 2/3 Marsh IIIb; 1/3 Marsh II			All 3 EMA positive pts with CD carried the known susceptibility alleles for CD - HLA-DQA1*0501 and HLA-DQB1*0201

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Rutz, 2002 Switzerland Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** IgA-EMA, indirect IF on ME; IgA-tTG- ELISA with lower threshold value 0.2 g/L; IgG-AGA and IgA-AGA **Biopsy criteria/description:** Marsh criteria; endoscopic biopsy **Confirmatory test:** endoscopic biopsy **Checked IgA def.** yes (0/1450)	**Ethnicity:** n/a **Patient type/# screened:** 1,450 students **Demographics:** age range 12-18 y; 871 (60.1%) F	Prevalence of CD was 1 in 132 (0.75%; 11/1450); as for the prevalence of test positivity: 11/1450 EMA/tTG positive; 10/11 (1refusal) EMA/TTG/AGA/AGG positive (second level of screening); 9/10 (1 refusal) biopsied: 8/9 Marsh III			Assessing prevalence of CD authors included 2 pts who were EMA and TTG positive but refused biopsy as well as 1 pts with positive tests and a normal mucosal histology, calling it latent CD; biopsy proven CD was diagnosed in 8 pts, but prevalence was calculated taking into account 11 pts

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Sanders, 2003 UK Duplicate: no	**Publication type:** Journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgG and IgA - ELISA; EMA-indirect IF of ME; methodology n/r **Biopsy criteria/description:** Revised Marsh **Confirmatory test:** biopsy **Checked IgA def.** yes	**Ethnicity:** n/a **Patient type/# screened:** 1,200 randomly selected individuals divided in 2 groups: visitors and pts: 1) 609 pts with a non CD-related symptoms in 338; 2) visitor group 591 individuals with a non-CD associated symptoms found in 409 (69.2%) **Demographics:** 1) pts group: age (y): median 48, range 16-91; 64.4% F; 2) visitors group: age (y): median 45, range 18-85; 61.1% F **Incidence:**	Prevalence of CD in primary care population was 1% (12/1200; 95% CI 0.4-1.3%); as for the prevalence of test positivity: 13.5% (162/1200) were antibody positives: 139 - IgG AGA positive, 10 - IgA AGA positive; 4 both IgA/IgG AGA positive, 3 - only EMA positive, 4 - EMA and IgG AGA positive; 2 all antibody positive; 23 pts were eligible for biopsy; out of 22 biopsies (1 pts refused) CD was confirmed in 12 pts			In the whole group of 1,200 screened individuals there were 3 subgroups of pts suffering from: 1) IDA - n=64 (5.3%); 2) IBS - n=123 (10.25%); and 3) fatigue - n=92 (7.7%); prevalence of CD in IDA group was 4.7% (3/64, 95% CI 0-9.8%); prevalence of CD in IBS group was 3.3% (4/123, 95% CI 0.1-0.6%); prevalence of CD in the fatigue group was 3.3% (3/92, 95% CI 0-7%)

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Sjoberg, 1994 Sweden Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgG AGA- ELISA, cut-off 330; IgA AGA- cut-off 8.5; (arbitrary cut-off values adopted based on normal samples) **Biopsy criteria/description:** Marsh; Watson Capsules **Confirmatory test:** biopsy **Checked IgA def.** n/a	**Ethnicity:** n/a **Patient type/# screened:** 1) 1,537 consecutive healthy blood donors; 2) 384 school children; 3) 944 women **Demographics:** 1) age (y): mean 38, range 19-70; 27.3% F; 2) 12 y; 51% F; 3) 57 y women only **Incidence:**	Prevalence of CD in blood donors was at least 1 in 1,500; as for test positivity: 22/1537 (1.43%) pts were positive for IgG and IgA AGA; 13 of these 22 pts were biopsied and 1 of 13 had biopsy confirmed CD	12 y old children- 15/384 (3.9%) were positive for IgG and/or IgA AGA	57 y old women- 11/944 (1.17%) were positive for IgG and/or IgA AGA;	No biopsy results for IgA IgG positive school children and middle aged women was reported

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Sjoberg, 1999 Sweden Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA-AGA, ELISA values above 8.5 arbitrary units were considered positive; IgG-AGA, ELISA values above 330 arbitrary units were considered positive; EMA, IF ME **Biopsy criteria/description:** subtotal and total VA considered diagnostic of CD. Infiltrative lesions, i.e., increased level of IELs were also considered as CD **Confirmatory test:** small bowel biopsy **Checked IgA def.** n/r	**Ethnicity:** n/a **Patient type/# screened:** 1,970 blood donors **Demographics:** 685 women 1,285 men (adults) mean age: 41.2 y, range 18-70 y % F: 35 **Incidence:** Time period: between Oct. 1996-Feb.1997 (4 mos)	Positive results from 1970 pts IgG AGA- 60/1970 (3%) IgA AGA- 150/1970 (7.6%); both IgA and IgG- were 25/210 for a total of 185/1970 (9.4%) who had either IgA or IgG. Those 185 serum samples were analysed for EMA 3/185 positive 3 had small bowel Bx, 2 subtotal VA, 1- total VA. One had classic CD and 2 had infiltrative lesions and were on GFD with improvement. Thus prevalence rate for confirmed CD 4:1970 (0.20%) if infiltrative lesions regarded as CD prevalence 6:1970 (0.30%)			

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Trevisol, 1999 Italy Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA-EMA; biopsy **Biopsy criteria/description:** subtotal or total VA **Confirmatory test:** ? **Checked IgA def.** n/r	**Ethnicity:** White Caucasians **Patient type/# screened:** healthy adult blood donors **Demographics:** n=4,000; mean age 35 y; range 18-60 **Incidence:**	By first serology: 10 By biopsy: 10			

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence				Comments
			Group 1	Group 2	Group 3	Group 4	
Ventura, 2001 Italy Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA-EMA , indirect IF using HU **Biopsy criteria/description:** ESPGAN a single specimen from the duodenal junction with a Watson capsule **Confirmatory test:** intestinal biopsy **Checked IgA def.** yes	**Ethnicity:** n/a **Patient type/# screened:** 19,791 children visiting family pediatrician over a 2 y period. Inclusion criteria "at risk": short stature; recurrent abdominal pain; IDA; enamel hypoplasia; recurrent aphthous stomatitis; autoimmune disease (such as IDDM, juvenile arthritis, autoimmune thyroiditis), occult hypertransaminasemia, IgA deficiency Down syndrome or CD in a first degree relative 240 met criteria; first-degree relatives of newly diagnosed CDs of this study (n=17?) **Demographics:** 240 (103 male 43%) mean age 4.8 y **Incidence:** Time period: Oct. 31, 1995- Oct. 31, 1997	Overall at risk 18/240 were EMA positive and confirmed positive with intestinal biopsy (7.5%)	Stature growth defect 8:105 (7.6%)	Recurrent abdominal pain 3:45 (6.6%)	Sideropenic anemia 4:17 (23.5%)	
			Group 5	**Group 6**	**Group 7**	**Group 8**	
			Autoimmune disease 1:19 (5.2%)	Down syndrome 1:11 (9.0%)	CD first-degree relative 1:14 (7.1%)		

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Volta, 2001 Italy Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA-EMA-HU **Biopsy criteria/description:** Roy-Choudhry; Subtotal villous strophy **Confirmatory test:** ? **Checked IgA def.** n/r	**Ethnicity:** Northern Italians **Patient type/# screened:** 3,483 general population **Demographics:** n=3,483; 12-65 y, only 784 in 12-25 group **Incidence:**	By EMA: 0.57% (20/3483); by biopsy: 0.49% (17/3483)			Prevalence of 0.57% (20/3483) if included three pts with normal villous but with increased IELs

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Weile, 2001 Denmark & Sweden Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** serum IgA; IgG-AGA; IgA-AGA, cut-off >40 units; EMA; in cases of IgA <0.07g/L, IgG-AGA was analyzed **Biopsy criteria/description:** n/a **Confirmatory test:** biopsy in the Swedish sample only **Checked IgA def.** yes; prevalence in both population of blood donors was 0.3%	**Ethnicity:** n/a **Patient type/# screened:** blood donors **Demographics:** Denmark: n=1,573 adults mean age: 41.4 y, range >18 y % F 40.9 Sweden: n=1,866 adults mean age = 37.6 y, range >18 y % F: 31.7 **Incidence:**	Denmark: by IgA-AGA 4% (61/1573), by EMA 0.25% (4/1573)	Sweden: by IgA-AGA 3.2% (60/1866), by EMA 0.27% (5/1866); by biopsy 0.27% (5/1866)		

Evidence Table 4 (cont'd): Prevalence/incidence of CD in the general population

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
West, 2003 UK Duplicate: no	**Publication type:** Journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA EMA, indirect IF on commercial ME using 1:10 dilution; tTGA, ELISA >3 U/mL considered positive **Biopsy criteria/description:** no biopsy done **Confirmatory test:** tTGA (no confirmatory test conducted i.e. biopsy) **Checked IgA def.** yes	**Ethnicity:** n/a **Patient type/# screened:** 7,550 general practice unselected **Demographics:** 7,527 adults aged 45-76 y not previously diagnosed with CD, mean age 59 y; 4,444 (59% F) **Incidence:** Time interval: 1990-1995	EMA positive 87/7527 (1.2%); EMA pos and abnormal tTGA 77/87 (89%)			In the whole group of 1,200 screened individuals there were 3 subgroups of pts suffering from: 1) IDA, n=64 (5.3%); 2) IBS, n=123 (10.25%); and 3) fatigue, n=92 (7.7%); prevalence of CD in IDA group was 4.7% (3/64, 95% CI 0-9.8); prevalence of CD in IBS group was 3.3% (4/123, 95% CI 0.1-0.6); prevalence of CD in the fatigue group was 3.3% (3/92, 95% CI 0-7)

Prevalence of CD in Associated Clinical Conditions—Patients with Suspected CD

Evidence Table 5: Prevalence of CD in patients with suspected CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Agardh, 2001 Sweden Duplicate: yes, Celiac 1 (Carlsson et al., Pediatrics 1999;103: 1248)	**Publication type:** journal **Study design:** retrospective cross-sectional prevalence **Test/methodology:** tTG; HLA DQB1; AGA ≥25 AU; EMA titres ≥1:5, biopsies **Biopsy criteria/description:** as described in Carlsson et al., 1999 (Pediatrics 103:1248) **Confirmatory test:** biopsy **Checked IgA def.** n/r	**Ethnicity:** n/a **Patient type/# screened:** Group 1: IDDM (three were known and treated CD cases) Group 2: generally healthy subjects **Demographics:** IDDM group: (n=165) with CD: median age 7 y, age range=1-13 y; 64% F Without CD: median age=10 y, age range 2-19 y, 44% F control group: (n=277) age range 11-16 y, 53% F **Incidence:** n/a	IDDM group: by AGA: 6.8% (11/162); by EMA: 4.9% (8/162); by biopsy: 3.7% (6/162); by IgA-tTG: 5.6% (9/162); by IgG-tTG: 6/162 (3.7%)	control group: by AGA: 8.7% (24/277); by EMA: 0% (0/277); by IgA-tTG: 0% (0/277); by IgG-tTG: 0% (0/277)		Type 1 diabetics having either DQB1*02 or DQB1*0302 had higher IgA-tTG levels than those not having these alleles (p=0.023)

Evidence Table 5 (cont'd): Prevalence of CD in patients with suspected CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Annibale, 2003 Italy Duplicate: no	**Publication type:** journal **Study design:** prospective prevalence **Test/methodology:** IgA-tTG, ELISA normal values were < 7UA/mL **Biopsy criteria/description:** Marsh **Confirmatory test:** Biopsy, antral, gastric body, and duodenal biopsy collected **Checked IgA def.** n/r	**Ethnicity:** n/a **Patient type/# screened:** IDA in premenopausal women **Demographics:** n=59 premenopausal women; age range 22-54 y with IDA Hb<12g/dLF **Incidence:** Time period: March-July 2000	7/59 (11.9%) had positive anti-tTG antibodies titre; biopsy-confirmed: 8.5% (5/59)			40/59 subjects tested positive for various tests including tTG for CD detection and progressed to have upper endoscopy with biopsy

Evidence Table 5 (cont'd): Prevalence of CD in patients with suspected CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Bardella 1991 Italy Duplicate: no	**Publication type:** journal **Study design:** prevalence **Test/methodology:** IgA-AGA **Biopsy criteria/description:** n/r **Confirmatory test:** **Checked IgA def.**	**Ethnicity:** n/r **Patient type/# screened:** suspected CD: iron-deficient, bowel disturbances, chronic intermittent diarrhea, severe malabsorption, tiredness and wt loss, mineral metabolism deficiencies, gluten-intolerance in childhood not further investigated **Demographics:** n=60; median age 28 (range 15-69); 41 F/19 M **Incidence:**	26			

Evidence Table 5 (cont'd): Prevalence of CD in patients with suspected CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Bardella 2001 Italy	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** IgA-tTG >10 AU/mL using GP liver; AGA >12 AU/mL; indirect IF EMA titre>1:10; biopsy **Biopsy criteria/description:** n/a? didn't mentioned the biopsy results, not used for case diagnosis **Confirmatory test:** none **Checked IgA def.** yes, one found	**Ethnicity:** n/a **Patient type/# screened:** suspected CD, confirmed and treated CD pts (to be excluded), disease control group (to be excluded) **Demographics:** n=80 suspected CD; adult; mean age 39 y; age range 17-79 y; 70% F **Incidence:**	50% (40/80)			

Evidence Table 5 (cont'd): Prevalence of CD in patients with suspected CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Bode 1993 Denmark Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** AGA, IgG, IgA **Biopsy criteria/description:** all biopsied **Confirmatory test:** all biopsied criteria not stated **Checked IgA def.** no	**Ethnicity:** n/r **Patient type/# screened:** suspected CD; all children **Demographics:** n=191; 74 F/117 M; median age: 2.75 y (range 0.33-15.5 y) **Incidence:**	14 cases (7.3%)			

Evidence Table 5 (cont'd): Prevalence of CD in patients with suspected CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Carroccio, 2002 Italy Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** serum EMA; serum anti-GP-tTG; serum anti-human-tTG **Biopsy criteria/description:** ESPGAN? Three groups: normal, partial VA or subtotal/total VA; positive for CD if partial or total VA **Confirmatory test:** biopsy **Checked IgA def.** Yes, none found	**Ethnicity:** n/a **Patient type/# screened:** adult pts with suspected CD **Demographics:** n=207; adult; median age 42 y; age range 17-84 y; 52.3% F **Incidence:**	By GP-tTG: 18.8% (39/207); by h-tTG: 14.5% (30/207); by biopsy: 11.6% (24/207)			

Evidence Table 5 (cont'd): Prevalence of CD in patients with suspected CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Chan, 2001 Canada Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** GP-tTG; EMA; biopsy **Biopsy criteria/description:** Carey capsule, or 4-6 duodenal biopsies at time of endoscopy; no grade provided, a diagnosed case=increased number of IELs with associated subtotal or total VA **Confirmatory test:** biopsy **Checked IgA def.** Yes, two found	**Ethnicity:** n/a **Patient type/# screened:** 77 pediatric pts with suspected CD; 16 type I diabetes **Demographics:** n=93; mean age: n/r; range 2 mos to 18 y; % F: n/r **Incidence:**	GI group: 12% (9/77)	DM group: 75% (12/16)		

Evidence Table 5 (cont'd): Prevalence of CD in patients with suspected CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Chartrand, 1997 Canada Duplicate: no	**Publication type:** journal **Study design:** prevalence **Test/methodology:** biopsy **Biopsy criteria/description:** ESPGAN **Confirmatory test:** **Checked IgA def.**	**Ethnicity:** n/r **Patient type/# screened:** suspected CD; n=179 **Demographics:** all children; mean age 5.2 y, range 0.5-18.1 y **Incidence:**	30 (17%); mean age 3.7 y (range 0.6-11.2); 17 F/13 M			

Evidence Table 5 (cont'd): Prevalence of CD in patients with suspected CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Csizmadia 1999, Netherlands Duplicate: no	**Publication type:** journal (research letter) **Study design:** cross-sectional Prevalence **Test/methodology:** IgA-EMA (methodology n/r) **Biopsy criteria/description:** n/r **Confirmatory test:** Small bowel biopsy, subset of 27 had HLA typing for DQ2 **Checked IgA def.** n/r	**Ethnicity:** n/r **Patient type/# screened:** 6,127 children between ages of 2-4 y general population **Demographics:** 6,127 pediatric pts between 2-4 y; gender:n/r **Incidence:** Time period: between May 1997-June, 1998	1.2% (75/6127) by IgA-EMA, 18/75 refused small bowel biopsy; 0.51% (31/75/6127) VA			26/27 with VA had the allele HLA-DQ2; prevalence 1:198 in children 2-4 y

Evidence Table 5 (cont'd): Prevalence of CD in patients with suspected CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Day, 2000 New Zealand Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** IgA, IgG-AGA, EMA **Biopsy criteria/description:** single pathologist, Marsh **Confirmatory test:** biopsy on EMA pos **Checked IgA def.** yes	**Ethnicity:** n/r **Patient type/# screened:** pediatric input or output; single center; suspected CD: failure to thrive, short stature, chronic GI symptoms; DM; histological findings of CD; 6 mos-15 y **Demographics:** mean age: 63 mos (range 6 mos-15 y); % M: 58 **Incidence:**	27/36 EMA+; 5/11 biopsy confirmed			

Evidence Table 5 (cont'd): Prevalence of CD in patients with suspected CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Fasano, 2003 USA Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** EMA-IF; ME or HU; positive at 1:10 ; all positive EMA tested with human tTG ELISA positive at 2 SD above mean of healthy controls; HLA DQ2 and DQ8 **Biopsy criteria/description:** single pathologist, Marsh **Confirmatory test:** htTG; biopsy; also small subset 98 EMA positive and 114 EMA neg had HLA DQ2/8 tested **Checked IgA def.** n/r	**Ethnicity:** 94% White; 3% Black; 1.5% Hispanic, 1% Asian, 0.5% other **Patient type/# screened:** 9,019 at risk of CD; 4,126 not at risk **Demographics:** at risk: symptoms of CD (1,326 children, 1,909 adults); CD associated disorders; 4,508 1st deg relatives; 1,275 2nd deg relatives; not at risk: 2,000 blood donors (mean age 39 y range 19-65 y); 1,119 school children (mean age 12.3 y, range 6-18 y); 1,007 adults and children for routine physical (mean age: 39 y, and 13.7 y; range: 19-71 y, and 2-18 y, respectively) **Incidence:**	1) At risk: a) 1st deg relatives-205/4,508 (4.55%); children-54/1,294 (4.17%); adults-151/3,214 (4.70%); b) symptomatic adults: 28/1,910 (1.47%); children: 53/1326 (4.00%) 2) Not at risk: 1/4,126 (0.75%); adults-27/2,845 (0.95%); children-4/1281(0.31%); biopsy in EMA+ Marsh I-0%; Marsh II-30/116 (25.9%); IIIa-46/116 (39.7%); IIIb-24/116 (20.7%); IIIc-16/116 (13.8%); HLA DQ2-76/98 (78%); DQ8-16/98 (16%); DQ2 and 8-6/98 (6%); all EMA pos were also tTG pos			

Evidence Table 5 (cont'd): Prevalence of CD in patients with suspected CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Fitzpatrick 2001 Canada Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence; authors state that the study is a case-control (doubtful) **Test/methodology:** IgA-EMA-IF using ME; positive when characteristic fluorescence pattern was produced **Biopsy criteria/description:** not performed **Confirmatory test:** None **Checked IgA def.** n/r	**Ethnicity:** n/r **Patient type/# screened:** 92 pts with recurrent abdominal pain screened for EMA positivity; 81 healthy children also screened for EMA positivity **Demographics:** 92 pts with recurrent abdominal pain; age: n/r; 62% F; 81 healthy controls; age: n/r; 42% F **Incidence:**	The prevalence of IgA-EMA positivity in children with recurrent abdominal pain was 1 in 92 (1%, 95% CI 0-6)	The prevalence of IgA-EMA positivity in controls was 1 in 81 (1%, 95% CI 0-7)		

Evidence Table 5 (cont'd): Prevalence of CD in patients with suspected CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Hill 2000 USA Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** IgA-EMA, IF on ME, methodology n/r; IgA and IgG-AGA, ELISA, methodology n/r **Biopsy criteria/description:** Marsh **Confirmatory test:** biopsy **Checked IgA def.** yes	**Ethnicity:** n/r **Patient type/# screened:** 1,200 pediatric group of individuals at risk of CD; pts were assigned to one of 7 groups: 1) chronic diarrhea (n=182); 2) abdominal pain (n=316); 3) IDDM (n=81); 4) short stature (n=259); 5) failure to thrive (n=123); 6) miscellaneous (Down's syndrome, thyroiditis, anemia, unexplained elevation of liver enzymes) (n=47); 7) asymptomatic relatives (n=192) **Demographics:** 1,200 pediatric group of individuals at risk of CD; age: mean n/r; range: 6 mos-20 y; % F n/r **Incidence:**	Prevalence of CD in pts at risk was 1 in 57 (21/1200); prevalence of test positivity: 2.8% (34/1200) was both EMA and AGA positive; 26 of pts (19 EMA positive) underwent biopsy and 21 were diagnosed with CD			15 pts out of 34 EMA positives refused biopsy; thus prevalence of biopsy proven CD in pts at risk was at least 1 in 57; if considered above mentioned 15 pts, prevalence of CD could have been 1 in 33 (36/1200)

Evidence Table 5 (cont'd): Prevalence of CD in patients with suspected CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Hin 1999 UK Duplicate: no	**Publication type:** journal **Study design:** cross-sectional Prevalence **Test/methodology:** EMA (ME); biopsy of positives; IgA levels, IgG-AGA for IgA deficient **Biopsy criteria/description:** Crosby capsule, EGD distal duod in 2 cases **Confirmatory test:** biopsy; 100% positive; IEL: 1/30; mild VA: 1/30; partial VA: 1/30; subtotal VA: 14/30; total VA: 13/30 **Checked IgA def.** yes	**Ethnicity:** n/a **Patient type/# screened:** entry criteria: IBS, anemia, histological findings of CD, malabsorption symptoms, diarrhea, fatigue, thyroid disease, DM, wt loss, short stature, failure to thrive, epilepsy, infertility, arthralgia, eczema; 1,000 screened **Demographics:** n=271 M, mean age: 49.9 y (range 1-84); n=729 F, mean age: 45.2 y, range 6 mos-85 y); 5.3% <10 y, 3.1% aged 80-90 y; % F: 73 **Incidence:** # New cases: 30 pts (8 M:22 F) +ve EMA and +ve biopsy Time period: 30/1 y Control popn: 7 /preceding 1 y in absence of case finding	Prevalence of CD in pts at risk was 1 in 57 (21/1200); prevalence of test positivity: 2.8% (34/1200) was both EMA and AGA positive; 26 of pts (19 EMA positive) underwent biopsy and 21 were diagnosed with CD			126 cases tested for anemia: 21 positive

Evidence Table 5 (cont'd): Prevalence of CD in patients with suspected CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Hoffenberg 2003 USA Duplicate: no	**Publication type:** journal **Study design:** prospective stratified cohort study **Test/methodology:** IgA tTG **Biopsy criteria/description:** Marsh score of 2 (enlarged crypts and increased numbers of intraepithelial lymphocytes) or 3 (any degree of VA) was considered evidence of CD **Confirmatory test:** TG autoantibody seropositivity on two separate occasions at least 6 mos apart; biopsy **Checked IgA def.** yes	**Ethnicity:** non-Hispanic white 56%, Hispanic 30%, African-American 7%, Asian-American 2%, or biracial/other 5% **Patient type/# screened:** 987 infants with high-risk genotypes: HLA DR3-3; DR3/4, DQB1*0302; DR4, DQ8 DR5/7 (since 2000) **Demographics:** 987 infants (tested over 5 y span) gender n/r **Incidence:** Time period: newborns between Dec 1993-Sept 1999		40/987 tested positive for tTG; 19/40 met criteria for CD (10 with intestinal biopsy and 9 with persistent tTG autoantibody seropositivity)		By the age of 5 y, the adjusted risk estimate of the frequency of evidence of CD in general Denver population is 0.9%(95% CI, 0.4-2.0) or 1 in 104 (1:49 to 1:221).

Evidence Table 5 (cont'd): Prevalence of CD in patients with suspected CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Hogberg, 2003 Sweden Duplicate: no	**Publication type:** journal **Study design:** longitudinal follow-up, incidence & prevalence **Test/methodology:** IgA-AGA, ELISA cut-off for a positive outcome 42.5 units; IgA-EMA, indirect IF using ME, antibody titre defined as the highest serum dilution yielding positive fluorescence; IgA-TGA, commercially available ELISA, highest cut-off value for positive results >30 units. **Biopsy criteria/description:** revised ESPGAN. Grade 1 normal; Grade 2 mild; Grade 3 moderate, Grade 4 total VA **Confirmatory test:** Small bowel biopsy **Checked IgA def.** yes, 5 cases found	**Ethnicity:** n/a **Patient type/# screened:** 120 first degree relatives of CD pts **Demographics:** n=56 parents (adults), mean age 53.6 y, range 43-78 y; n=44 siblings (ped and adult), mean age 27.4 y, range 15-49 y; n=20 offspring, mean age 6.5 y, range 1-16 y; gender n/r **Incidence:** Time period: 20 y follow-up study. Original study period Sept 1975-Feb 1981	10/120 (8.3%) prevalence, 2 were diagnosed in the original study 20 y prior, 8 new cases from the present follow-up study group. (biopsy confirmed); serum results: IgA-AGA 8/120 pos; IgA-EMA 0.5% (6/120) pos; IgA-TGA 3.8% (4/104) pos (3 pts with positive biopsy were not tested for IgA TGA)			IgA deficient pt, IgG antibodies positive. Biopsy findings were in serologically positive relatives IgA-TGA was performed in sera of n=104 when access to assay became available 1 yr later.

Evidence Table 5 (cont'd): Prevalence of CD in patients with suspected CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Pittschieler, 2003 Italy Duplicate: no	**Publication type:** journal **Study design:** prospective prevalence **Test/methodology:** EMA, HUC examined by fluorescence. Absence of binding was considered a negative test; HLA typing was done exclusively once diagnosis of CD was confirmed (micro-lymphocytotoxic technique) **Biopsy criteria/description:** Small intestinal biopsy of first jejunal loop. Ferraris Watson capsule. Normal values being <3.2 cells/mm γ/δ T-cell receptors by immuno-histology **Confirmatory test:** biopsy, HLA typing **Checked IgA def.** yes, none found	**Ethnicity:** n/a **Patient type/# screened:** 92 first-degree relatives of CD pts. Yearly testing over 12 y period **Demographics:** n=92 at risk (first-degree relatives; 18 offspring and 74 siblings); aged 2-18 y **Incidence:** Time period: 12 y time period Jan 1990-Dec 2001	6.5% (6/92) confirmed CD by both serology & biopsy within a few months of CD diagnosis in one of their relatives. 5 total VA and 1 partial VA; over 2-5 y a further 5.8% (5/86) confirmed positive with both HUC EMA-IgA and biopsy 1 partial VA and 4 total VA; combined prevalence=12% (11/92); 11/11 were carriers of HLA DQ2/ heterodimers			all 11 were clinically silent for CD

Evidence Table 5 (cont'd): Prevalence of CD in patients with suspected CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Thomas, 1992 England Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** biopsy **Biopsy criteria/description:** normal histology; mild enteropathy; moderate enteropathy; severe enteropathy; response to GFD **Confirmatory test:** n/r **Checked IgA def.** yes	**Ethnicity:** n/r **Patient type/# screened:** pediatric pts presenting with chronic diarrhea **Demographics:** n=381; 64% <2 y; 20% 2-5 y; 16% 5-15 y; % F: 41.5 **Incidence:**	2/97 mild enteropathy; 1/38 moderate enteropathy; 27/34 severe enteropathy=7.9%			

Evidence Table 5 (cont'd): Prevalence of CD in patients with suspected CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Tursi, 2003 Italy Duplicate: no	**Publication type:** journal **Study design:** prevalence **Test/methodology:** IgA IgG, ELISA lower limit of positivity of IgA 0.2 EU/mL and IgG 10.0 EU/mL; IgA-EMA, indirect IF on ME; IgA-tTG, ELISA using GP liver substrate, lower limit of positivity was 7 UA/mL **Biopsy criteria/description:** Marsh criteria 6 biopsies from small bowel from the second part of duodenum. Marsh Type I -'infiltrative' lesions with >30 lymphocytes/100 epithelial cells; Type II-'infiltrative/hyperplastic' lesions; Type III-'partial (sub)total VA; partial VA Marsh IIIa); subtotal VA Marsh IIIb); and total VA as Marsh IIIc) **Confirmatory test:** Biopsy **Checked IgA def.** yes	**Ethnicity:** n/r **Patient type/# screened:** 111 first-degree relatives of pts with CD **Demographics:** at risk n=111 first-degree relatives: mean age 28.7 y, range (10-65 y); 38 M, 73 F **Incidence:** n/r	CD diagnosed in 49/11 screened relatives (44.14%) prevalence; Prevalence AGA 36.73%; EMA 38.78% ; anti-tTG 44.89%			Prevalence of antibodies was higher in severe histological lesions (Marsh IIIb-c) than in not so severe lesions (Marsh I-IIIa). Note: prevalence of AGA was higher than that of EMA/anti-tTG in less severe histological lesions

Evidence Table 5 (cont'd): Prevalence of CD in patients with suspected CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
van Mook, 2001 Netherlands Duplicate: no	**Publication type:** journal **Study design:** retrospective prevalence **Test/methodology:** EGD; upper digestive tract endoscopy in 10/35; duodenal biopsies taken in 15/35 **Biopsy criteria/description:** Marsh **Confirmatory test:** biopsy **Checked IgA def.** no	**Ethnicity:** n/r **Patient type/# screened:** 35 pts with IDA, anaemia defined as Hb <8.0 mmol/L in men or < 7.4 mmol/L in women. Iron deficiency defined as a serum ferritin level ≤20 µg/L for men or ≤10 µg/L in women; or serum iron concentration ≤45 µg/dL with a transferrin saturation of 10% or less, or the absence of iron stores in bone marrow biopsy specimens. **Demographics:** n=35 pts: median age 71 y, range 22-89 y; 22 F (63%) and 13 M (37%), **Incidence:** n/r	2.9% (1/35) Marsh IIIc on both biopsy and endoscopy			

Evidence Table 5 (cont'd): Prevalence of CD in patients with suspected CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Ventura, 2001 Italy Duplicate: no	**Publication type:** journal **Study design:** cross-sectional Prevalence **Test/methodology:** IgA EMA, indirect IF using HUC **Biopsy criteria/description:** ESPGAN a single specimen from the duodenal junction with a Watson capsule **Confirmatory test:** intestinal biopsy **Checked IgA def.** yes	**Ethnicity:** n/r **Patient type/# screened:** 19,791 children visiting family pediatrician over a 2 y period. Inclusion criteria "at risk": short stature; recurrent abdominal pain; IDA; enamel hypoplasia; recurrent aphthous stomatitis; autoimmune disease (such as IDDM, juvenile arthritis, autoimmune thyroiditis),occult hypertransaminasemia, IgA deficiency Down's syndrome or CD in a first degree relative; 240 met criteria; first-degree relatives of newly diagnosed CDs of this study (n=17?) **Demographics:** n=240: mean age 4.8 y; 103 M (43%) **Incidence:** Time period: Oct. 31, 1995-Oct. 31, 1997	Overall at risk 18/240 were EMA positive and confirmed positive with intestinal biopsy (7.5%);	Stature growth defect 8/105 (7.6%)	Recurrent abdominal pain 3/45 (6.6%)	
			Group 4	**Group 5**	**Group 6**	
			Sideropenic anemia 4/17 (23.5%)	Autoimmune disease 1/19 (5.2%)	Down syndrome 1/11 (9.0%)	
			Group 7			
			CD first degree relative 1/14 (7.1%)			

Prevalence of CD in High-Risk Patients—Type I Diabetes

Evidence Table 6: Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Agardh, 2001 Sweden Duplicate: yes, Celiac 1 – Carlsson et al., Pediatrics 1999;103: 1248	**Publication type:** journal **Study design:** retrospective cross-sectional prevalence **Test/methodology:** tTG; HLA-DQB1; AGA≥25 AU; EMA titres≥ 1:5, biopsies **Biopsy criteria/description:** as described in Carlsson et al., 1999 Pediatrics 103:1248 **Confirmatory test:** biopsy **Checked IgA def.** n/r	**Ethnicity:** n/a **Patient type/# screened:** 1. IDDM (3 were known and treated CD cases); 2. generally healthy subjects **Demographics:** ped; 1. IDDM group (n=165)- with CD: median age 7 y, age range 1-13 y, 64% F; without CD: median age 10 y, age range 2-19 y, 44% F. 2. control group (n=277)- age range 11-16 y, 53% F **Incidence:**	IDDM group: by AGA: 6.8% (11/162); by EMA: 4.9% (8/162); by biopsy: 3.7% (6/162); by IgA-tTG: 5.6% (9/162); by IgG-tTG: 6/162 (3.7%)	Control group: by AGA: 8.7% (24/277); by EMA: 0% (0/277); by IgA-tTG: 0% (0/277); by IgG-tTG: 0% (0/277)		Type 1 diabetics having either DQB1*02 or DQB1*0302 had higher IgA-tTG levels than those not having these alleles (p=0.023)

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Arato, 2002 Hungary Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** EMA, indirect IF; serum IgA measured to avoid false-negative IgA-EMA tests in cases of IgA deficiency (serum IgA<0.2g/L); jejunal biopsy using Crosby capsule for EMA positives; intraepithelial gamma/delta T-cells elevated if >7 cells/mm (95% CI) **Biopsy criteria/description:** other: not described **Confirmatory test:** jejunal biopsy **Checked IgA def.** yes; none found	**Ethnicity:** n/a **Patient type/# screened:** type I diabetes **Demographics:** n=205; randomly selected ped pts with IDDM; mean age 11.6 y; age range 2.0-17.0 y; 42.9% F **Incidence:** n/a	By EMA: 11.7% (24/205); by biopsy: 8.3% (17/205)			Randomly selected subject pool, considered more representative of the population than in the other studies. No significant difference among EMA positive children with or without jejunal VA

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Bao, 1999 USA Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** tTG (ELISA) >0.05=positive; IgA-EMA using indirect IF; HLA genotype DQ B1 typing of peripheral WBCs with PCR amplification and hybridization; DQ alpha-typing performed with ampliType; DQ2, DQ8 **Biopsy criteria/description:** Other: not described **Confirmatory test:** *consent to biopsies in only 20 of the 98 tTG positives **Checked IgA def.** no, not mentioned	**Ethnicity:** n/a **Patient type/# screened:** type I diabetes **Demographics:** n=847; children and adult; mean age 14.5 y; range 0.7-77.7 y; % F? **Incidence:** n/a	By tTG only: 11.6% (98/847); by tTG & EMA: 5.8% (49/847); by biopsy: 1.8% (15/20/98/847)			Levels of tTG IgA and IgA-EMA were correlated: r=0.44, p=0.002. Prevalence of tTG was higher in diabetics with HLA DQ2 or DQ8.

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Barera, 1991 Italy Duplicate: no	**Publication type:** journal **Study design:** **Test/methodology:** AGA IgA then if negative, IgG AGA **Biopsy criteria/description:** subtotal VA **Confirmatory test:** none **Checked IgA def.**	**Ethnicity:** n/a **Patient type/# screened:** type I diabetes **Demographics:** n=498; children **Incidence:** n/a	By first AGA-IgA: 30 Biopsy proven: 16			Levels of tTG IgA and IgA-EMA were correlated: r=0.44, p=0.002. Prevalence of tTG was higher in diabetics with HLA DQ2 or DQ8.

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Barera, 2002 Italy Duplicate: no	**Publication type:** journal **Study design:** prospective cohort; 6 years follow-up [did not give incidence measures, only prevalence] **Test/methodology:** EMA, indirect IF; serum IgA <0.05 g/L in the presence of normal IgG and IgM were regarded as selective IgA deficiency; duodenojejunal biopsy in >8 y children with positive EMA; upper endoscopy for younger children, mucosal histology **Biopsy criteria/description:** Marsh; three types of lesions: 1. Infiltrative lesion normal mucosa, 2. Hyperplastic lesion with enlarged crypts infiltrated by IELs, 3. Some degree of VA with inflammation and hyperplastic crypts; diagnosis considered positive with demonstration of type 2 or 3 lesion **Confirmatory test:** biopsy **Checked IgA def.** yes, 2 selective IgA deficiency	**Ethnicity:** n/a **Patient type/# screened:** type I diabetes **Demographics:** n=273; children; mean age 8.28 y, age range 0.6-18.7 y; 42.5% F **Incidence:** n/a	By 1 EMA only: 5.5% (15/273); after second EMA: 3.7% (10/273); by biopsy: 3.3% (9/10/273); if add the 1 excluded case (because diagnosed CD before developed IDDM): 3.6% (10/274)			Levels of tTG IgA and IgA-EMA were correlated: r=0.44, p=0.002. Prevalence of tTG was higher in diabetics with HLA DQ2 or DQ8.

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Calero, 1996 Spain Duplicate: no	**Publication type:** journal **Study design:** **Test/methodology:** IgA-AGA if positive on two occasions **Biopsy criteria/description:** subtotal VA **Confirmatory test:** none **Checked IgA def.**	**Ethnicity:** n/a **Patient type/# screened:** type I diabetes **Demographics:** n=141; children **Incidence:** n/a	By first AGA-IgA: 12 Biopsy proven: 4			

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Cronin, 1997 Ireland Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** EMA; biopsy **Biopsy criteria/description:** other: not mentioned **Confirmatory test:** biopsy **Checked IgA def.** no, not mentioned	**Ethnicity:** n/a **Patient type/# screened:** IDDM pts **Demographics:** n=101 diabetic pts and n=51 controls; adolescent and adult; age range for diabetic pt 15-59 y. Other info n/a **Incidence:** n/a	By EMA: 7.9% (8/101) By biopsy: 5.0% (5/101)			

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
De Block, 2001 Belgium Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional Prevalence] **Test/methodology:** islet cell antibodies (ICA) - IF, cut-off level was >12 JDF; antibodies to glutamic acid decarboxilase-65 (GADA) - radiobinding assay, cut-off level was >2.6% tracer bound GADA; tyrosine phosphate antibodies (IA2A) - radiobinding assay, cut-off level was >0.5% tracer bound IA2A; thyroid peroxidase antibodies (aTPO) - radiobinding assay, cut-off was >100 U/mL; parietal cell antibodies (PCA) - IF, positivity at >1:20 dilution; antibodies to intrinsic factor (AIF) - radiobinding assay; anti-adrenal antibodies (AAA) - IF; anti-EMA - IF on a ME, positivity at >1:10 dilution; HLA DQ **Biopsy criteria/description:** no biopsy performed **Confirmatory test:** none **Checked IgA def.** n/r	**Ethnicity:** all Caucasians **Patient type/# screened:** 399 pts with IDDM screened for different autoimmune diseases (176 children <18 y; 223 adults) **Demographics:** 399 pts with IDDM screened for different autoimmune diseases; age (y): mean 26±16, range n/r; 53% F **Incidence:** n/a	Prevalence of CD in 399 pts with IDDM was 0.75% (3/399); prevalence of test positivity: 1) ICA - 39% (157/399); GADA - 70% (278/399); IA2A - 44% (177/399); aTPO - 22% (87/399); PCA - 18% (73/399); AAA - 1% (5/399); IgA EMA - 2% (9/399)			IgA-EMA was detected in 2.3% of IDDM pts particularly in HLA-DQA1*0501-DQB1*0201 subjects

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
De Vitis, 1996 Italy Duplicate: no	**Publication type:** journal **Study design:** **Test/methodology:** IgA, IgG then IgA EMA **Biopsy criteria/description:** Marsh VA **Confirmatory test:** biopsy **Checked IgA def.**	**Ethnicity:** n/a **Patient type/# screened:** type I diabetes **Demographics:** n=1,114; children & adults **Incidence:** n/a	By first IgA: 121 Biopsy proven: 63			78/121 biopsied

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Fasano, 2003 USA Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** EMA, IF ME or HU; positive at 1:10 ; all positive EMA tested with human tTG ELISA positive at 2 SD above mean of healthy contols; HLA DQ2 and DQ8 **Biopsy criteria/description:** single pathologist, Marsh **Confirmatory test:** htTG; biopsy; also small subset 98 EMA-positive and 114 EMA-neg had HLA DQ2/8 tested **Checked IgA def.** n/r	**Ethnicity:** 94% White; 3% Black; 1.5% Hispanic, 1% Asian, 0.5% other **Patient type/# screened:** 9,019 at risk of CD; 4,126 not at risk **Demographics:** at risk: symptoms of CD (1,326 children, 1,909 adults); CD-associated disorders; 4,508 1st deg relatives; 1,275 2nd deg relatives; not at risk: 2,000 blood donors (mean age 39 y range 19-65 y); 1,119 school children (mean age 12.3 y, range 6-18 y); 1,007 adults and children for routine physical (mean age 39 y, and 13.7 y, range, 19-71 y, and 2-18 y) **Incidence:**	1) At risk - a) 1st deg relatives - 205/4,508 (4.55%); children - 54/1,294 (4.17%); adults - 151/3,214 (4.70%); b) symptomatic adults - 28/1,910 (1.47%); children - 53/1,326 (4.00%); 2) Not at risk - 1/4126 (0.75%); adults - 27/2,845 (0.95%); children - 4/1281(0.31%); biopsy in EMA pos - Marsh 1- 0%; 2 - 30/116 (25.9%); 3a - 46/116 (39.7%); 3b - 24/116 (20.7%); 3c - 16/116 (13.8%); HLA DQ2-76/98 (78%); DQ8 - 16/98 (16%); DQ2 and 8 - 6/98 (6%); all EMA pos were also tTG pos			

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Frazer-Reynolds, 1998 Canada Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA EMA - IF using ME, positive if staining at a dilution of 1:10; total serum IgA, measured using rate nephelometry **Biopsy criteria/description:** Carey capsule; Marsh criteria **Confirmatory test:** biopsy **Checked IgA def.** yes; none of IDDM pts had IgA deficiency; 3 pts in suspected malabsorption group were found to have IgA deficiency	**Ethnicity:** 94% white; 3% Black; 1.5% Hispanic, 1% Asian, 0.5% other **Patient type/# screened:** 236 pts with IDDM screened for CD; 56 pts who underwent intestinal biopsy for suspected malabsorption **Demographics:** 236 pts with IDDM; age (y): mean n/r, range 1-18; 50% F; 56 pts with GI complaints; age (y): mean n/r, range n/r; 43% F **Incidence:**	Estimated prevalence of CD in 236 pts with IDDM was 5.1% (12/236; 95% CI 2.7-8.8); as for test positivity: none were IgA deficient; 19 pts were IgA EMA positive; 2 refused biopsy; of 17 pts with IgA EMA, 12 had CD on biopsy	Estimated prevalence of CD in 56 pts with suspected malabsorption was 9% (5/56); as for test positivity: 3 pts were EMA positive and all had biopsy proven CD; 3 pts were IgA deficient and EMA negative and 1 of them was found to have CD on biopsy; 1 of 50 IgA-sufficient and EMA negative pts had biopsy proven CD		Sensitivity and specificity of IgA EMA for the detection of CD in all 73 biopsied pts (both IDDM and GI pts) were 88.2% (15/17; 95% CI 63.6-98.5), respectively; when the 3 IgA deficient pts were excluded, sensitivity increased to 93.7% (15/16; 95% CI 69.8-99.8) and specificity remained unchanged (49/54, or 90.7%; 95% CI 79.7-96.9)

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Gillett, 2001 Canada Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA EMA, IF on HU, starting dilution 1:5; positive sample were tested at increased dilutions until fluorescence disappeared; IgA tTG, ELISA; reference range of 140 AU/mL or less was calculated to include 3 SD above the mean (99% confidence limit) **Biopsy criteria/description:** endoscopic biopsy; ESPGAN criteria; 4 biopsy specimens from distal duodenum; elevated IEL count was defined as more than 40 IELs per 100 enterocytes **Confirmatory test:** biopsy **Checked IgA def.** yes; using Nor-Partigen Total IgA Kit; 1 pt was found to be IgA-deficient	**Ethnicity:** 209 White, 12 East Indian, 7 Asian, 4 First Nation, 1 African **Patient type/# screened:** 233 pts with IDDM screened for CD **Demographics:** 233 pts with IDDM screened for CD; age (y): median 12.9, range 1.3-19.2; 46% F **Incidence:**	Prevalence of CD in IDDM pts was 7.7% (18/233); prevalence of test positivity: 8.2% (19/233) was both EMA and AGA positive and all were white; 18 of these 19 pts underwent biopsy (1 was previously diagnosed with CD and was not offered biopsy); CD was confirmed in 14 of these 18 pts			

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Hansen, 2001 Denmark Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA AGA - ELISA, cut-off n/r; IgA EMA - IF on a ME; IgA tTG - ELISA; thyroid antibodies: thyroid peroxidase (TPO) and thyroglobulin (TG), using radioimmunoassay **Biopsy criteria/description:** endoscopic biopsy; CD was diagnosed when mucosa showed partial or total VA, crypt hyperplasia and IEL infiltration **Confirmatory test:** endoscopic biopsy **Checked IgA def.** yes; none was IgA deficient	**Ethnicity:** n/a **Patient type/# screened:** 106 pts with IDDM screened for CD; 106 aged-, and sex-matched healthy controls **Demographics:** 106 pts with IDDM screened for CD; 2 had been previously diagnosed with CD; age (y): median 12.8, range 2.3-18.2; 47% F; median duration of IDDM 4.8 y, range 0.2-13.3 y; 1 pt had a second-degree relative with CD; 106 aged-, and sex-matched healthy controls; age (y): median 12.9, range 1.3-18.3; 47% F; none had relatives with CD **Incidence:**	Screening revealed 9 biopsy proven CD in the 104 pts with IDDM, giving a prevalence of CD 10.4% (95% CI 4.6-16.2%), (11/106 - 2 pts had CD prior to screening); as for test positivity: of 104 tested pts 7 had IgA AGA, 19 - IgG AGA, and 10 - IgA EMA+ IgA tTG; 9 out of 10 EMA+tTG positive pts underwent biopsy (1 refused) and were found all of them to have CD	Control: none had been diagnosed with CD; as for test positivity: 1 had IgA AGA, 9 - IgG AGA, none - EMA or tTG		Screening revealed IDDM pts with CD were significantly younger than the group of IDDM without CD (p=0.017); IDDM+CD group also had an earlier onset of diabetes: median 3.2 y (range 0.7-9.3 y), compared with 7.4 y (range 1.3-16.6 y) in pts without CD (p=0.005); in pts with IDDM+CD the height standard deviation score (SDS) was significantly lower compared with diabetics without CD (p=0.019); no statistically significant difference with regards of weight SDS and body mass index SDS; thyroid antibodies were significantly more frequent in IDDM+CD (36% - 4/12), whereas 12/94 (13%) of IDDM without CD and 2/106 (2%) of controls had detectable thyroid antibodies (p=0.04)

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Jager, 2001 Germany Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** DDM-associated antibodies: islet cell antibodies (ICA) - detected by IF, insulin autoantibodies (IAA) - radioimmunoassay, anti-IA-2 antibodies, and anti-GAD65 antibodies (anti-GADA) - both radioligand binding; thyroid disease-associated antibodies: anti-TPO and anti-TG - both ELISA; pernicious anemia-associated antibodies: anti-gastric parietal cell antibodies (anti-GPC) - IF; adrenalitis-associated antibodies: anti-adrenal cortex antibodies - IF; CD-associated antibodies: IgA AGA, IgG AGA, IgA-tTG - all ELISA **Biopsy criteria/description:** n/r **Confirmatory test:** none **Checked IgA def.** n/r	**Ethnicity:** n/a **Patient type/# screened:** 197 pts with a new onset of IDDM diagnosed according to WHO criteria; 882 first-degree relatives; 150 healthy controls without a family history of IDDM; **Demographics:** 197 pts with IDDM (age (y): median 16, range 5-27, 43% F); 882 first-degree relatives - 485 were parents (age (y): median 43, range 22-59), 382 siblings and 15 offsprings of IDDM pts (age (y): median 16, range 2-41) **Incidence:**	Recent-onset IDDM (n=197): IgG AGA - 10.2%; IgA AGA - 7.6%; anti-tTG IgA - 9.7%; at least 1 antibody positive - 16.8%	First-degree relatives (n=882): IgG AGA - 5.6%; IgA AGA - 2.6%; anti-tTG IgA - 3.2%; at least 1 antibody positive- 7.3%	Healthy control subjects (n=150): IgG AGA - 3.2%; IgA AGA -2.0%; anti-tTG IgA - 2.6%; at least 1 antibody positive- 4.6%	

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Kaukinen, 1999 Finland Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA EMA - IF, screening dilution of 1:5 was considered as a positive; IgA and IgG AGA - ELISA, for IgA AGA lower limit of positivity was 0.2 EU/mL and for IgG AGA - 10.0 EU/mL **Biopsy criteria/description:** endoscopic biopsy; ESPGAN criteria; 7 forceps biopsy specimens; histological classification as 1) normal; 2) mild partial VA; 3) severe partial VA; 4) subtotal VA; 3 and 4 were considered as CD **Confirmatory test:** endoscopical biopsy; performed in 6 (10%) previously diagnosed CD and in 28 pts without the diagnosis of CD; (23 refused biopsy, 3 - lost to follow-up, 2 - died); HLA DR and HLA DQ alleles in a dilution of 1:1500 using PCR/RFLP method **Checked IgA def.** n/r	**Ethnicity:** n/a **Patient type/# screened:** 62 adult pts with more than one autoimmune endocrinologic disorder **Demographics:** 67% F); ATD+addison's disease - 4 (age (y): median 52, range 34-53; 50% F); APECED - 4 (age (y): median 26, range 17-51; 25% F); IDDM+alopecia areata - 3 (age (y): median 20, range 17-30; 67% F); ATD+alopecia areata - 3 (age (y): median 48, range 33-57; 100% F); ATD+addison's disease+alopecia areata - 1 (age (y): median 42; 0% F); IDDM+addison's disease - 1 (age (y): median 32; 100% F) **Incidence:**	In total 7 (11%) out of 62 pts were diagnosed to have biopsy-proved CD; 6 (10%) were previously diagnosed with CD, and 1 (3.6%) of the 28 pts undergoing biopsy also was diagnosed with CD; EMA was positive in 1 (3.6%) pts with a newly diagnosed CD; IgA AGA - in 7 (25%), and IgG AGA - in 1 (3.6%); 14 of 26 subjects were HLA-DQ2 positive, in addition 4 had a celiac-type DQ8 haplotype; thus, 18 (69%) had celiac-type and 8 (31%) - a non-celiac genetic background;			IDDM - insulin dependent diabetes mellitus; ATD - autoimmune thyroid disorder (including autoimmune thyroiditis or Graves' disease); APECED - autoimmune polyendocrinopathy-candidosis-cetodermal dystrophy; 11% prevalence does not necessarily depict the true frequency of CD in pts with more than one autoimmune disorder; all 62 pts were treated in endocrinological clinic, which can lead to referral bias

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Kordonouri, 2000 Germany Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA anti-tTG - ELISA antibody titers above 15 were considered positive; EMA - IF, fluorescence at 1:5 dilution was considered as a positive; IgA and IgG AGA - ELISA, for both methods a level above 35 AU were considered as a positive **Biopsy criteria/description:** endoscopical biopsy; Marsh criteria **Confirmatory test:** endoscopical biopsy; Marsh criteria (stated as a reference 9); done in 13 CD pts out of 23 suspected CD pts (7 lost to follow-up, 3 - refused biopsy); 9 were found to have biopsy proven CD; IEL per 100 enterocytes counted after immunohistochemical staining for CD8+ cells (normal values in children up to 26 IEL per 100 enterocytes) **Checked IgA def.** yes; 9 pts out of initial 529 had below normal IgA levels and were excluded from further analysis	**Ethnicity:** n/a **Patient type/# screened:** 520 pts with IDDM and no clinical signs of CD **Demographics:** 520 pts with IDDM; age (y): median 14.2, range 1.6-27.3; 47% F; medium duration of IDDM 4.0 y, range 0-23.6 y **Incidence:**	23 (4.4%) of the 520 pts with IDDM were found to be positive for IgA anti-tTG; 18 (3.5%) - for EMA; and 18 (3.5%) - for IgA AGA; prevalence of biopsy proven CD in the whole group of IDDM pts was at least 1.7% (9/520), because in 10 pts with increased IgA anti-tTG levels biopsy was not performed; all 9 pts with biopsy confirmed CD were positive for anti-tTG, and 8 - for EMA			Study demonstrates that elevated IgA anti-tTG, especially when present on more than one occasion, may be more sensitive than EMA for detecting a silent form of CD; while prevalence of positive IgA anti-tTG was 4.4%, the incomplete prospective histological assessment by biopsy precludes a final calculation of the prevalence of biopsy-proven CD in this pts group

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Lampasona, 1999 Italy Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional Prevalence **Test/methodology:** human tissue transglutaminase C - TGCA IgA and TGC IgG; the threshold for positivity was the upper first percentile of normal controls, respectively 0.9 AU for IgG TGCA and 0.3 for the IgA TGCA; typing of HLA for 128 pts with IDDM either using the standard microcytotoxicity test on lymphocytes isolated by immuno-magnetic beads or by sequence specific PCR on DNA extracted from blood mononuclear cells; subjects grouped as DR3/X, DR4/Y, DR3/4, or DRX/Y **Biopsy criteria/description:** endoscopical biopsy; Marsh criteria **Confirmatory test:** n/r **Checked IgA def.** n/r	**Ethnicity:** n/a **Patient type/# screened:** 287 pts with a new onset IDDM; 119 pts with NIDDM; 213 pediatric controls with no family history of diabetes **Demographics:** 287 pts with IDDM; age (y): median 10, range 0.8-33; 41% F; 119 pts with NIDDM; age (y): median 65, range 42-87; 45% F; 213 controls; age (y): median 4.4, range 0.1-11.2; 49% F **Incidence:**	Increased levels of TGCA were detected in 122 of 287 pts with IDDM (43%; CI 37-48%); of the pts 25 (9%; CI 6-13%) had raised levels of IgA TGCA and 121 (42%; CI 36-48%) - IgG TGCA; in 24 pts (8%; CI 5-12%) both IgA and IgG TGCA were found	In the NIDDM group 2 pts had low levels of IgG TGCA only; as for HLA typing, 34 had DR3/4 genotype, 41 - DR3/X, 38 - DR4/Y and 15 - DRX/Y; increased TGCA were found in 22 (65% CI 46-80%) with the DR3/4 genotype, 19 of those with DR4/Y (50%; CI 33-67%), 16 with DR3/X (39%; CI 24-55%) and in 3 with DRX/Y (20%; CI 4-48%)		Almost 10% of pts have autoimmunity typical of CD and another 30% have low level TGCA antibody binding; this high prevalence suggests either involvement of the gut in the pathogenesis of IDDM or that transglutaminase is a secondary autoantigen resulting from beta-cell destruction

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Li Voon Chong, 2002 UK Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA EMA - IF; IgA AGA - ELISA; total serum IgA **Biopsy criteria/description:** n/r **Confirmatory test:** biopsy **Checked IgA def.** yes	**Ethnicity:** n/a **Patient type/# screened:** 509 pts with IDDM assessed during 1998; treated autoimmune thyroid disease - AITD - present in 28 (5.5%); 7 (1.4%) out of 509 had known CD; 38 pts with coexisting IDDM and AITD, but without known CD studied during 1999; and 112 pts with IDDM alone and without known CD assessed during 1999 **Demographics:** age (y): mean 29.4, range 16-45; 41% F; 38pts with coexisting IDDM and AITD screened for CD: age (y): mean 35.6, range 17-53; 66% F; 112 pts with IDDM alone screened for CD: age (y): mean 30.6+-9, range 16-57; 43% F **Incidence:**	1) prevalence of known CD in 509 IDDM pts was 1.4% (7/509); 2 of these 7 pts later on developed AITD	2) in the 38 pts with coexisting IDDM and AITD screening revealed 1 pts with increased IgA EMA, but normal IgA AGA levels; none were diagnosed with biopsy proven CD, making a prevalence of CD 0% in this group	3) in the 112 pts with IDDM alone screening for CD revealed 2 pts with increased IgA EMA and normal IgA AGA, 1 of whom had biopsy confirmed CD, making a prevalence of CD in this group 0.9%	Undiagnosed CD in pts with both IDDM and AITD is not increased compared with pts with IDDM alone; because 2 of 7 pts with known CD subsequently developed hypothyroidism, authors suggest that pts with known CD and IDDM be annually screened for AITD

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Lorini, 1996 Italy Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA and IgG AGA: ELISA; levels more than 2 SD were considered abnormal - for IgA >10 AU; for IgG - >7500 AU; R1-ARA: IF on rat kidney and liver; IgA EMA: IF on a distal ME; HLA-DR3, HLA-DR4, HLA-DR7 **Biopsy criteria/description:** n/r **Confirmatory test:** intestinal biopsy in 6 pts with constantly elevated IgA AGA during a follow-up **Checked IgA def.** n/r	**Ethnicity:** n/a **Patient type/# screened:** 133 pts with IDDM; 45 age-matched, apparently normal controls **Demographics:** 133 IDDM - age (y): mean 14.1, range 1.4-28.4; 47.3% F; 53 pts were considered at the onset of IDDM and 49 of them were also investigated for a 1-10 y follow-up; 45 aged-matched controls (CO); age: mean, range n/r **Incidence:**	IDDM pts: 3.75% (5/133)	IDDM pts: 3.75% (5/133)		At the diagnosis of IDDM IgA AGA were elevated in 32% (17/53), and during a follow-up it decreased within a normal limits in 13 pts; out of 32 pts with IgA AGA normal levels at the diagnosis of IDDM, 2 developed IgA AGA increased levels during a follow-up; in all pts with IgA AGA positivity during a follow-up, R1-ARA and EMA levels were also increased; high IgA AGA levels at the onset of IDDM are a transient abnormal immunological response and do not predict the occurrence of CD; they should not be considered a primary indication for performing a diagnostic intestinal biopsy unless R1-ARA and EMA are present too; HLA-Dr3 and/or DR4 were present in all 5 pts with AGA, R1-ARA and EMA positive

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Not, 1998, USA Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgG and IgA-AGA - ELISA (goat immunoglobulin) cut-off was above mean ± 2 SD; IgA-EMA (IgA AGA or IgG AGA) - indirect IF on either ME or HU, cut-off n/r **Biopsy criteria/description:** n/r **Confirmatory test:** no biopsy in the 96 IgA/IgG pos or 8 EMA positive pts; HLA haplotype typed in 4 EMA-positive and 23 EMA-negative donors; all 4 EMA-positives carried CD-associated alleles: 3 had DQA1*0501 and DQB1*0201 haplotype and 1 - DQA1*03 and DQB1*0302 **Checked IgA def.** yes; none of 86 donors with positive IgG AGA and negative IgA AGA/EMA had IgA deficiency	**Ethnicity:** 1,740 Caucasians (87%), 230 African-American (11.5%), and 30 Asians (1.5%) **Patient type/# screened:** 2,000 healthy blood donors **Demographics:** mean age: 39 y % F: 48 **Incidence:**	Prevalence of test positivity: 4.8% (96/2,000) for IgA and/or IgG; 0.4% (8/2,000) for EMA			No biopsy performed to diagnose CD; Confirmatory test: HLA DQA1& DQB1

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Page, 1994, UK	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA-AGA-in-house ELISA; titers of >90 U/L were considered abnormal **Biopsy criteria/description:** n/a **Confirmatory test:** Biopsy **Checked IgA def.** yes (8 pts had IgA deficiency and 1 was found to have CD)	**Ethnicity:** n/a **Patient type/# screened:** 1,785 diabetic pts (43% with IDDM and 57% with NIDDM) **Demographics:** n/r **Incidence:**	Prevalence of test positivity: IGA-AGA was positive in 4.1% (73/1785); 49 of these 73 pts were biopsied; CD was diagnosed in 13 of 49 biopsied pts; in 8 out of 1,765 pts IgA and 1 pt was diagnosed with CD; in general, prevalence of newly diagnosed CD was at least 0.78% (14/1785); the overall prevalence of CD in the whole group (4 pts were previously diagnosed with CD) was at least 1% (18/1789); 0.4% (8/2000) for EMA			Prevalence of CD in IDDM pts group was at least 1:50 compared with 1:340 in pts with NIDDM

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Rensch, 1996 USA Duplicate: no	**Publication type:** journal **Study design:** **Test/methodology:** EMA **Biopsy criteria/description:** Loss of villous architecture, crypt hyperplasia, and increased IELs **Confirmatory test:** none **Checked IgA def.**	**Ethnicity:** n/a **Patient type/# screened:** type I diabetes **Demographics:** n=47; adults **Incidence:** n/a	By first EMA: 3 Biopsy proven: 3			

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Roldan, 1998 Spain Duplicate: no	**Publication type:** journal **Study design:** **Test/methodology:** IgA, IgG AGA (and known cases, and some tested with EMA) **Biopsy criteria/description:** ESPGAN **Confirmatory test:** none **Checked IgA def.**	**Ethnicity:** n/a **Patient type/# screened:** type I diabetes **Demographics:** n=117; children **Incidence:** n/a	By first IgA: 19 Biopsy proven: 7			Mixed group diagnosed by different means

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Rossi, 1993, USA Duplicate: no	**Publication type:** journal **Study design:** **Test/methodology:** EMA **Biopsy criteria/description:** ESPGAN **Confirmatory test:** none **Checked IgA def.**	**Ethnicity:** **Patient type/# screened:** type I diabetes **Demographics:** n=211; children **Incidence:**	By first EMA: 10 Biopsy proven: 3			Only 3/10 biopsied

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Sategna-Guidetti, 1994 Italy Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA EMA - IF ME **Biopsy criteria/description:** Roy-Choudhury capsule from upper jejunum; criteria of Roy-Choudhury **Confirmatory test:** endoscopic biopsy and Roy-Choudhury capsule **Checked IgA def.** no	**Ethnicity:** n/a **Patient type/# screened:** 383 consecutive IDDM adults; 151 CD pts (as true positives) and 250 healthy and diseased controls (as true negatives) to assess IgA EMA test sensitivity and specificity **Demographics:** IDDM - age (y): mean 39, range 16-84; 43.9% F **Incidence:**	Prevalence of test positivity: CD pts -145/151 (96%) EMA positive(1:50-1:>2000); sensitivity of IgA EMA was 96%	Prevalence of biopsy proven CD in IDDM group was at least 2.6% (10/383): 2 pts out of 12 IgA EMA positives refused biopsy; prevalence of IgA EMA positivity was 3% (12/383)	Controls-0/437 EMA positive; specificity of IgA AGA was 100%	Prevalence of CD in IDDM pts group was at least 1:50 compared with 1:340 in pts with NIDDM

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Saukkonen, 1996 Finland Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** gA- reticulin-indirect IF goat anti-human antiserum; IgA and IgG AGA - ELISA cut-off >30% of an intralaboratory standard (intra and inter-assay CV of 5.6% and 10.5% for IgA, and 6.9% and 16.1% - for IgG, respectively) **Biopsy criteria/description:** n/r **Confirmatory test:** Biopsy for all pts with abnormally high levels of antibodies but not for those with initially (diagnosis of IDDM) positive antibodies but negative at follow-up (6, 12, 18, 24 and 36 mos) **Checked IgA def.** no	**Ethnicity:** n/a **Patient type/# screened:** 776 IDDM children **Demographics:** children less than 16 y **Incidence:**	Prevalence of CD in IDDM pts was at least 2.4% (19/776); prevalence of test positivity: at the diagnosis of IDDM and or at 24/36 months follow-up 76/775 positive for IgA-ARA and or IgA-AGA; 35 of these 76 pts were biopsied; in 17 of 35 pts biopsy confirmed CD was found; 2 pts out of 76 pts with a negative antibodies/symptomatic/ were diagnosed by biopsy; overall, 19/776 biopsy confirmed cases; 19/19 positive IgA-ARA; 14/18 genotyped for HLA DR positive for DR3 and 10 (56%) positive for DR4; DQB2 present in 17/18 (94%)			The observed prevalence of CD is an underestimate, because biopsy was not performed in 17 pts who screened positive for IgA ARA or AGA in the follow-up sample

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Schober, 2000 Austria Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** EMA - indirect IF of ME, any positive reading at a dilution of 1:10 was considered as a positive; IgG-AGA and IgA-AGA - ELISA; cut-off for IgG AGA was ->30 AU; for IgA-AGA - >25 AU **Biopsy criteria/description:** Modified Marsh and Crowe; Watson-type capsule **Confirmatory test:** Biopsy **Checked IgA def.** yes	**Ethnicity:** n/a **Patient type/# screened:** 403 children and adolescents with type I diabetes **Demographics:** age (y): mean 12.4, range 1-22; 47.9% F **Incidence:**	Overall prevalence of biopsy proven CD was 1.49% (6/403); as for prevalence of test positivity: 12 pts had increased IgA EMA; 11 of these 12 were biopsied (1 refusal); on biopsy: 3 (0.74%) had Marsh I; 2 (0.49%) - Marsh 0; 1 - Marsh IIIa/c and 5 Marsh IIIc (1.49%)			

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Sigurs, 1993 Sweden Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA- AGA - ELISA, cut off level at least 25 AU; IgA and IgG ARA - IF; titers equal to or diluted more than 1:5 were considered positive **Biopsy criteria/description:** Watson Capsule **Confirmatory test:** Biopsy **Checked IgA def.** yes (3 cases)	**Ethnicity:** n/a **Patient type/# screened:** 436 children with IDDM **Demographics:** age (y): mean 13.6+-4.1, range 2-21 y, 46.3% F **Incidence:**	Minimum prevalence of CD in IDDM pts was 4.6% (21/459); prevalence of newly diagnosed CD was 3.4% (15/436); as for prevalence of test positivity: 4.3% (19/436) were IgA AGA positive; 18 of these 19 pts were biopsied; minimum PPV for IgA ARA was 77% (13 of 17)			

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Sjoberg, 1998 Germany Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA-AGA - ELISA, titre > 8.5 AU was considered positive; IgG-AGA - ELISA, titre > 330 AU was considered positive **Biopsy criteria/description:** Marsh; Watson Capsule or gastroscopy and biopsy **Confirmatory test:** EMA-indirect IF of ME- titre ≥ 5; biopsy **Checked IgA def.** no	**Ethnicity:** n/a **Patient type/# screened:** 1,664 diabetes pts (848 IDDM; 745 NIDDM; 71 secondary diabetes) **Demographics:** IDDM - age (y): mean 46.1, range 17-86; 52.8% F; NIDDM - age (y): mean 61.7, range 24-92; 47.3 F; secondary diabetes - age (y): mean 53.9, range 33-77, 9.8% F **Incidence:**	Prevalence of biopsy proven CD was 1.8% (15/848): 8 out of 848 were previously diagnosed with CD; as for prevalence of test positivity: 258/848 were positive for IgA and/or IgG-AGA; 22/258 were positive for EMA giving a prevalence of 2.6%; 7/20 were biopsy positive (14/20 potential CD:4 death; 3 refused biopsy)	NIDDM- 1/745 previously diagnosed CD; 1/745 EMA positive; prevalence in total 0.27%	Secondary diabetes group- 3/71 IgG-AGA positive; no EMA positives; no previously diagnosed CD; prevalence of CD was 0%	Pts with previously known CD had more symptoms, more deficiency states and more autoimmune diseases than those identified by screening ($p<0.001$); IDDM pts with a diabetes duration of 31-40 y were characterised by a higher prevalence of CD than pts with a duration of less than 30 y (6.7% vs. 1.7%; $p<0.02$)

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Spiekerkoetter, 2002 Germany Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** human tTG: IgA/IgG assay cut-off of 9.0 units, IgG-tTGA cut-off 7.0 units, IgA-tTGA cut-off 8.3 units **Biopsy criteria/description:** Marsh **Confirmatory test:** biopsy **Checked IgA def.** no, not mentioned	**Ethnicity:** n/a **Patient type/# screened:** ped IDDM pts **Demographics:** n=205; ped; median age=12 y 7 mos; age range=3-19.5 y; 47.3% F **Incidence:**	By IgA/IgG-tTG: 6.3% (13/205); by IgG-tTG: 5.4% (11/205); by biopsy 2.9% (6/8/13/205) [only 8 of the 13 with elevated tTGA levels agreed to biopsy]			

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Talal, 1997 USA Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** EMA, cut-off at dilution ≥ 1:10; biopsy **Biopsy criteria/description:** ESPGAN **Confirmatory test:** small bowel biopsy **Checked IgA def.** yes	**Ethnicity:** n/a **Patient type/# screened:** adult diabetic pts **Demographics:** n=185; adult; other info n/a **Incidence:**	By EMA: 4.9% (9/185); by biopsy 2.2% (4/5/9/185) [only 5 of the 9 EMA positives underwent biopsy]			

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Valerio, 2002 Italy Duplicate: Yes (see Celiac 1)	**Publication type:** journal **Study design:** Prevalence **Test/methodology:** IgA AGA, ELISA; IgG AGA, ELISA; IgA EMA, indirect IF (substrate n/r) **Biopsy criteria/description:** ESPGAN **Confirmatory test:** small bowel biopsy **Checked IgA def.** yes	**Ethnicity:** n/a **Patient type/# screened:** 383 type 1 diabetes pts **Demographics:** 383 Type 1 diabetics (194 M, 189 F, 49% F) age < 18 y **Incidence:** Time period: from January 1992 to December 2000	Type 1 diabetics 32/383 (8.3%); 2 out of 32 had IgA deficiency			

Evidence Table 6 (cont'd): Prevalence/incidence of CD in patients with diabetes

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Vitoria, 1998 Spain Duplicate: no	**Publication type:** journal **Study design:** Prevalence **Test/methodology:** AGA; EMA; intestinal biopsy; IAA, GAD65, IA2, ICA tests to assess IDDM-related pancreatic autoimmunity among CD pts **Biopsy criteria/description:** ESPGAN **Confirmatory test:** biopsy **Checked IgA def.** yes, none found	**Ethnicity:** n/a **Patient type/# screened:** confirmed CD pts and IDDM pts; exclude confirmed CD pts for data extraction, irrelevant data **Demographics:** 93 IDDM: pedi; mean age=10.5 y; ?% F **Incidence:**	IDDM group: by AGA 17.2% (16/93); by EMA 7.25% (7/93); by biopsy 6.5% (6/93)			IDDM could develop in pts with silent, non-treated CD through gluten-mediated immune activation

Prevalence of CD in High-Risk Patients—Relatives

Evidence Table 7: Prevalence of CD in relatives of patients with CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Book, 2003 USA Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** IgA-EMA; tTG-ELIZA; HLA DQA1, DQB1 **Biopsy criteria/description:** n/a **Confirmatory test:** biopsy; diagnosis based on positive biopsy or had positive EMA and tTG serologies **Checked IgA def.** n/a	**Ethnicity:** n/a; but all families were Caucasian **Patient type/# screened:** Relatives of CD pts **Demographics:** n=163 first-degree relatives, n=82 second-degree relatives, n=47 first cousins; ped & adult; mean age=?; age range=2-78 y; % F? **Incidence:**	Parents: 14.7%	Siblings: 21.3%	Offspring: 14.7%	HLA DQ available in 34/37 of the seropositives, all but one was DQ2 or DQ8
			Group 4	**Group 5**		
			Second-degree relatives: 19.5%	First cousins: 17.0%		

Evidence Table 7 (cont'd): Prevalence of CD in relatives of patients with CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Corazza, 1997 Italy Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** AGA **Biopsy criteria/description:** Some VA **Confirmatory test:** n/a **Checked IgA def.** n/a	**Ethnicity:** n/a **Patient type/# screened:** Relatives of CD pts **Demographics:** n=328 first-degree relatives **Incidence:**	4%			

Evidence Table 7 (cont'd): Prevalence of CD in relatives of patients with CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Farre, 1999 Spain Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA EMA - IF either on a ME (n=550), or HU (n=119); positive ab 1:5 dilution and characteristic honeycomb staining pattern; IgA AGA - ELISA; values over 40 AU were considered positive; HLA-DQ2 (DQA1*0501 and DQB1*0201 alleles) were assayed in 169 pts; typing performed by PCR amplification **Biopsy criteria/description:** Watson-Crosby capsule; classification: 1) total VA; 2) severe partial VA with crypt hyperplasia; 3) minor non-specific abnormalities; 4) morphologically normal; 1 and 2 were diagnostic criterias for CD **Confirmatory test:** Watson-Crosby capsule; HLA-DQ2 typing performed in 169 first-degree relatives of CD pts, in 60 CD pts and in 50 ethnically matched controls from the general population **Checked IgA def.** yes, results were not explicitly given in the text. Presumably there was no case of IgA deficiency found because the alternative IgG test for such pts were not used or mentioned in the results section	**Ethnicity:** n/a **Patient type/# screened:** 675 first-degree relatives in 227 families of CD probands (149 pediatric and 78 adult pts) **Demographics:** 669 first-degree relatives in 227 families of CD probands (149 pediatric and 78 adult pts) screened for CD (6 were diagnosed with CD prior to screening and excluded from the study, but accounted for overall prevalence); out of 669 relatives: 331 were parents (163 fathers, 168 mothers), 260 siblings (123 brothers and 137 sisters), and 78 children (42 sons and 36 daughters) **Incidence:**	The prevalence of unrecognised CD in first-degree relatives of CD pts was 4.6% (31/669); the overall prevalence of CD in this group is 5.5% (37/675 - 6 relatives of CD pts were diagnosed with CD prior to the study); as for test positivity: IgA EMA was positive in 39/669 (5.8%) and IgA-AGA - in 13/669 (1.9%) relatives; simultaneous positivity occurred in 12 of 669 relatives (1.8%); of 39 EMA-positive relatives, biopsy has been done in 32 pts (7 refused) and CD were found in 31 individuals			HLA-DQ2 was typed in 12 of the 32 EMA-positive relatives who underwent biopsy and was positive in all cases; HLA-DQ2 haplotype was also present in 64% (108/169) of relatives, 93% (56/60) CD pts and 18% (9/50) of controls; all unrecognised CD relatives detected by screening were DQ2 positive

Evidence Table 7 (cont'd): Prevalence of CD in relatives of patients with CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Fasano, 2003 USA Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** EMA - IF; ME or HU; positive at 1:10; all positive EMA tested with human tTG ELISA positive at 2 SD above mean of healthy controls; HLA DQ2 and DQ8 **Biopsy criteria/description:** single pathologist, Marsh **Confirmatory test:** htTG; biopsy; also small subset 98 EMA positive and 114 EMA neg had HLA DQ2/8 tested **Checked IgA def.** n/a	**Ethnicity:** 94% white; 3% black; 1.5% Hispanic, 1% Asian, 0.5% other **Patient type/# screened:** 9,019 at risk of CD; 4,126 not at risk **Demographics:** at risk - symptoms of celiac (1326 children, 1909 adults); CD associated disorders; 4,508 1st deg relatives; 1,275 2nd deg relatives; Not at risk – 2,000 blood donors (mean age 39 range 19-65); 1,119 school children (mean age 12.3, range 6-18); 1,007 adults and children for routine physical (mean age 39, and 13.7, range, 19-71, and 2-18) **Incidence:**	1) At risk - a) 1st deg relatives - 205/4,508 (4.55%); children - 54/1,294 (4.17%); adults - 151/3,214 (4.70%); b)symptomatic adults - 28/1,910 (1.47%); children - 53/1,326 (4.00%); 2) Not at risk - 1/4126 (0.75%); adults - 27/2,845 (0.95%); children - 4/1,281(0.31%); biopsy in EMA pos - Marsh 1- 0%; 2 - 30/116 (25.9%); 3a - 46/116 (39.7%); 3b - 24/116 (20.7%); 3c - 16/116 (13.8%); HLA DQ2-76/98 (78%); DQ8 - 16/98 (16%); DQ2 and 8 - 6/98 (6%); all EMA pos were also tTG pos			

Evidence Table 7 (cont'd): Prevalence of CD in relatives of patients with CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Hill 2000 USA Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA-EMA - IF on ME, methodology n/a; IgA and IgG-AGA - ELISA, methodology n/a **Biopsy criteria/description:** Marsh **Confirmatory test:** Biopsy **Checked IgA def.** yes	**Ethnicity:** n/a **Patient type/# screened:** 1,200 pediatric group of individuals at risk of CD; pts were assigned to one of 7 groups: 1) chronic diarrhea (n=182); 2) abdominal pain (n=316); 3) IDDM (n=81); 4) short stature (n=259); 5) failure to thrive (n=123); 6) miscellaneous (down's syndrome, thyroiditis, anemia, unexplained elevation of liver enzymes) (n=47); 7) asymptomatic relatives (n=192) **Demographics:** 1,200 pediatric group of individuals at risk of CD; age (y): mean n/a, range 6 mos - 20 y; % F n/a **Incidence:**	Prevalence of CD in pts at risk was 1 in 57 (21/1200); prevalence of test positivity: 2.8% (34/1200) was both EMA and AGA positive; 26 of pts (19 EMA positive) underwent biopsy and 21 were diagnosed with CD			15 pts out of 34 EMA positives refused biopsy; thus prevalence of biopsy proven CD in pts at risk was at least 1 in 57; if considered above mentioned 15 pts prevalence of CD could have been 1 in 33 (36/1200)

Evidence Table 7 (cont'd): Prevalence of CD in relatives of patients with CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Hogberg, 2003 Sweden Duplicate: no	**Publication type:** journal **Study design:** longitudinal follow-up, incidence & prevalence **Test/methodology:** IgA-AGA, ELISA cut off for a positive outcome 42.5 units; IgA-EMA, indirect IF using ME the antibody titre was defined as the highest serum dilution yielding positive fluorescence; IgA-TGA, commercially available ELISA, highest cut-off value for positive results >30 units. **Biopsy criteria/description:** revised ESPGAN. Grade 1 normal; Grade 2 mild; Grade 3 moderate, Grade 4 total VA **Confirmatory test:** Small bowel biopsy **Checked IgA def.** yes, 5 cases found	**Ethnicity:** n/a **Patient type/# screened:** 120 first degree relatives of CD pts **Demographics:** 56 parents (adults) mean age 53.6 range (43-78); 44 siblings (ped and adult) mean age 27.4 y age range (15-49); offspring 20 mean age 6.5 y age range (1-16). Gender not reported **Incidence:** Time period: 20 y follow-up study. Original study period Sept. 1975- Feb. 1981	10/120 (8.3%) prevalence, 2 were diagnosed in the original study 20 y prior, 8 new cases from the present follow-up study group. (biopsy confirmed); Serum results; IgA AGA 8/120 pos; IgA EMA 0.5% (6/120) pos; IgA TGA 3.8% (4/104) pos (3 pts with positive biopsy were not tested for IgA TGA)			IgA deficient pt, IgG antibodies positive. Biopsy findings were in serologically positive relatives IgA TGA was performed in sera of n=104 when access to assay became available 1 y later.

Evidence Table 7 (cont'd): Prevalence of CD in relatives of patients with CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Holm, 1993 Finland Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** biopsy **Biopsy criteria/description:** Some VA **Confirmatory test:** n/a **Checked IgA def.** n/a	**Ethnicity:** n/a **Patient type/# screened:** Relatives of celiac pts **Demographics:** n=121 first-degree relatives **Incidence:**	10.7%			

Evidence Table 7 (cont'd): Prevalence of CD in relatives of patients with CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Korponay-Szabo, 1998 Hungary Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional Prevalence **Test/methodology:** IgA EMA - IF using ME and human duodenum as substrate; serum total IgA, deficiency was defined as total serum IgA<0.1 g/L **Biopsy criteria/description:** Watson capsule; histological evaluation according to the grading of Fontaine and Navarro; ESPGAN criteria for CD **Confirmatory test:** biopsy; Watson-Crosby capsule; biopsy done in 77 out of 81 pts with positive IgA EMA **Checked IgA def.** yes; 2.1% (21/997) of all family members studied were IgA deficient; among CD diagnosed pts 10.8% (9/83) were IgA deficient	**Ethnicity:** n/a **Patient type/# screened:** 997 family members of 396 pts with CD screened for CD **Demographics:** 997 family members of 396 pts with CD screened for CD (521 parents, 368 siblings, 54 children and 54 second-degree relatives); age: mean/median, range n/a **Incidence:** # New cases: 75 new cases of CD; incidence of CD was 7.6% (75/989)	Prevalence of CD in the whole screened population was 8.3% (83/997); prevalence for first-degree relatives in total: 8.6% (80/943)	parents: 4.2% (22/521)	siblings: 13.8% (51/368)	Screening revealed 75 new CD cases (in 71 IgA EMA were positive and in 4 - negative); 8 pts had been previously diagnosed with CD and were on a GFD and thus EMA negative; the total number of CD in the family members was 83; in 55 families two, in 10 families 3, in 1 family 4 and in 1 more family 6 members were affected by CD
			Group 4	**Group 5**		
			children: 12.9% (7/54);	second-degree relatives: 5.6% (3/54)		

Evidence Table 7 (cont'd): Prevalence of CD in relatives of patients with CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Kotze, 2001 Brazil Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** EMA **Biopsy criteria/description:** n/a **Confirmatory test:** n/a **Checked IgA def.** n/a	**Ethnicity:** n/a **Patient type/# screened:** Relatives of celiac pts **Demographics:** n=115 first-degree relatives **Incidence:**	3.5%			Negative serology; EMA titre =1/5

Evidence Table 7 (cont'd): Prevalence of CD in relatives of patients with CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Mustalahti, 2002 Finland Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional Prevalence **Test/methodology:** IgA-EMA- indirect IF on a HU, serum dilution of at least 1:5 was considered positive; IgA and IgG AGA - ELISA, level of AGA was considered positive when mean+-2 SD of healthy controls; IgA-tTG-ELISA, values at least 20 AU were considered positive **Biopsy criteria/description:** Small-bowel biopsies with pediatric or adult Watson capsule or forceps from the distal duodenum **Confirmatory test:** Biopsy; DQ2 and DQ8 typing on PCR **Checked IgA def.** n/a	**Ethnicity:** n/a **Patient type/# screened:** 466 healthy first-degree family members of CD pts **Demographics:** 466 healthy first-degree family members of CD pts; age (y): median 41, range 2-90 **Incidence:**	Prevalence of CD in healthy, first-degree relatives of CD pts was 6.2% (29/466); as for test positivity: 72/466 pts (44 EMA pos 9.4% and 48 IgA pos 10.3%) were positive for EMA and IgA; IgA-tTG was positive in 12.9% (60/466) pts; IgA-EMA detected 97% of CD (28/29) and IgA-AGA detected - 51% (15/29); all 44 IgA EMA and 19/28 AGA positive pts were positive for DQ2; all 29 newly diagnosed CD pts were DQ2 positive			15 pts out of 34 EMA positives refused biopsy; thus prevalence of biopsy proven CD in pts at risk was at least 1 in 57; if considered above mentioned 15 pts prevalence of CD could have been 1 in 33 (36/1,200)

Evidence Table 7 (cont'd): Prevalence of CD in relatives of patients with CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Pittschieler, 2003 Italy Duplicate: no	**Publication type:** journal **Study design:** Prospective prevalence **Test/methodology:** EMA, HUC examined by fluorescence. Absence of binding was considered a negative test; HLA typing was done exclusively once diagnosis of CD was confirmed= micro-lymphocytotoxic technique **Biopsy criteria/description:** Small intestinal biopsy of first jejunal loop. Ferraris Watson capsule. Normal values being less than 3.2 cells/mm γ/δ T-cell receptors by immunohistology **Confirmatory test:** biopsy, HLA typing **Checked IgA def.** yes, none found	**Ethnicity:** n/a **Patient type/# screened:** 92 first-degree relatives of CD pts. Yearly testing over 12 year period **Demographics:** 92 at risk (first-degree relatives; 18 offspring and 74 siblings) aged 2-18 y **Incidence:** Time period: 12 year time period January 1990-December 2001	6.5% (6/92) confirmed CD by both serology & biopsy within a few months of CD diagnosis in one of their relatives; 5 total VA and 1 partial VA; over 2-5 y a further 5.8% (5/86) confirmed positive with both HU EMA IgA and biopsy 1 partial VA and 4 total VA; combined prevalence=12% (11/92); 11/11 were carriers of HLA DQ2/ heterodimers			All 11 were clinically silent for CD

Evidence Table 7 (cont'd): Prevalence of CD in relatives of patients with CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Polvi, 1996 Finland Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional Prevalence **Test/methodology:** DQA1*0501 and DQB1*0201 **Biopsy criteria/description:** n/a **Confirmatory test:** n/a **Checked IgA def.** n/a	**Ethnicity:** n/a **Patient type/# screened:** 31 CD index pts; 14 silent CD pts; 29 healthy siblings of CD index pts; 32 controls; **Demographics:** n/a **Incidence:**	Prevalence of DQA1*0501 and DQB1*0201 positivity in CD index group: 100% (31/31) CD index pts were positive; relative risk for having at least two alleles 156 **Group 4** prevalence of DQA1*0501 + DQB1*0201 in cadaver organ donors: 28% (9/32) controls	Prevalence of DQA1*0501 and DQB1*0201 positivity in silent CD group: 100% (14/14) silent CDs	Prevalence of DQA1*0501 and DQB1*0201 positivity in siblings of index CD pts: 48% (14/29) healthy sibs	There was a very strong association of DRA1*0501 and DQB1*0201 alleles positivity with CD; the RR for an individual having at least 2 susceptibility alleles suffering from CD was as high as 156 ($p<0.001$); RR was also high (67; $p<0.001$) when the index cases were compared with their siblings. The etiologic fraction in both cases was 0.99

Evidence Table 7 (cont'd): Prevalence of CD in relatives of patients with CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Robinson, 1971 UK	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** small bowel biopsy, Crosby capsule **Biopsy criteria/description:** Other: normal, convoluted, and flat; either partial or subtotal VA represents confirmed CD **Confirmatory test:** biopsy **Checked IgA def.** n/a	**Ethnicity:** n/a **Patient type/# screened:** relatives of celiac pts **Demographics:** 1) n=68, first-degree relatives=parents & siblings; 2) n=164, second-degree relatives=uncles & aunts, 50.6% F; 3) n=238, third-degree relatives=cousins, 52.1% F **Incidence:**	First-degree: 4.4% (3/68)	Second-degree: 0% (0/164)	Third-degree: 0% (0/238)	

Evidence Table 7 (cont'd): Prevalence of CD in relatives of patients with CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Rolles, 1974 UK Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** biopsy **Biopsy criteria/description:** n/r **Confirmatory test:** n/a **Checked IgA def.** n/a	**Ethnicity:** n/a **Patient type/# screened:** Relatives of CD pts-CD child in family **Demographics:** n=72 first-degree relatives **Incidence:**	5.6%			

Evidence Table 7 (cont'd): Prevalence of CD in relatives of patients with CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Rostami, 2000 Netherlands	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** IgA-EMA- indirect IF on a primate ileum; min 1:5 and 1:100; IgA-AGA- ELISA, >25 AU/mL was considered positive; IgA by nephelometry **Biopsy criteria/description:** EPSGAN; Marsh 1992 **Confirmatory test:** biopsy in all pts with positivity of symptoms and/or serology tests **Checked IgA def.** yes; 3 individuals were found to have IgA deficiency	**Ethnicity:** n/a **Patient type/# screened:** 388 first-degree relatives of CD pts **Demographics:** age (y): mean 39, range 1-80; 60% F **Incidence:** # New cases: 17 new cases of CD; incidence of 5% (17/338)	Overall prevalence of CD in the first-degree relatives of CD pts was 11% (37/338); there were 17 new and 20 previously diagnosed CD cases; as for the prevalence of test positivity: 28% (96/338) were positive for clinical complaints + lab tests (4 pts) and serology screening; 17/96 (18%) biopsy positive; 6/17 Marsh IIIc; 5/17 Marsh IIIa; 6/17 strongly positive EMA and AGA			

Evidence Table 7 (cont'd): Prevalence of CD in relatives of patients with CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Stokes, 1976 UK Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional Prevalence **Test/methodology:** biopsy **Biopsy criteria/description:** biopsy result must be grade III to be considered confirmed CD: subtotal VA **Confirmatory test:** biopsy **Checked IgA def.** n/a; no serology done; not mentioned	**Ethnicity:** n/a **Patient type/# screened:** Relatives of CD pts **Demographics:** n=326; children and adult; other info n/a **Incidence:** # New cases: 17 new cases of CD; incidence of 5% (17/338)	Fathers: 10.3% (4/39)	Mothers: 2.3% (1/43)	Brothers: 7.4% (5/68)	
			Group 4	**Group 5**	**Group 6**	
			Sisters: 17.3% (13/75)	Sons: 8.3% (5/60)	Daughters: 17.1% (7/41)	
			Group 7			
			19.2% (35/182 total biopsied)			

Evidence Table 7 (cont'd): Prevalence of CD in relatives of patients with CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Tursi, 2003 Italy Duplicate: no	**Publication type:** journal **Study design:** Prevalence **Test/methodology:** IgA IgG AGA, ELISA lower limit of positivity of IgA 0.2 EU/mL and IgG 10.0 EU/mL; IgA EMA, indirect IF on ME; IgA tTG, ELISA using GP liver substrate lower limit of positivity of these antibodies was 7 UA/mL **Biopsy criteria/description:** Marsh criteria 6 biopsies from small bowel from the second part of duodenum. Marsh Type I -'infiltrative' lesions with >30 lymphocytes/100 epithelial cells; Type II-'infiltrative/hyperplastic' lesions; Type III-'partial (sub)total VA; partial VA Marsh IIIa); subtotal VA Marsh IIIb); and total VA as Marsh IIIc) **Confirmatory test:** Biopsy **Checked IgA def.** yes	**Ethnicity:** n/a **Patient type/# screened:** 111 first-degree relatives of pts with CD **Demographics:** at risk 111 first degree relatives 38 M, 73 F, mean age 28.7 y, range (10-65 y); 65.8% F **Incidence:** n/a	CD diagnosed in 49/11 screened relatives (44.14%) prevalence; prevalence AGA 36.73%; EMA 38.78% ; anti-tTG 44.89%			Prevalence of antibodies was higher in severe histological lesions (Marsh IIIb-c) than in not so severe lesions (Marsh I-IIIa). Noteworthy, prevalence of AGA was higher than that of EMA/anti-tTG in less severe histological lesions

Evidence Table 7 (cont'd): Prevalence of CD in relatives of patients with CD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Vitoria, 1994 Spain Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional Prevalence **Test/methodology:** IgA-AGA>0.085 AU in children, >0.128 AU in adults; IgG-AGA>0.45 AU in children, >0.317 AU in adults; EMA titre≥1:5 **Biopsy criteria/description:** ESPGAN **Confirmatory test:** biopsy **Checked IgA def.** n/a	**Ethnicity:** n/a **Patient type/# screened:** first degree relatives of celiac pts **Demographics:** n=642 (380 parents, 249 siblings, 13 offspring); ped and adult; mean age= 27 y; age range=1-64 y; 52.3% F **Incidence:**	Fathers: by IgA-AGA: 11.7% (21/180), by IgG-AGA: 13.9% (25/180), by EMA: 0% (0/180), by biopsy: 0.56% (1/180)	Mothers: by IgA-AGA: 9% (18/200), by IgG-AGA: 8.5% (17/200), by EMA: 2.5% (5/200), by biopsy: 2.5% (5/200))	Brothers: by IgA-AGA: 8.3% (10/120), by IgG-AGA: 9.2% (11/120), by EMA: 3.3% (4/120), by biopsy: 2.5% (3/120)	
			Group 4	**Group 5**		
			Sisters: by IgA-AGA: 12.4% (16/129), by IgG-AGA: 15.5% (20/129), by EMA: 5.4% (7/129), by biopsy: 6.2% (8/129)	Offspring: by IgA-AGA: 15.4% (2/13), by IgG-AGA: 23.1% (3/13), by EMA: 7.7% (1/13), by biopsy: 7.7% (1/13)		

Prevalence of CD in Associated Clinical Conditions—Anemia

Evidence Table 8. Prevalence of CD in patients with anemia

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Akerman, 1996 Israel Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** EGD/Biopsy **Biopsy criteria/description:** subtotal or greater VA **Confirmatory test:** n/a **Checked IgA def.** n/a	**Ethnicity:** n/a **Patient type/# screened:** out pts with IDA (50% symptomatic) **Demographics:** 93 pts; mostly adults although some teens **Incidence:**	By biopsy: 13			

Evidence Table 8 (cont'd): Prevalence of CD in patients with anemia

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Annibale, 2001 Italy Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** antral (n=3 per pt)/body(n=3)/duodenal(n=2) biopsies; assessment of gastritis according to Sydney system; March classification for CD; colonoscopy for suspicious lesions; refusal of colonoscopy led to double-contrast barium enema; NOTE: no serology done **Biopsy criteria/description:** n/a **Confirmatory test:** Biopsies **Checked IgA def.** no, not mentioned	**Ethnicity:** n/a **Patient type/# screened:** iron deficiency **Demographics:** 81 pts; adult; median age 54; range 23-87; 74% F **Incidence:**	By biopsy: 6% (4/71); 71 pts formed the final sample of completely examined subjects. Note: no serology done			The celiac pts are younger compare to the rest of the sample

Evidence Table 8 (cont'd): Prevalence of CD in patients with anemia

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Annibale, 2003 Italy Duplicate: no	**Publication type:** journal **Study design:** Prospective prevalence **Test/methodology:** IgA tTG, ELISA normal values were <7 UA/mL **Biopsy criteria/description:** Marsh **Confirmatory test:** Biopsy - antral, gastric body, and duodenal biopsy collected **Checked IgA def.** n/a	**Ethnicity:** n/a **Patient type/# screened:** IDA in premenopausal women **Demographics:** 59 premenopausal women age range 22-54 y with IDA Hb < 12g/dLF **Incidence:** Time period: March-July 2000	7/59 (11.9%) had positive tTG antibodies titre; biopsy-confirmed: 8.5% (5/59)			40/59 subjects tested positive for various tests including tTG for CD detection and progressed to have upper endoscopy with biopsy

Evidence Table 8 (cont'd): Prevalence of CD in patients with anemia

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Corazza, 1997 Republic of San Marino Duplicate: no	**Publication type:** journal **Study design:** Cross-sectional prevalence **Test/methodology:** indirect IF EMA titre >1:5; biopsy **Biopsy criteria/description:** n/a **Confirmatory test:** biopsy **Checked IgA def.** no, but mentioned as such that it could have caused some misclassification of pts, but the effect should be minimal given the powerful sensitivity and specificity of EMA test	**Ethnicity:** n/a **Patient type/# screened:** random sample stratified for age and sex **Demographics:** n=2,237; adult median age: 44 y; range: 20-87 y % F: 53.2 **Incidence:**	By both EMA and biopsy: 1 in 559 pts, or 1.79 per 1000 [0.18%]			

Evidence Table 8 (cont'd): Prevalence of CD in patients with anemia

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Dickey, 1997 UK Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** EMA; AGA; methodology and cut-off levels n/a **Biopsy criteria/description:** endoscopic biopsy; criteria n/a; finding of VA and IELs in duodenal biopsy **Confirmatory test:** Biopsy **Checked IgA def.** n/a	**Ethnicity:** n/a **Patient type/# screened:** 41 pts with IDA and no specific GI symptoms or evidence of a bleeding on FOBT or upper GI and colonic endoscopy screened for achlorhydric gastric atrophy and CD **Demographics:** 41 pts with IDA screened for achlorhydric gastric atrophy and CD; age (y): mean 59, range 15-84; 61% F **Incidence:**	prevalence of CD in IDA pts was 10% (4/41); EMA was positive in 3 (75%) of 4 these pts; prevalence of EMA and/or AGA being positive was 10% (4/41)			The celiac pts are younger compared to the rest of the sample

Evidence Table 8 (cont'd): Prevalence of CD in patients with anemia

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Howard, 2002 UK Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** IgA/IgG-AGA and EMA then biopsy **Biopsy criteria/description:** n/a **Confirmatory test:** n/a **Checked IgA def.** n/a	**Ethnicity:** n/a **Patient type/# screened:** IDA identified through lab **Demographics:** 258 adult pts with IDA, folate **Incidence:**	By first serology: 28 by biopsy: 12			24/28 biopsied

Evidence Table 8 (cont'd): Prevalence of CD in patients with anemia

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Kepczyk, 1995 USA Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** EGD/biopsy **Biopsy criteria/description:** VA, crypt hyperplasia, inflammatory infiltrate **Confirmatory test:** n/a **Checked IgA def.** n/a	**Ethnicity:** n/a **Patient type/# screened:** Mostly symptomatic out pts with IDA **Demographics:** 39 adult pts with IDA **Incidence:**	By biopsy: 4			

Evidence Table 8 (cont'd): Prevalence of CD in patients with anemia

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
McIntyre, 1993 UK Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** duodenal biopsy performed in 50 pts; upper GI endoscopy performed in 108 pts **Biopsy criteria/description:** n/a **Confirmatory test:** prevalence of biopsy proven CD was at least 6% (3/50) **Checked IgA def.** No serology tests done. Results based on clinical findings of upper & lower GI symptoms. Prevalence was calculated only in biopsy-performed group of pts that consisted of 50 individuals.	**Ethnicity:** n/a **Patient type/# screened:** 111 pts with IDA **Demographics:** 111 pts with IDA; age (y): mean 63+-17.3, range 20-86; 61.3% F **Incidence:**	Prevalence of biopsy proven CD was at least 6% (3/50)			The celiac pts are younger compare to the rest of the sample

Evidence Table 8 (cont'd): Prevalence of CD in patients with anemia

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Oxentenko, 2002 USA Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** endoscopic biopsy of second and third parts of duodenum **Biopsy criteria/description:** CD was defined as total or partial VA with IELs **Confirmatory test:** Biopsy **Checked IgA def.** NA	**Ethnicity:** n/a **Patient type/# screened:** 113 pts with IDA **Demographics:** age (y): mean 55.6+-15.3, median 54, range 20-86; 71.7% F **Incidence:**	Prevalence of CD was 15% (17/113); 10 of these 17 pts had positive endoscopic markers suggestive of CD; 8 pts with endoscopic markers present did not have CD on biopsy			Only biopsy/no serology tests performed

Evidence Table 8 (cont'd): Prevalence of CD in patients with anemia

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Ransford, 2002 UK Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** IgA-EMA-ME, titers> 1:5 was considered positive; IgA tTG- ELISA, cut-off n/a **Biopsy criteria/description:** revised ESPGAN; duodenal histologic changes were graded according to Marsh criteria **Confirmatory test:** Biopsy **Checked IgA def.** No	**Ethnicity:** n/a **Patient type/# screened:** 484 anemic pts; 498 age and sex matched controls **Demographics:** n/a **Incidence:**	Prevalence of newly diagnosed CD in anemic pts was at least 2.2% (11/484)prevalence of CD in "EMA positive pts" was 1 in 28; prevalence of CD in "definite celiac + ↑IELs alone+not biopsied group" was 1 in 30; prevalence of CD in "definite celiac and ↑IELs alone group" was 1 in 35; prevalence of CD in "definite celiac group" was 1 in 44	Prevalence of newly diagnosed CD in age & sex matched non-anemic pts was 0.2% (1/484); prevalence of CD in "EMA positive group" was 1 in 83; prevalence of CD in "definite celiac +↑IELs alone+ not biopsied group" was 1 in 100; prevalence of CD in "definite celiac & ↑IELs alone group" was 1 in 166; prevalence of CD in "definite celiac group" was 1 in 498		Prevalence of newly diagnosed CD in anemic pts was at least 2.2% (11/484) compared with 0.2% (1/484) that of non-anemic pts ($p<0.01$)

Evidence Table 8 (cont'd): Prevalence of CD in patients with anemia

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Unsworth, 2000 UK Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** plasma diluted 1:5 IgA EMA using HU as substrate. Seropositives tested for IgA and IgG AGA ELISA and tTG ELISA using GP liver tTG **Biopsy criteria/description:** n/a **Confirmatory test:** small bowel biopsy **Checked IgA def.** n/a	**Ethnicity:** n/a **Patient type/# screened:** 110,937 blood donors, 1,380 anaemic donors, 483 with haemaglobin(<11 g/dL for women; <13.5 g/dL for men) **Demographics:** 1,380 anemic adult blood donors 87% women (age range n/a) 483 anemic pts meeting criteria 84% women **Incidence:**	IgA EMA positive 32/483 (6.6%); 25/32 had small bowel biopsy (4 pts lost to follow-up and 3 refused further testing) 22/25 had positive small bowel biopsy(88%) Subsequent results 22/32 cases IgA AGA positive; 26/32 cases were either IgA or IgG AGA pos; 31/32 cases pos using ME as substrate rather than HU; 31/32 were IgA tTG antibody pos	Prevalence of newly diagnosed CD in age & sex matched non-anemic pts was 0.2% (1/484); prevalence of CD in "EMA positive group" was 1 in 83; prevalence of CD in "definite celiac +↑IELs alone+ not biopsied group" was 1 in 100; prevalence of CD in "definite celiac & ↑IELs alone group" was 1 in 166; prevalence of CD in "definite celiac group" was 1 in 498		Prevalence of newly diagnosed CD in anemic pts was at least 2.2% (11/484) compared with 0.2% (1/484) that of non-anemic pts (p<0.01)

Evidence Table 8 (cont'd): Prevalence of CD in patients with anemia

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
van Mook, 2001 Netherlands Duplicate: no	**Publication type:** journal **Study design:** retrospective prevalence **Test/methodology:** EGD; upper digestive tract endoscopy in 10/35; duodenal biopsies taken in 15/35 **Biopsy criteria/description:** Marsh **Confirmatory test:** biopsy **Checked IgA def.** no	**Ethnicity:** n/a **Patient type/# screened:** 35 pts with IDA, anaemia defined as Hb below 8.0 mmol/L in men or below 7.4 mmol/L in women. Iron deficiency defined as a serum ferritin level equal to or below 20 ug/L for men equal to or below 10 ug/L ; or serum iron concentration equal to or below 45 ug/dl with a transferrin saturation of 10% or less, or the absence of iron stores in bone marrow biopsy specimens. **Demographics:** 35 pts, 22 F (63%) and 13 M (37%), median age 71 y range (22-89 y) **Incidence:** n/a	2.9% (1/35) Marsh III(C) on both biopsy and endoscopy			

Prevalence of CD in Associated Clinical Conditions—Low Bone Mineral Density (BMD)

Evidence Table 9: Prevalence of CD in patients with low BMD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Gonzalez, 2002 Argentina Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** IgA and IgG-AGA, ELISA; cut-off levels: for IgA, 15 AU/mL; for IgG, 20 AU/mL; IgA-EMA, IF on ME; positive if fluorescence at 1:5 dilution; 1st level of screening: measuring of IgA and IgG-AGA; 2nd level of screening: measuring of IgA and IgG-EMA and total serum IgA if AGA positive; 3rd level of screening: biopsy in EMA positives **Biopsy criteria/description:** endoscopic biopsy; CD was diagnosed when mucosa showed VA, crypt hyperplasia and intraepithelial lymphocytic infiltration (>30%) **Confirmatory test:** EMA; biopsy **Checked IgA def.** yes; 2 controls were found to have very low IgA level	**Ethnicity:** n/a **Patient type/# screened:** 127 consecutive postmenopausal pts with verified osteoporosis screened for CD; 747 controls screened for CD taken from a population-based study aiming to determine the prevalence of CD in Argentina **Demographics:** n=127 postmenopausal pts with osteoporosis; age: mean 68 y, range 50-82 y; n=747 controls; age: mean 29 y, range 16-79 y **Incidence:** Time period: prevalence of CD in 127 postmenopausal pts with osteoporosis was 1/127, or 7.9x1000 (95% CI 0.2-43.1); as for test positivity: AGA was found in 8 of 127 pts on level 1; 1 of these 8 pts was EMA-positive on the 2nd level and eligible for biopsy which established a diagnosis of CD Control popn: estimated prevalence of CD in control women population was 6/747, or 8x1000 (95% CI 3.3-18.3); as for test positivity: AGA was found in 96 of 747 (12.8%) pts on level 1; 4 pts were EMA-positive and 2 other pts had very low serum IgA on the 2nd level and all 6 were eligible for biopsy which established a diagnosis of CD in all cases	Prevalence of CD in 127 postmenopausal pts with osteoporosis was 1/127, or 7.9x1000 (95% CI 0.2-43.1); as for test positivity: AGA was found in 8 of 127 pts on level 1; 1 of these 8 pts was EMA-positive on the 2nd level and eligible for biopsy which established a diagnosis of CD			Prevalence of CD in postmenopausal osteoporotic women was similar to that of the general population

Evidence Table 9 (cont'd): Prevalence of CD in patients with low BMD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Lindh, 1992 Sweden Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** IgA-AGA, micro ELISA method; cut-off point was selected to be a 2 SD above the mean in a healthy population of blood donors **Biopsy criteria/description:** endoscopic biopsy; methodology and criteria of CD diagnosis n/r **Confirmatory test:** endoscopic biopsy **Checked IgA def.** n/r	**Ethnicity:** n/a **Patient type/# screened:** 92 consecutive pts with idiopathic osteoporosis screened for CD **Demographics:** n=92 consecutive pts with idiopathic osteoporosis screened for CD; 91% F (mean age 66±12 y)/ 9% M (mean age 50±12 y); **Incidence:** prevalence of CD was 3% (3/92); IgA-AGA was positive in 11 of 92 pts and biopsy was performed in 6 pts	Prevalence of CD was 3% (3/92)			

Evidence Table 9 (cont'd): Prevalence of CD in patients with low BMD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Mather, 2001 Canada Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** IgA-EMA, IF of ME **Biopsy criteria/description:** increased number of IELs with associated subtotal or total VA **Confirmatory test:** biopsy **Checked IgA def.** yes	**Ethnicity:** n/a **Patient type/# screened:** 96 consecutive idiopathic low BMD pts **Demographics:** n=96: mean age 57 y; range 18-86 y; 81.3% (78) F, 18.7% M (18) **Incidence:** n/a	7 (7.3%) of 96 pts were EMA pos at titers of ≥1:10; all biopsies were negative; prevalence of 0%			

Evidence Table 9 (cont'd): Prevalence of CD in patients with low BMD

Author, Year, Location	Study Design	Patient Characteristics	Prevalence			Comments
			Group 1	Group 2	Group 3	
Nuti, 2001 Italy Duplicate: no	**Publication type:** journal **Study design:** cross-sectional prevalence **Test/methodology:** IgA-AGA, ELISA cut-off level of 10 U/mL-1 **Biopsy criteria/description:** intestinal biopsy criteria n/r **Confirmatory test:** TG-ab-ELISA with cut-off 22 AU; intestinal biopsy **Checked IgA def.** no	**Ethnicity:** n/a **Patient type/# screened:** 255 females with osteoporosis **Demographics:** mean age 66.6 y, range 36-65 y **Incidence:** n/a	53/255 pos IgG-AGA; 24/53 pos TG-ab (9.4%); intestinal biopsy in 10/24 resulted in 6 confirmed CDs			

Celiac 3: Risk of Lymphoma in CD

Evidence Table 10: Risk of lymphoma in patients with CD

Author, year, country	Methods	Participants	Outcomes	Measures of risk (lymphoma)	Measures of risk (mortality)
Askling, 2002, Sweden Other reports of same cohort: Peters et al., Arch Int Med 2003;163:1566	Retrospective Cohort Study Study Dates: 1964-1994 **Group selection** Pts with CD: All individuals discharged at least once with a Dx CD (ICD 7-9) Controls: (5-y)Age/sex and (1 y) calendar period matched cancer incidence rate and mortality rate for the entire Swedish population **Institution:** All hospitals in Sweden (Swedish input register) **Ascertainment of outcome** Cancer register, register of causes of death, population register, register of population changes. Pathology report from Cancer Registry for lymphomas Dx'ed 1990 or after **Blinding** n/r **Follow-up** See ascertainment of outcome. 3.4% excluded with incomplete/non matching ID numbers	**CD pts**: n = 11,019 Mean age at CD Dx: 17.4 (range 0->70) Mean follow-up: 9.8 y (range 0-32) 97236 PYs Proportion of females: 59% Proportion on GFD: n/r % refractory CD: n/r Clinical presentation: symptomatic (admission to hospital)	**Lymphoma** # lymphomas 38 Lymphoma types: NHLs **Dead patients** Mean age at death: 68.6 Time from CD Dx: n/r **Death from NHL** #Ls in celiacs: 33 # expected lymphomas:2.9 **Death from all causes** # deaths 828 # expected 419.3 **Exclusions** Were lymphomas occurring prior to CD Dx included? NO Were lymphomas occurring within 12 months of CD Dx included? NO Were incidental lymphomas found at autopsy included? NO	**SIR NHL** 6.3 95% CI 4.2-125 **SIR 1-4 y Dx**: 9.7 95% CI 6.3-14 **SIR >5 y Dx**: 3.8 95% CI 2.2-6 **SIR age 0-59:** 6.0 95% CI 3.7-9.5 **SIR age >60:** 5.8 95% CI 3.7-8.5 **SIR childhood Dx (age 0-19):** 1.9 95% CI 0.4-5.5 **SIR adult Dx (age 20-59):** 7.7 95% CI 4.9-12 **SIR late Dx (age >60):** 6.3 95% CI 3.8-9.8 SIR 1970-79: 12 (3.8-28) SIR 1980-89: 8.5 (5.5-13) SIR 1990-95: 3.4 (1.9-5.7) P (linear trend) 1964-95 0.025	**SMR all causes** 2 95% CI 1.8-2.1 **SMR 1-4 y Dx:** 2.2 (1.9-2.4) **SMR 5-9 y Dx:** 2.0 (1.8-2.2) **SMR >10 y Dx:** 1.7 (1.5-2.0) P(trend) 0.12 **SMR from NHL** 11.4 (7.8-16)

Evidence Table 10 (cont'd): Risk of lymphoma in patients with CD

Author, year, country	Methods	Participants	Outcomes	Measures of risk (lymphoma)	Measures of risk (mortality)
Collin, 1996, Finland Other reports of same cohort: Collin et al. Gut 1994;35:1215	Prospective cohort study (Collin et al. Gut 1996;38:528) Study dates: 1970-1993 Case-control study (Collin et al. Gut 1994;35:1215) **Group selection** Pts with CD: All consecutive biopsy-proven CD (ESPGAN criteria) Controls: age/sex matched for Finnish population (database not stated) Controls(2): age/sex and year matched outputs for upper endoscopy **Institution:** single tertiary care institution **Ascertainment of outcome** Finnish cancer registry, Statistics Finland **Blinding** n/r **Follow-up** See ascertainment of outcome. 290 pts available for biopsy 6-12 mos post Dx	**CD pts**: n = 383 Mean age at CD Dx: 41.8 (range 16-78) Mean follow-up: 8.1 y 3107 PYs Mean follow-up (case-control) 3.1 (range 0.5-11) Proportion of females: 73% Proportion on GFD: Strict GFD 75% Partial GFD 8% Normal diet 6% Unknown 11% Compliance monitoring: control bx and dietary assessment 6-12 mos after Dx Clinical presentation: 82% symptomatic 18% serology Dx	**Lymphoma** # lymphomas 1 Lymphoma types: # expected lymphomas 0.4 **Death from all causes** # deaths: 31 (8.1%) # expected: see graph p1217 of Collin et al. Gut 1994;35:1215 **Exclusions** Were lymphomas occurring prior to CD Dx? NO Were lymphomas occurring within 12 months of CD Dx? YES Were incidental lymphomas found at autopsy included? PROBABLY NOT	**SIR NH** 2.66 95% CI 0.07-14.8	10 and 15 y survival rates of pts with CD did not differ significantly from the rates seen in general population

Evidence Table 10 (cont'd): Risk of lymphoma in patients with CD

Author, year, country	Methods	Participants	Outcomes	Measures of risk (lymphoma)	Measures of risk (mortality)
Corrao, 2001 Italy Other reports of same cohort: none	Prospective Cohort Study Dates: 1962-1994 **Group selection** Pts with CD: All consecutive biopsy-proven CD (ESPGAN criteria) Controls: (5-y) age/sex and calendar year matched national life tables and regional mortality rates **Institution:** 11 GI units throughout Italy selected for quality of record keeping; mostly tertiary **Ascertainment of outcome** phone interview (pts, relatives); death certificates, Italian National Institute of Statistics **Blinding** n/r **Follow-up** see ascertainment of outcome. 8 pts or their relative not tracked down; excluded. 50 pts lost to follow-up	**CD pts**: n = 1,072 Mean age at CD Dx: 35.7 (range 18->50) Median Dx delay 17 months Mean follow-up: 6.0 y 6444 PYs Proportion of females: 76% Proportion on GFD: 59% Strict GFD: 59% Not likely: 15% Unknown: 27% Compliance monitoring: control biopsy and phone interview 1999 % refractory CD: none Clinical presentation: 55% symptomatic 39% mild symptoms 6% serology Dx	**Death from NHL** # lymphomas: 16 lymphoma types: # expected lymphomas 0.2 **Death from all causes** # deaths 53 # expected 25.9 **Exclusions** Were lymphomas occurring prior to CD Dx included? NO Were lymphomas occurring within 12 months of CD Dx included? YES Were incidental lymphomas found at autopsy included? PROBABLY NOT	**SIR NHL**	**SMR from NHL:** 69.3 95% CI 40.7-112.6 **SMR all causes:** 2.0 95% CI 1.5-2.7 SMR 0-3 y Dx: 0.98 (0.97-0.98) SMR >3 y Dx: 0.98 (0.96-0.99) SMR age 18-29 at Dx: 2.5 (0.5-7.3) SMR age 30-49 at Dx: 2.4 (1.3-4.0) SMR age >50 at Dx: 1.9 (1.3-2.6) SMR 1962-74: 3.2 (1.4-6.3) SMR 1975-84: 1.8 (1.0-3.1) SMR >1985: 2.0 (1.3-2.8) SMR Dx delay <1 y: 1.5 (0.9-2.3) SMR Dx delay 1-10 y: 2.6 (1.6-4.1) SMR Dx delay >10 y: 3.8 (2.2-6.4) SMR symptoms: 2.5(1.8-3.4) SMR mild symptoms: 1.1 (0.5-2.2) SMR asymptomatic: 1.2 (0.1-7.0) SMR GFD: 0.5 (0.2-1.1) SMR unlikely: 6.0 (4.0-8.8) SMR uncertain: 2.0 (1.2-3.0)

Evidence Table 10 (cont'd): Risk of lymphoma in patients with CD

Author, year, country	Methods	Participants	Outcomes	Measures of risk (lymphoma)	Measures of risk (mortality)
Cottone, 1999, Sicily Other reports of same cohort: none	Retrospective cohort study Study dates: 1980-1997 **Group selection** CD pts: All biopsy-proven CD Controls: Age and sex-matched reported mortality from same period and region; cancer registry of the city of Ragusa in Sicily 1983-1987 **Institution:** Single institution; referral basis for all of Sicily **Ascertainment of outcome** Hospital medical records were reviewed; pathology specimen reviewed **Blinding** n/r **Follow up** 5% incomplete records	**CD pts**: n = 228 Mean age at CD Dx: 34.7 adult Dx: 98% Range of age: n/r Mean follow-up: 73 mos (range 1-204) #PYs: n/r Proportion of females: 76% Proportion on GFD: 100% Compliance monitoring: serial EMA % refractory CD: n/r Clinical presentation: Anemia 60% Malabsorbtion 20% Other 10% Asymptomatic 10% % inputs: 29% Marsh grade: n/r	**Lymphoma** #NHLs in celiacs: 7 Lymphoma types: ETCL (4), B-cell (2), other NHL(1) # expected lymphomas: 1.824 % silent: n/r % Dx'ed during childhood : 0 **Lymphoma patients** Mean age: 59.4 Time from CD Dx: 78 mos % compliant to diet: 100 incidence NHL: 3% expected incidence: 0.8 **Death from lymphoma** # death from lymphoma in celiacs: 5 # expected death from lymphomas: n/r **Death from all causes** # death CD: 12 # expected deaths: 3.12 # deaths within 4 y of Dx: 8 # expected: 1.48 **Exclusions** Were lymphomas occurring prior to Dx of CD included? NO Were lymphomas occurring within 6 months of CD Dx included? NO Were incidental lymphomas found at autopsy included? No such cases	SIR NHL **3.75** P<0.01	**SMR from NHL** n/r **SMR all causes** 3.8 95% CI 1.9-6.7 **SMR 4 y from:** 5.8 **95% CI 2.5-11.5**

Evidence Table 10 (cont'd): Risk of lymphoma in patients with CD

Author, year, country	Methods	Participants	Outcomes	Measures of risk (lymphoma)	Measures of risk (mortality)
Delco, 1999 US Other reports of same cohort: none	Case-control study Study Dates: 1986-1995 **Group selection** Pts with CD: all consecutive pts discharged with CD Dx (ICD 579.0) Controls: 5 randomly selected controls from same annual data file per case **Institution:** All US VA hospitals **Ascertainment of outcome** Pt Treatment File of the VA. Validity of records checked with 7 pt files **Blinding** n/r **Follow-up** See ascertainment of outcome.	**CD pts**: n = 458 **Controls:** n=2692 Mean age: celiacs: 63.8 +/- 12.4 controls: 59.7 +/- 14.8 p< 0.0001 Proportion of females: celiacs 4% controls 2% p=0.105 Race: celiacs: 93% whites controls: 74% p<0.0001 Proportion on GFD: n/r Compliance monitoring: n/a % refractory celiac: n/r Clinical presentation: 100% symptomatic (all discharged Dx)	**Lymphoma** # lymphomas n/r Lymphoma types: n/r # expected lymphomas n/r **Death from all causes** # deaths: n/r **Exclusions** Were lymphomas occurring prior to CD Dx included? NO Were lymphomas occurring within 12 mos of CD Dx included? LIKELY Were incidental lymphomas found at autopsy included? LIKELY Pts with repeated admission within 1 y were excluded	**OR NHL** 4.53 95% CI 2.01-10.23	

Evidence Table 10 (cont'd): Risk of lymphoma in patients with CD

Author, year, country	Methods	Participants	Outcomes	Measures of risk (lymphoma)	Measures of risk (mortality)
Green, 2003, US (New York) Other reports of same cohort: none	Prospective Cohort Study Dates: 1981-2000 **Group selection** Pts with CD: All consecutive biopsy-proven CD (ESPGAN criteria) Controls: (5-y) age/sex and calendar year matched site-specific incidence rates from the National Cancer Institute's Surveillance, Epidemiology and End Results Program for whites **Institution:** single tertiary care institution **Ascertainment of outcome** pt interview, review of pathology records **Blinding** n/r **Follow-up** pts not followed-up excluded; # n/r	**CD pts**: n = 381 Mean age at CD Dx: 44 +/- 18 y Duration of CD symptoms prior to Dx: 5y +/- 8 Mean follow-up: 6 +/- 11 y 1977 PYs Proportion of females: 64% Proportion on GFD: 100% of NHLs after Dx Compliance monitoring: clinical interview, yearly AGA and EMA after 1993 % refractory celiac: n/r Clinical presentation: n/r	**NHL 1 mos after CD Dx** # lymphomas 5 # expected lymphomas n/r Age at Dx cancer 62+/- 8 y Mean duration from celiac Dx 5+/- 4 y Mean follow-up 16+/- 17 y **NHL before/1 mos after CD Dx*** # NHLs: 4 # expected: 0.7 **NHL any time before/after Dx celiac*** # NHLs: 9 # expected: 1 Lymphoma types: B-cell(3), T-cell (4 – ETCL 3), large cell (2) **Exclusions** Were lymphomas occurring prior to Dx of celiac included? YES* Were lymphomas occurring within 1 months of celiac Dx included? YES* Were incidental lymphomas found at autopsy included? n/r *separate analysis	**SIR NHL** 6.2 95% CI 2.9-14 **SIR NHL before/1 mos after Dx celiac:*** 5.3 (2.3-13) incidence NHL 135/100 000 PYs expected 14.8/100 000 PYs **SIR NHL any time before/after Dx celiac:*** 9.1 (4.7-13)	

Evidence Table 10 (cont'd): Risk of lymphoma in patients with CD

Author, year, country	Methods	Participants	Outcomes	Measures of risk
Holmes, 1989, England Other reports of same cohort: Holmes et al. Gut 1976;17:612; Harris et al, Am J Med 1967;42:899	Prospective cohort study Study Dates: 1941-1985 **Group selection** Pts with CD: All biopsy-proven CD Controls: Age/sex matched incidence for 2 calendar periods standardized to ICD 8,200 and 202, in West Midlands region **Institution:** Single institution **Ascertainment of outcome** Direct pt interview, case notes, GP, Birmingham and West Midlands cancer registry, autopsy results, death certificates, pathology specimen reviewed **Blinding** n/r **Follow-up** Use of the family Practitioner Committee records and National Health Service Central Register at Southport; 2% drop outs	**CD pts**: n = 210 Mean age at CD Dx: n/r Adult Dx: 80%* (*from Harris et al, Am J Med 1967;42:899) Mean follow-up: 17.4 PYs for men, 19.4 PYs for women; minimum 13 y Proportion of females: 55.2% Proportion on GFD: 51% strict GFD 51% reduced gluten 27% normal diet 22% Compliance monitoring: direct interview; repeat biopsy in 86 pts % refractory CD: 17 poor response to GFD Clinical presentation: n/r	**Lymphoma** #NHLs in celiacs: 9 lymphoma types: n/r # expected lymphomas: 0.21 **Lymphoma patients** Mean age: n/r Time from CD Dx: n/r % compliant to diet: 2 NHL in strict GFD 7 NHL in gluten diet **Death from lymphoma** Not calculated **Death from all causes** Not calculated **Exclusions** Were lymphomas occurring prior to Dx of CD included? NO Were lymphomas occurring within 12 months of CD Dx included? NO Were incidental lymphomas found at autopsy included? No such cases	SIR NHL **42.7** 95% CI 19.6-81.4 **SMR from NHL** n/r **SMR all causes** Not calculated **SMR 4 y from Dx** n/r **SIR NHL** **Strict gluten-free diet** 44.4 **Gluten diet** 100

Evidence Table 10 (cont'd): Risk of lymphoma in patients with CD

Study	Methods	Participants	Outcomes	Measures of risk
Logan, 1989, Scotland Other reports of same cohort: Celiac 2; Logan et al. Gastroenterol 1986;90:334 Celiac 2; Logan et al. BMJ (Clinical research ed) 1983;286:95	Prospective cohort study Study dates: 1979-1986 **Group selection** Pts with CD: All biopsy-proven CD entered in register for Edinburgh and the Lothians Controls: Age/sex matched mortality for Scotland and corresponding person-years over 5 calendar periods standardized to ICD 8 200-203, in Scotland **Institution:** All hospitals in Edinburgh and the Lothian region, postal survey of all GPs, Scottish in-pt Statistics 1961-1977, local branch of Celiac Society **Ascertainment of outcome** Death certificates **Blinding** n/r **Follow-up** National Health Service Central Record, death certificates, Scottish national death records. 6% lost to follow-up	**CD pts**: n = 653 Mean age at CD Dx: n/r Mean follow-up: 13.5 y 8823 PYs Proportion of females: 60% Proportion on GFD: n/r % refractory CD: n/r Clinical presentation: n/r	**Lymphoma** # lymphomas: n/r Lymphoma types: most lymphosarcomas or reticulum-cell sarcomas, 2 Hodgkins **Dead patients** Mean age at death: 60.8 Time from CD Dx: n/r **Death from lymphoma** #s in celiacs: 17 #expected lymphomas: 0.55 **Death from all causes** # deaths 115 # expected 61.8 **Exclusions** Were lymphomas occurring prior to CD Dx included? NO Were lymphomas occurring within 12 months of CD Dx included? YES Were incidental lymphomas found at autopsy included? NO	**SIR NHL:** n/r **SMR from lymphoma** 31 p<0.001 **SMR(L) 0-1 y Dx**: 108 **SMR(L) 2-4 y Dx**: 9 **SMR (L) 5-49 y Dx**: 22 **SMR all causes** 1.9 95% CI 1.5-2.2 **SMR <1 y from Dx:** 4.1 **SMR 1-2 y from Dx:** 3.2 **SMR 3-4 y from Dx:** 2.2 **SMR 5-9 y from Dx:** 1.5 **SMR 10-14 y from Dx:** 1.5 **SMR childhood Dx:** 1.4 95% CI 0.4-3.7 **SMR adult Dx:** 1.9 95% CI 1.5-2.3 **SMR late Dx:*** 1.7 95% CI 0.02-4.8 *obvious CD symptoms during childhood

Evidence Table 10 (cont'd): Risk of lymphoma in patients with CD

Author, year, country	Methods	Participants	Outcomes	Measures of risk (lymphoma)	Measures of risk (mortality)
Selby, 1979 Australia Other reports of same cohort: none	Retrospective cohort study dates: 1959-1978 **Group selection** Pts with CD: All consecutive biopsy-proven CD (ESPGAN criteria) Controls: ?age/sex and period matched incidence New South Wales Central cancer Registry **Institution:** Single tertiary care institution **Ascertainment of outcome** direct call or via medical officer **Blinding** n/r **Follow-up** Direct call or via medical officer. 21% lost to follow-up	**CD pts**: n = 93 Mean age at diagnosis celiac: 40 (range 14-70) Duration of Sx of celiac prior to Dx: 3yrs (range 2 wks to 26 y) Mean follow-up: 6 y (max 9 y) Proportion of females: 67% Proportion on GFD: 100% of NHLs after Dx Compliance monitoring: n/r % refractory CD: none of the lymphoma pts Clinical presentation: 100% malabsorption: 18% malabsorption during childhood	**Lymphomas** # lymphomas 4 # expected lymphomas 0.081 Age at cancer Dx: 47.5 Mean duration from CD symptoms: 11 y (range 2-26 y) Mean follow-up: n/r Lymphoma types: ETCL (2), lymphosarcoma (1), histiocytic medullary reticulosis (1) **Exclusions** Were lymphomas occurring prior to Dx of celiac included? NO Were lymphomas occurring concomitantly with celiac Dx included? NO Were incidental lymphomas found at autopsy included? n/r	**SIR NHL 4.94** p< 0.0005 symptom duration (cancer vs none): not significant age Dx (cancer vs none): not significant	

Celiac 4: Consequences of Testing for CD

Evidence Table 11: Consequences of testing for CD

Study, Year Country	Methods	Participants	Outcomes	Results	Limitations
Addolorato, 2001 Italy	Study type - 1996-1998 Population-group selection Celiacs – newly diagnosed CD selected from outpatient clinic of 234 adult CD Controls - healthy asymptomatic controls - matched for age, sex and SES Loss to follow-up -8 Setting - Tertiary	Celiac - n=43 enrolled Controls - n=59 Proportion F – 60% Mean follow-up - 1 y Mean age: 29.8 ± 7.4	Before/after GFD Anxiety State and Trait Anxiety Inventory test Depression SDS self rating depression scale	Before – CD 71.4% showed high levels of state anxiety, 25.7% showed anxiety as a trait and 57.1% positive for depression compared to 23.7%, 15.2% and 9.6% of controls Post GFD – 25.7% still affected by state anxiety, 17% trait anxiety, and 45.7% depressed Significant decrease in state anxiety ($p<0.001$) No significant changes in trait anxiety or depression - anxiety in CD predominantly reactive	Small sample, loss to 8 pts to follow-up Relatively short follow-up

Evidence Table 11 (cont'd): Consequences of testing for CD

Author, Year Country	Methods	Participants	Outcomes	Results	Limitations
Amin, 2002 UK	Study type - case controlled longitudinal 1994-1998 Population-group selection Celiacs – 11 pts, EMA + and biopsy diagnosed CD out of 230 pts screened from diabetic clinic Controls – 22 matched for age, sex and duration of diabetes, with negative serology Institution – diabetic clinic – tertiary care hospital	Celiacs: n=11 (6 had repeat small bowel Bx on GFD) Controls: n=22 Age: 8.1 (1.2-16.1) cases, 7.4 (1.3-14.8) controls Proportion of females: 54.5% Duration of type 1 diabetes: 4.2 y Mean follow-up - 4 y BMISDS -1.2 ± 0.1 (SEM) vs -0.1 ± 0.1 WtSDS - 0.7 vs 0.5 HbgA1c – 8.3 cases 9.8 controls Insulin units – 0.8	BMI Hgba1c Insulin regimens	Cases: 1.1 ± 0.1 Controls: 1 ± 0.1 Cases: 8.3 ± 0.2 Controls: 10 ± 0.2 Insulin regimens increased but did not differ significantly between Insulin units 1.0 All reverted to antibody negative	Small sample Selection of controls

Evidence Table 11 (cont'd): Consequences of testing for CD

Study, Year Country	Methods	Participants	Outcomes	Results	Results after treatment
Annibale, 2001 Italy	Study type - prospective 1994- May 1997 Population-group selection Celiacs- 190 consecutive adults with iron deficiency anemia, 26 pts were diagnosed with CD after duodenal biopsy	Celiac n= 26 of 190 pts with IDA Proportion F - 92% Mean age - 31.3 Mean follow-up – 24 mos 77% had total VA, 23% had subtotal atrophy 11 did not have symptoms apart from anemia 42% BMI	Iron Deficiency (Hgb<14 for men and 12 for women, MCV <80, low serum iron and low serum ferritin Ferritin Nutritional parameters Repeat endoscopy – 6 mos	6 mos 77.8% recovered IDA, but only 5 of 18 (27.8%) developed normal ferritin levels 12 mos – 94.4% recovered from anemia and 50% from iron deficiency – all pts had normal RDW 24 mos, 55% recovered from iron deficiency	Recovery from IDA occurs within the initial 6-12 mos but only 50% recover from iron deficiency In subgroup of pts (n=7) who had repeat biopsies at 6 and 12 mos – inverse correlation between histological grade and increase in Hgb

Evidence Table 11 (cont'd): Consequences of testing for CD

Author, Year Country	Methods	Participants	Outcomes	Results		Limitations
Arato, 2002 Hungary	Study type – longitudinal – own controls Population-group selection Celiacs selected from 205 children with type 1 diabetes randomly selected, screened with EMA and then confirmed with biopsy Controls – no controls	Celiacs: n=17 % Females -59% Mean follow-up – 3 mos BMI 14.2 vs 16.3 for controls 11 had silent CD, 6 had mild GI symptoms	BMI HbA1c Insulin requirements	Before 16.8 <0.05 7.82 NS 0.48	After GFD 0.64 <0.05	Short follow-up small sample size No controls

Evidence Table 11 (cont'd): Consequences of testing for CD

Author, Year Country	Methods	Participants	Outcomes	Results	Limitations
Bardella, 1985 Italy	Study type - prospective Population-group selection Celiacs- adults with biopsy confirmed disease (Grade II or IV – Scott/Losowsky) and malabsorption Controls ---none	Celiacs - n=26 Proportion F - 81% Mean age - 42.2 Mean follow-up– 55.4 mos on GFD (range 13-137 mos)	Clinical Symptoms CBC, biochemical parameters Repeat biopsy (17/26)	8 pts good health and normal blood tests and 18 had some clinical or biochemical abnormalities, 4 pts had recurrent abd pain, meteorism and diarrhea, 2 isolated episodes of diarrhea and 5 meteorism alone present, 11 pts had one more of anemia, moderate abnormal of calcium, alkaline phosphatase, phosphorus 13 showed grade II, and 4 grade III – all improved by 1 or 2 grades but none returned to normal	

Evidence Table 11 (cont'd): Consequences of testing for CD

Author, Year Country	Methods	Participants	Outcomes	Results	Limitations
Bardella, 2000 Italy	Study type - case control 1962-1994 Population-group selection Celiacs not clear how selected 33.4% asymptomatic Controls – 2/pt matched for age and sex	Cases 71 out of 212 43 diagnosed as children, 28 as adults Control - n=142 Proportion F - 72%	Weight BMI Fat mass Lean mass	Lower than controls 55.5 vs 58.7 kg (p=0.004) 20.9 vs 22.4, p=0.03 22.9 vs 27.5, p<0.05 38.8 vs 40.5 (p<0.03)	

Evidence Table 11 (cont'd): Consequences of testing for CD

Author, Year Country	Methods	Participants	Outcomes	Results	Limitations
Barera, 2000 Italy	Study type - case control prospective Population-group selection Celiacs – biopsy confirmed CD, 4 classic symptoms, remainder atypical Controls - age and sex matched healthy controls	Celiac cases - n=29 Control - n=29 Age - 9.54 + 3.42 Proportion F: 51.7% Mean follow-up – 1.20 ± 0.15 Compliance – EMA ab	Body composition - DEXA Weight (kg) Height (cm) BMI (kg/m2) Lean Mass	At baseline, weight, fat mass, BMD, lean mass of limbs all lower in the pts versus controls, after 1 y no significant differences in body composition between pts and controls Untreated / Treated 30.3 + 11.5kg / 34.7 + 12.3 134.9 + 19 / 140.9 + 18.4 16.7 +4.5 / 17.3 + 3.1 166.8 / 179.9 +42	

Evidence Table 11 (cont'd): Consequences of testing for CD

Author, Year Country	Methods	Participants	Outcomes	Results	Limitations
Boersma, Netherlands 2002	Study type - 1994-1995 prospective study Population-group selection Celiacs-children with newly diagnosed celiac (symptoms and biopsy confirmed) CD patients acted as own controls	Celiacs - 28 Proportion F - 68% Mean follow-up - 3 y BMI	BMI BMI-SDS Height SDS	BMI - SDS for CD improved significantly after a GFD over 1st half year. (P<0.001) Height for SDS for CD showed a continuous significant increment over the first 3 y of GFD (p<0.001)	With institution of GFD also noted increased sensitivity to GH, and levels of IGF-1, IGF-2, and IGFBP-3 rise

Evidence Table 11 (cont'd): Consequences of testing for CD

Study, Year Country	Methods	Participants	Outcomes	Results	Risk estimates
Ciacci, 1996 Italy	Study type - case control Before after Population-group selection Celiacs Untreated CD had a least one pregnancy when symptoms leading to diagnostic workup for CD were present Controls (treated) had at least one pregnancy after 1 y of GFD Setting- Tertiary clinic	Celiac - n=94 Controls - n= 31 12 separate women acted as own control for before after study to assess impact of the GFD (not included in other analysis). Mean age at diagnosis of untreated older than treated (37.3 vs 22.4) Mean follow-up of treated celiacs 9.2 ± 1.4 Mean weight of treated 50.6 vs 50 Mean BMI 20.1 vs 19.4 Clinical symptoms of untreated group 33% did not have diarrhea, 24.5% did not have anemia	Number of pregnancies/woman Number of abortions/woman Abortion to pregnancy ratio Low birth weight baby to pregnancy ratio	2.72 in untreated vs 1.36 in controls 0.489 ± 0.08 untreated vs 0.032 ± 0.032 treated (17.8% vs 2.4%) 0.153 ± 0.027 vs 0.024 ± 0.024 0.126 ± 0.037 vs 0.024 ± 0.024 (prevalence 12.7% vs 2.4% in treated) *controls not age matched – cases significantly older	RR of abortion 8.9 (95% CI 1.19, 31.9) RR of low birth weight baby 5.84 (95% CI 1.07, 31.9) 12.7% vs2.4% Did not have external control group or control for confounders.

Evidence Table 11 (cont'd): Consequences of testing for CD

Author, Year Country	Methods	Participants	Outcomes	Results	Limitations
Fabiani, 1996 Italy	Study type - longitudinal Population-group selection Celiacs – biopsy proven CD from screening of adolescents (n = 6,315) Controls - none Loss to follow-up – 5	Celiacs – n=28 adolescents (age 11-14) Proportion F -74% Mean F/U 23 ± 7 mos	Compliance	52% on strict GFD 47% partial adherence weight gain 12/23 (52%), height gain (11/25)	

Evidence Table 11 (cont'd): Consequences of testing for CD

Author, Year Country	Methods	Participants	Outcomes	Results	Limitations
Fabiani, 2000 Italy	Study Type – case control 1992-1994 Population-group selection Celiacs – selected from screening program for CD ages 11-14, Group A and second group B from pts diagnosed due to typical symptoms (biopsy proven ESPGAN) Loss to follow-up: Group A 5 pts, Group B 2 pts	Celiacs Group A n = 22 pts, asymptomatic Group B n = 22 pts symptomatic Mean follow-up – 5 y Age at diagnosis of CD: Group A 13 y, Group B 4.3 y	Compliance – FFQ conducted by dietician Anthropometric assessment	Adherence to treatment lower in Group A (23%) asymptomatic versus Group B (68%) p value sig BMI no differences between groups	*Difference in adherence could be related to age at diagnosis

Evidence Table 11 (cont'd): Consequences of testing for CD

Author, Year, Country	Methods	Participants	Outcomes	Measures of risk	Limitations
Fickling, 2001, UK	Retrospective case-control study **Group selection** CD pts: All individuals with CD who attended gastroenterology output depart and those with CD who were member of local celiac society Controls: Age/sex matched controls/ one per case selected from database of normal adults who had a bone densitometry performed with the same machine **Institution:** District general hospital **Ascertainment of outcome:** By questionnaire on fracture history with attempt to verify by case notes **Blinding:** n/r **Follow-up:**	**CD pts:** n=75; 15 with metabolic bone disease and CD **Controls:** n=75 **Mean age of CD pts:** 52 y **Proportion on GFD:** full details n/r **Median duration of GFD:** 3.4 y **Proportion of females:** 80% **Clinical presentation:** n/r **BMI:** n/r	**Fractures:** CD pts: 16/75 (21%), 10 before Dx, 6 after Dx Controls: 2/74 (3%) **BMD-DXA** 6 pts had histomorphometry- 3 pts had osteomalacia	RR: 7.0 (95% CI 4.2-125) Increased history of past fractures in subjects with CD. ($p<0.001$) No difference in BMD in those with and without fractures, but pts who had a fracture were older (56.3 vs 50.3)	Not population-based Selection bias No mention that CD was confirmed by biopsy

Evidence Table 11 (cont'd): Consequences of testing for CD

Author, Year Country	Methods	Participants	Outcomes	Results	Limitations
Greco, 1997 Italy	Study type- cohort Population- Celiac – biopsy confirmed pts (92.8%) consecutively recruited from a celiac clinic, 22 pts diagnosed on basis of immunological and clinical findings	Celiac - 306 adolescent/young adults Cases of CD Mean age – 15.9 (range 10-27) Proportion F - 60.8% Mean follow-up BMI	Compliance – one month retrospective questionnaire	Three groups 1. Strict GFD 73% 2. Occasional relapse 15% 3. Full gluten containing 12% Females more compliant (80% vs 64% of males)	88.4% of younger teenagers on a strict diet vs 68.8% of older patients >18 y Avg monthly cost GFD 242,000 Italian Lire, 3 million per year 11.32 kg/month

Evidence Table 11 (cont'd): Consequences of testing for CD

Study, Year Country	Methods	Participants	Outcomes	Results	Risk estimates
Johnston, 1998 N. Ireland	Study type-case control Longitudinal follow-up of cases 1983-1998 Population-Group Selection- population survey Celiacs -screening detected CD of 1823 subjects with serology (n=113)- 3% also had inflammatory bowel disease Controls – 89 age and sex matched randomly selected from survey – antibody negative Ascertainment of outcome – death certificates	Celiac n= 89 (72 followed) – 20 biopsied and 13 untreated detected Controls 89 Proportion F – 53 % Mean follow-up – 11.6 y	Mortality- overall and cancer Number of deaths compared to Registrar General's reports 1983-1994	13 subjects with positive serology died, 4 with malignant disease No increase in all-cause mortality or number of cancer-related deaths in screening detected cases of CD	Cancer death RR 0.94 (95% CI 0.3-2.4) All deaths RR 0.92 (95% CI 0.5, 1.6) Limitations Incomplete follow-up Response rate for biopsy (20/72 is low)

Evidence Table 11 (cont'd): Consequences of testing for CD

Author Year Country	Methods	Participants	Outcomes	Results	Limitations
Kemppainen, 1998 Finland	Study type - cohort Duration: 1988-1990 Population-group selection Celiacs: newly diagnosed biopsy confirmed, symptomatic, all started on GFD Partial (8), subtotal (17) and total VA (15) Controls –none Institution – Tertiary care Kuopio University Hospital Follow-up – 6 pts lost to follow-up	Celiacs – 40 pts Age – 47 for men and 44 for women Proportion F - 70% Proportion on GFD -100% Mean follow-up: 1 y Duration of CD symptoms – males 15.8, women 13.1 BMI 25 in men, and 24 in women	Nutritional status examined by food records, and BMI Ferritin/biochemical values Biopsy	BMI increased after GFD, decreased intakes of fibre, thiamine Most of abnormal biochemical values improved, in 1 pt with subtotal atrophy – low Hgb, in pts with subtotal VA – 7 pts had low Hgb and 5 low ferritin After GFD – VA improved in all patients 29 pts had partial VA, 2 subtotal, 3 normal villi	Baseline - serum ferritin lower in pts with total VA, also had low RBC folate, and ferritin

Evidence Table 11 (cont'd): Consequences of testing for CD

Study, year, location	Methods	Participants	Outcomes	Measures of risk	Limitations
Moreno, 2004, Argentina	Case-control, cross sectional **Group selection** CD pts: unselected 53% classic symptoms, 36% subclinical, 11% silent by screening Controls**:** 296 age and sex matched diagnosed with functional disorders **Institution:** 2 different tertiary referral centres **Ascertainment of fracture**: in-person interview	**CD pts:** n=148 unselected CD **Controls:** n=296 (2:1) **Mean age CD pts:** Classic: 44 y Subclinical/silent: 38 y **Age at diagnosis:** Classic: 42 y Subclinical/silent: 36 y **BMI:** 22/22.1 **Proportion females:** 79 % **Clinical presentation:** 53% classical CD; 35% subclinical, and 11% silent	**Fractures:** classic symptoms vs subclinical overall -51pts **Fractures-peripheral** 47% symptomatic CD vs 15% controls 20% subclinical/silent CD vs 14% controls Mean BMD femoral neck	OR 3.6 (95% CI 1.7-7.5) OR for symptomatic pts 5.2 (95% CI 2.8-9.8) OR 1.7 (0.7-4.4) Fractures not any greater in subclinical cases of CD vs controls Higher for subclinical/silent cases vs classical $p<0.05$ (T score -0.6 vs -1.5) Pts with CD–sig more fractures in 5th/6th decade; also sig more low trauma fractures than controls	Choice of controls–functional disorders/cases Fractures not verified by X-ray report

Evidence Table 11 (cont'd): Consequences of testing for CD

Study, Year Country	Methods	Participants	Outcomes	Measures of risk - mortality	Measure of risk
Nielsen, 1985 Denmark	Study type - retrospective cohort Study dates 1964-82 Population-group selection Celiacs – histologic diagnosis of CD - 100 Ascertainment of outcome-Central person register, cancer registry Tertiary hospital	Celiac pts - 98 Proportion F - 61 % Median age at diagnosis – 41 (F) and 42 (M) Mean follow-up - 18 y % refractory CD - 24% treated with prednisone since did not respond to GFD Compliance – not described how assessed	Mortality	5-y survival 88%, 10-y survival 68.5% (23 deaths, 4 deaths attributed to malignancy) 8 pts developed cancer Responders to GFD – 2.2 extra mortality factor Non-responders to GFD - 5.8 Compliance with diet – 3.2 Non compliant – 4.5	SMR 3.4

Evidence Table 11 (cont'd): Consequences of testing for CD

Author, Year Country	Methods	Participants	Outcomes	Results	Limitations
Poddar, 2002 India	Study type – case control longitudinal Period – 09/1997 – 12/1998 Population-group selection Celiacs-104 children with clinical symptoms evaluated for celiac disease, 57 biopsy confirmed. Excluded those who did not have good response to diet Controls –Those who did not have celiac on biopsy of the initial 104	Celiacs - n= 57 Controls - n=47 Proportion F - Mean follow-up - 19.6 ± 8 mos 17% poor compliance	Height Weight gain Symptoms	Height 88 ± 5% of expected vs 94 ± 5% of expected baseline and follow-up (p=ns) 66% ± 14 vs 86% ± 11 of expected (p<0.001) Improved in 16 ± 9.8 days 34% had poor compliance to diet	Did not analyze on basis of compliance Selection of cases

Evidence Table 11 (cont'd): Consequences of testing for CD

Author, Year Country	Methods	Participants	Outcomes	Results	Limitations
Rea, 1996 Italy	Study type - case control longitudinal Study dates 1992-1994 Population-group selection Celiacs - newly diagnosed children, biopsy confirmed Controls-age and sex matched healthy controls	Celiacs: n= 23 Controls: n=23 Proportion of F - 65.2% Mean age 4.7 ± 0.76 Mean follow-up - 1 y Co-interventions All patients received vitamin D 1000 IU If iron was low, received iron supplementation	Height, BMI Weight, fat area index Triceps subscapular skin fold Biochemical values	Height. BMI, triceps skinfold, fat area index, and weight for height index all improved significantly. Hgb, iron, protein, albumin, triglycerides, calcium and zinc significantly improved Transferring, cholesterol, phosphorus and alk phos were not different	Compared to controls height still significantly lower

Evidence Table 11 (cont'd): Consequences of testing for CD

Study, Year Country	Methods	Participants	Outcomes	Results	Results after treatment
Sategna-Guidetti, 2001 Italy	Study type - longitudinal case control 1996-1998 Population-group selection Celiacs consecutive newly diagnosed CD patients, biopsy proven Controls – age, sex matched healthy volunteers recruited among medical and nursing staff, blood donors or pts affected by COPD, peptic ulcer disease, no past history of thyroid dysfunction Excluded conditions that could affect thyroid function, excluded CD by means of EMA or biopsy Setting- 5 Italian centres	Celiac - 241 Proportion F – 73% Controls – 212 Mean follow-up- 1 y Clinical presentation Typical in 49%, atypical in 44% and silent in 16%	BMI Thyroid function (serum fT3 and fT4 by RIA, TSH by IRA and thyroid microsomal antibodies	Similar in patients with and without thyroid disease Thyroid dysfunction in 73/241 (30.3%) vs 11.3% ($p<0.0005$) Thyroid disease 3 X higher than controls Hypothyroidism diagnosed in 12.9% vs 4.2% of controls ($p<0.003$) 128 pts reassessed at 1 y 91 had normal thyroid, 37 some impairment Subclinical hypothyroidism improved in 71% patients with nonautoimmune thyroid disease	Improvements in BMI, nutritional indices, albumin and serum iron with GFD Gluten withdrawal (confirmed by biopsy recovery) seemed to normalize nonautoimmune thyroid disease 5.5% of pts with normal thyroid function while untreated developed thyroid dysfunction one y later

Evidence Table 11 (cont'd): Consequences of testing for CD

Author, Year Country	Methods	Participants	Outcomes	Results	Limitations
Saukkonen, 2002 Finland	Study type - longitudinal Population-group selection Celiacs, screened 776 children with type 1 diabetes with serology/biopsy (over 2.7 y period) – own controls Controls - none	Celiacs/type 1 diabetes n = 18 Mean onset of diabetes – 8.0 ± 4.5 y Proportion F – 50% Mean follow-up – 1 y	HbA1c GI symptoms	No change in HbA1c levels with GFD Symptoms which were reported in a retrospective questionnaire resolved in all but 2 pts	Significant increase in weight for height after diagnosis No changes in Ht SDS

Evidence Table 11 (cont'd): Consequences of testing for CD

Author, Year Country	Methods	Participants	Outcomes	Results	Limitations
Smecuol, 1997 Argentina	Study type - longitudinal Dates – 1991-1993 Population-group selection Celiacs unselected consecutive patients with newly diagnosed CD, all were symptomatic, and biopsy confirmed acted as own controls	Celiac – 47 and 25 pts re-evaluated in 1995 Proportion F - Mean follow-up - 37 mos Compliance- 15 pts – strict GFD, 10 partial GFD BMI	Fat and bone mass Lean tissue mass Weight and tricep skin fold thickness Mid arm circumference and muscle mass	Significant increase in fat/bone mass No change in lean tissue mass Increases in body weight/triceps skinfold thickness	

Evidence Table 11 (cont'd): Consequences of testing for CD

Study, year, location	Methods	Participants	Outcomes	Measures of risk	Limitations
Thomason, 2003, UK	Case-control study Self-report data from questionnaire. **Group selection:** CD pts: all biopsy-proven CD (also clinical/serology) from population based registers for Derby and Nottingham (less than 7% did not have a bx) Only pts born prior to 1950 included. Controls: age/sex matched random sample from Nottingham family health services. **Ascertainment of fractures:** Self-report of low trauma and non low trauma fractures **Blinding:** Investigator who categorized fractures as low trauma or not was blinded to whether case or control	**CD pts:** n = 244 (70% females) **Controls** n=161 **Mean age:** CD: 60.2 y (10.1) Controls: 61.2 y **Mean BMI:** CD: 23.9 Controls: 25.8 **Proportion of females:** 70% **Proportion on GFD**: n/r **Clinical presentation:** n/r **Dx of osteoporosis:** 7.4% of cases versus 3.1% of controls **Smokers:** CD: 52.5% Controls: 43.3% **Cointerventions:** 4% of CD pts reported taking calcium **HRT:** 31% vs 21% of controls (significant), adjusted for HRT use in analysis	**Fractures – low trauma** (fall from a standing height or less) **Any fracture:** CD: 82 (34.5%) Control: 53 (33.3%) **Forearm/wrist:** CD: 39 (16.4) Control: 22 (13.8) **Low trauma fracture:** CD: 37 (15.7%) Control: 21 (13.8%) (20 reported first fracture before diagnosis of CD and 10 pts reported first fracture after diagnosis) Logistic regression to estimate odds ratio for fracture – adjusted for sex and age group Cox's proportional Hazard model was used and only first low trauma fracture was included.	OR 1.05 (0.68-1.02) OR1.21 (0.66-2.25) OR 1.16 (0.65-2.10) When adjusted age, sex, BMD and smoking the OR 1.13 (95% CI 0.6-2.12) Before Dx: HR 1.24 (95% CI 0.65 – 2.39) Small but statistically-significant increase in risk of fractures	Retrospective Self- report data Response rate to questionnaire was 72% in controls, 89% in celiacs Inadequate power to detect fractures

Evidence Table 11 (cont'd): Consequences of testing for CD

Study, year, location	Methods	Participants	Outcomes	Measures of risk	Limitations
Vasquez, 2000, Argentina	Cross-sectional case-control study Retrospective historical review **Group selection** CD pts: All biopsy-proven celiac disease, and clinical picture, excluded those with secondary osteoporosis other than celiac Controls: 165 subjects selected from output clinic and selected if their final diagnosis was a functional disorder. Excluded cases with known metabolic bone disorders **Institution:** Single institution Buenos Aires –tertiary hospital **Ascertainment of fracture** Pt interview, case report Vertebral fractures –X-ray of lumbar spine (not thoracic) in all CD and 62% of controls Did not have medical records of trauma events **Blinding**: n/r	**CD pts**: n=165 **Median Age:** CD: 40 (16-74) y; 23% over 50 y **Mean age of controls:** 41 y **Median time from symptoms to diagnosis**: 7 y **Proportion of females:** 86.6% **Proportion on GFD:** 69% Strict GFD: 44.8% Reduced gluten: 24% Untreated: 31% **Mean BMI:** 21.4 **Clinical presentation:** malabsorption or subclinical **Post-treatment biopsy:** histological improvement in 38 pts **Serology:** negative or reduced titres in all with positive serology	**Fractures** Peripheral: Cases: 41 (25%) Controls: 14 (8%) Total number of fractures: 51 in celiacs/15 in controls **BMD:** Lower in those with fractures (non-significant) **Exclusions:** Only lumbar spine X-rays from 68 pts and 78 controls were considered to be of adequate quality	**OR: Peripheral fractures** (25%) of CD pts and (8%) of controls; 3.5 (95% CI 1.8-7.2) p<0.0001, wrist most common **Vertebral fractures** 4/78 (5%) of controls versus 9/68 (13%); OR 2.8 (0.7, 1.15) incomplete ascertainment X-rays: 68 pts and 78 controls were adequate quality Most fractures observed pre-diagnosis Only 7% of pt developed fractures after starting GFD	Cases were from a malabsorption clinic and therefore may have included subjects with more severe disease

Evidence Table 11 (cont'd): Consequences of testing for CD

Study, Year, Location	Methods	Participants	Outcomes	Measures of risk	Limitations
Vestergard, 2002, Denmark	Retrospective cohort study, population based Study dates: 1983-1996 **Group selection** CD pts: All individuals admitted/ discharged with a CD Dx (ICD -8) Controls: 3 controls per case population age and sex matched **Institution:** Danish hospitals **Ascertainment of fracture** National pt discharge register–any fracture registered during hospitalization **Confounders:** not examined **Blinding:** n/r Comorbidity examined by assessing hospital admissions for conditions that may alter risk of fractures. (2.8% in CD vs 2.6% in controls)	**CD pts:** n=1,021; 7,774 PY **Controls:** n=23; 316 PY **Mean age at diagnosis:** 31.5 y (24.7) **Mean follow-up:** 7,774 PYs cases; 23,316 controls **Proportion of females:** 58% **Proportion on GFD:** n/r **Compliance:** n/r **Clinical presentation:** symptomatic (admission to hospital) **BMI:** n/r **Age at first fracture after diagnosis:** 40.2 y **Exclusions** ? output diagnosis of CD	**All Fractures: CD vs control** Before diagnosis: 24/7774 PY vs 103/23,316 PY After diagnosis: 65/6,675 vs 223/21,468 PY **Spine Rib Pelvis Osteoporosis**	**Incidence rate ratio (ALL)** Before Dx: 0.70 (0.45,1.09) After Dx: 0.94 (0.71, 1.24**)** (spine, rib, pelvis) IRR 2.14 (0.70, 6.57) pre IRR 1.07 (0.39, 2.95) post IRR 3.00 (0.21,41.9) pre IRR 1.29 (0.4, 4.09) post	Used only hospital-based discharge data therefore could miss output fractures (wrist, rib) Based on assumption that majority of pts pre 1996 hospitalized early in course of disease Validity of diagnosis verified in random sample and was low (78%) n=9 therefore potential for misclassification Selection of controls Some of controls had diseases that may have increased fracture risk

Evidence Table 11: Consequences of testing for CD

Study, Year, Location	Methods	Participants	Outcomes	Measures of risk	Limitations
West, 2003, UK	Retrospective matched cohort study Study dates: 1987-2002 **Group selection** Pts with CD: All recorded Dx of CD recorded 1-yr after beginning of the GPRD record; no biopsy data Controls: matched by age, sex, general practice and follow-up time, excluded any who had record of a gluten-free prescription **Primary care database:** GPRD established in 1987 **Ascertainment of fracture:** Admin database **Blinding:** n/r **Examined potential confounders**	**CD pts**: n=4,732 observ. time (27,116) **Controls:** n=23,620 (149,896 y of risk) **Mean age at diagnosis celiac**: 43.5 y Range of age: n/r **Proportion of females:** 67% **Proportion smoke:** 13% (controls 15.4%) **Mean follow-up:** 5.7 y, # PYs: 27,116 10% of cohort did not receive a prescription for GFD, 36% 0-10 and 54≥10 **BMI:** Celiacs: <25 – 47% Controls: <25: 30.6% ≤18.5; 4.2% vs 1.2% of controls	**Fracture: CD vs control** **Any fracture:** 356/4732 vs 1524/23616 137.9/10,000 PYs vs 105.9/10,000 in controls **Hip:** 8.9/10,000 PY versus 4.7/10,000 PY **Ulna/radius fracture:** 24.9/10,000 versus 14.1/10,000 No difference in risk of fracture in period after diagnosis compared to before diagnosis	HR: 1.30 (95% CI, 1.16,1.46) (report prevalent and incident) HR 1.9 (1.20,3.02) HR 1.77 (1.35,2.34) Absolute diff in overall fracture rate was 3.2/1000 person y 0.97/1000 for hip fracture in those >45 y	Celiacs more frequent attenders, ?overestimate rate of some fractures relative to controls Misclassification – accuracy of diagnosis of CD ? Less likely in UK, since GPs not likely to write prescription for GFD

Evidence Table 11 (cont'd): Consequences of testing for CD

Author, Year Country	Methods	Participants	Outcomes	Results	Limitations
Westman 1999 Australia	Study type- Population-coexisting type 1 diabetics and CD identified from database of Diabetes Center-biopsy proven Controls- 40 matched for age and sex and duration of IDDM	Cases 20 Control 40 Duration of diabetes: 7.2 vs 7.3 y controls % F - 75% Mean follow-up Compliance with FFQ	Height SDS Weight SDS BMI SDS HbA1c Compliance	No difference in height SDS, weight SDS or BMI SDS 8.48 ± 0.98% for CD vs 8.87 ± 1.46 for controls 30% strict GF, 30% trace gluten and 40% significant amt of gluten	

Celiac 5: Promoting or Monitoring Adherence to a GFD

Evidence Table 12: Promoting or monitoring adherence to a GFD

Author, year, location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Anson, 1990, Israel	**Publication type:** journal **Study design:** Parental questionnaire **Ethnicity:** n/r **Population type:** Jewish children with CD **Biopsy criteria:** n/r **Checked IgA def.** no **Serologic tests:** Test name: n/a Methodology: n/a Cut-off: n/a	**Celiac Group 1** •31 children judged GFD compliant based on symptoms and/or serology and/or biopsy •age: n/r •%F: n/r **Celiac Group 2** • 12 children judged non-compliant with GFD • age: n/r • %F: n/r	Baseline **Biopsy** **AGA** **EMA** **tTG**				Compliance correlated with father being professional/parental education/parental ability to choose GF items from menu.

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, year, location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Bardella, 2001, Italy	**Publication type:** journal **Study design:** case-control **Ethnicity:** n/r **Population type:** CD: details of diagnosis n/r **Biopsy criteria:** Marsh **Checked IgA def.** Yes, excluded **Serologic tests:** Test name: IgA-AGA; IgA-EMA; IgA-tTG Methodology: n/r; ME; GP liver Cut-off: 12 AU/mL; 1:10; 10 AU/mL	**Celiac Group 1** • 40 pts, untreated CD • mean age: 38 y (range 16-77 y) **Celiac Group 2** • 25 CD; poor GFD compliance • age: n/r • gender: n/r **Celiac Group 3** • 22 CD. Compliant GFD<2 y • age: n/r • gender: n/r **Celiac Group 4** • 148 CD. Compliant GFD >2 y • age: n/r gender: n/r Control • 110 non-CD controls • age: nr • gender: n/r	Baseline **Biopsy** **AGA** Group 1 95%; Group 2 100%; Group 3 40.9%; Group 4 16.2%; Control 10.9% **EMA** 100%; 100%; 54.5%; 9.5%; 2.7% **tTG** 100%; 100% 63.6%; 14.2% 1.8%				Serology falls with increasing length of compliance with GFD. No biopsy to correlate serology with in this study.

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, year, location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Bartholomeus, 1990, Australia	**Publication type:** journal **Study design:** cohort **Ethnicity:** n/r **Population type:** CD: details of diagnosis n/r **Biopsy criteria:** Adults partial VA, subtotal VA, total VA; children ESPGAN **Checked IgA def.** No **Serologic tests:** Test name: IgA-AGA Methodology: n/r Cut-off: n/r	**Celiac Group 1** • 17 CD. GFD >6 mos adults and children • age: n/r • gender: n/r **Celiac Group 2** • 12 adults and children with CD on gluten • age: n/r • gender: n/r	Baseline				PPV of IgA AGA for non-compliance 78.5% for pts on GFD > 6 mos. How compliance ascertained not described.
			Biopsy				
			AGA Group 1 3 (17%); Group 2 11 (92%)				
			EMA				
			tTG				

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, year, location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Burgin-Wolff, 1991, Switzerland	**Publication type:** journal **Study design:** case-control **Ethnicity:** n/r **Population type:** Classic CD **Biopsy criteria:** CD if subtotal VA, total VA **Checked IgA def.** no **Serologic tests:** Test name: IgA-AGA ; IgA-EMA Methodology: n/r; ME Cut-off: n/r; 1:10	**Celiac Group 1** • 134 children CD on GFD • mean age: n/r (range 3-18 y) • gender: n/r	Baseline	GC 36 to 90 d	GC 3 to 12 mos	GC > 3 y	With gluten challenge IgA AGA and EMA seroconversion. AGA rises faster but positivity wanes with time.
			Biopsy				
			AGA 23% IgA-AGA	97%	5%	49%	
			EMA 13% EMA	65%	84%	93%	
			tTG				

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, year, location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Dickey, 2000, Northern Ireland	**Publication type:** journal **Study design:** prospective cohort **Ethnicity:** n/r **Population type:** Adults with classic CD **Biopsy criteria:** Marsh **Checked IgA def.** Yes. Excluded **Serologic tests:** Test name: IgA EMA Methodology: ME Cut-off: >1:5	**Celiac Group 1** • 53 pts • mean age: 55; range 16-80 y • 74% F	Baseline	GFD 3 mos	GFD 6 mos	GFD 12 mos	EMA failed to detect VA in a significant number. These cases were felt to have VA due to non-compliance.
			Biopsy 12 partial VA; 21 subtotal VA; 21 total VA			20 normal;1 Marsh 1 (high IEL); 22 partial VA; 10 total VA or subtotal VA	
			AGA				
			EMA 100%	42%	25%	0% normal; 0% Marsh 1; 2 (9%) partial VA; 3 (30%) subtotal/total VA	
			tTG				

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, year, location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Fabiani, 1996, Italy	**Publication type:** journal **Study design:** retrospective cohort **Ethnicity:** n/r **Population type:** CD identified by screen **Biopsy criteria:** no description **Checked IgA def.** No **Serologic tests:** Test name: IgA AGA; IgA EMA Methodology: n/r Cut-off: n/r	**Celiac Group 1** • 12 children reporting strict GFD • age: n/r • gender: n/r **Celiac Group 2** • 11 children reporting non-compliance • age: n/r • gender: n/r	Baseline				Among pts reporting dietary transgressions 9 of 11 (81%) had normal AGA and EMA.
			Biopsy				
			AGA				
			EMA				
			tTG				

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, year, location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Fabiani, 2000, Italy	**Publication type:** journal **Study design:** case-control **Ethnicity:** n/r **Population type:** CD identified in a mass screen **Biopsy criteria:** ESPGAN **Checked IgA def.** No **Serologic tests:** Test name: n/a Methodology: n/a Cut-off: n/a	**Celiac Group 1** • 22 children CD identified by screen • mean age: 17.9 y; range: n/r • 59% F **Control** • 22 children with classic CD on GFD • mean age: 16 y; range: n/r • 59% F	Baseline **Biopsy** **AGA** **EMA** **tTG**				5 (23%) identified by screen reporting strict GFD. 15 (68%) of controls reporting strict GFD? Less compliant if screened CD.

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, year, location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Fabiani, 2001, Italy	**Publication type:** journal **Study design:** case-control **Ethnicity:** n/r **Population type:** Classic CD **Biopsy criteria:** Marsh **Checked IgA def.** Yes. Excluded **Serologic tests:** Test name: IgA-tTG Methodology: GP liver Cut-off: 7 AU	**Celiac Group 1** • 176 pt new diagnosis CD • mean age: 16.4 y; range 0.3-87.4 y • 65% F **Celiac Group 2** • 172 CD on GFD > 1 • mean age: 17.6 y; range 0.3 - 89.8 y • 66% F **Control** • 206 healthy and non-CD • mean age: 15.6 y; range 0.4-78 y • 50% F	Baseline				tTG value higher in higher Marsh lesions. Values tend to be higher in pts admitting dietary transgressions.
			Biopsy Group 1: 0 Marsh 1, 29 (16%) Marsh 2, 145 (84%) Marsh 3 Group 2: n/a Control: n/a				
			AGA				
			EMA				
			tTG Group 1: mean tTG Marsh 2 16 AU, Marsh 3 22 AU Group 2: strict GFD 4.2 AU, GFD transgressions 9.9 AU Control: 2.7 AU				

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, year, location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Fotoulaki, 1999, Greece	**Publication type:** journal **Study design:** prospective cohort **Ethnicity:** n/r **Population type:** Classic and atypical CD **Biopsy criteria:** ESPGAN **Checked IgA def.** yes **Serologic tests:** Test name: IgG AGA; IgG AGA; IgA EMA Methodology: n/r; ME Cut-off: 0.3 control; 0.3 control; 1:2.5	**Celiac Group 1** • 30 CD • mean age: n/r; range 1-24 y • 57% F **Celiac Group 2** • median age: (range:) • % F:	Baseline	GFD 12 mos	GC 6 mos		Study suggests that after 1 y GFD, AGA and EMA predict Marsh 0 or 1 lesion. Significance of Marsh 1 unclear.
			Biopsy n/r	100% Marsh 0 or 1 (breakdown not given)	All Marsh 2 or 3 (breakdown not given)		
			AGA IgG AGA 100%; 90% IgA AGA	40% IgG AGA; 0% IgA AGA	100% IgG: 90% IgA		
			EMA 100%	0%	90%		
			tTG				

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, year, location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Hogberg, 2003, Sweden	**Publication type:** journal **Study design:** retrospective cohort **Ethnicity:** n/r **Population type:** Classic CD **Biopsy criteria:** ESPGAN **Checked IgA def.** no **Serologic tests:** Test name: IgG-EMA; IgA-EMA; tTG Methodology: ME; GP Cut-off: 1:10;1:10;>25 AU	**Celiac Group 1** • 29 adults diagnosed as a child • mean age: 26 y; range 19-34 y • 69% F	Baseline				12/15 (80%) diagnosed before age 4 judged GFD compliant (serology/questionnaire) vs. only 5/14 (36%) diagnosed after age 4.
			Biopsy				
			AGA				
			EMA				
			tTG				

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, year, location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Jackson, 1985, Ireland	**Publication type:** journal **Study design:** questionnaire **Ethnicity:** n/r **Population type:** Classic CD **Biopsy criteria:** CD if severe VA and gluten response **Checked IgA def.** no **Serologic tests:** Test name: n/a Methodology: n/a Cut-off: n/a	**Celiac Group 1** • 50 children CD • median age: 9.9 y; range 1.5-19 y • 58% F	Baseline				GFD judged by parental questionnaire correlated with parental membership in Celiac society, parental knowledge of CD.
			Biopsy				
			AGA				
			EMA				
			tTG				

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, year, location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Kaukinen, 2002, Finland	**Publication type:** journal **Study design:** prospective cohort **Ethnicity:** n/r **Population type:** Classic CD **Biopsy criteria:** Marsh **Checked IgA def.** Yes. Excluded **Serologic tests:** Test name: IgA-EMA; IgA-tTG Methodology: HU; GP liver Cut-off: 1:5; 1:20	**Celiac Group 1** • 87 pts on GFD • mean age: 49 y; range 22-73 y • 72% F	Baseline GFD median 1 y				tTG sens 41% spec 88% PPV 61% NPV 77% EMA sens 26% spec 93% PPV 63% NPV 74%
			Biopsy 60 (69%) Marsh 0-II; 27 (31%) Marsh III				
			AGA				
			EMA 7 (26%) in Marsh III				
			tTG 11 (41%) in Marsh III				

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, year, location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Lamontagne, 2001, Canada	**Publication type:** journal **Study design:** questionnaire **Ethnicity:** n/r **Population type:** **Biopsy criteria:** **Checked IgA def.** **Serologic tests:** Test name: n/a Methodology: n/a Cut-off: n/a	**Celiac Group 1** • 234 CD. Members of Quebec Celiac Foundation • mean age: 49 y; range 18-84 y • 75% F	Baseline				Older age (p<.05) & high level of confidence in treatment information from gastroenterologists & dieticians (p<.005) assoc. compliance.
			Biopsy				
			AGA				
			EMA				
			tTG				

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, year, location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Lee, 2003, USA	**Publication type:** journal **Study design:** retrospective cohort **Ethnicity:** n/r **Population type:** Classic CD **Biopsy criteria:** Normal, partial VA or total VA **Checked IgA def.** n/r **Serologic tests:** Test name: IgG AGA; IgA AGA; IgA AMA Methodology: n/r Cut-off: n/r	**Celiac Group 1** • n=39 pts on GFD • mean age: 52 y; range 20-74 y • 60% F	Baseline	1-14 y after initial Dx: only 12 pts re-biopsied; 31/39 had serology			Persistent abnormal biopsy on GFD. Only 21% had normal duodenal biopsy; serology was negative despite some VA in most.
			Biopsy Normal 8 (21%); partial VA 27 (69%); total VA 4 (10%); IEL/epithelial cells 61	IEL/100 epithelial cells-38; villous height to crypt ratio improved in all but one; none normalized			
			AGA	7; IgG only 4; IgG and IgA in 3			
			EMA	1 of 31			
			tTG				

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, year, location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Ljungman, 1993, Sweden	**Publication type:** journal **Study design:** questionnaire **Ethnicity:** n/r **Population type:** Classic CD **Biopsy criteria:** ESPGAN **Checked IgA def.** n/r **Serologic tests:** Test name: n/a Methodology: n/a Cut-off: n/a	**Celiac Group 1** • 47 children born between 1973-78 • mean age: n/r • gender: n/r	Baseline **Biopsy** **AGA** **EMA** **tTG**				Self-assessed GFD compliance correlated with knowledge as scored on a test and with a feeling of being well informed.

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, year, location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Martini, 2002 Italy	**Publication type:** journal **Study design:** prospective cohort **Ethnicity:** n/r **Population Type:** Classic CD **Biopsy criteria:** Marsh **Checked IgA def.** n/r **Serologic tests:** Test name: IgA-EMA; IgA-tTG-1; tTG-2, tTG-3, tTG4, tTG-GP Methodology: ME; HU; HU; HU; HU; GP Cut-off: 1:5; n/r	**Celiac Group 1** • 109 CD on GFD 1 y • median age: 37 y; range 21-72 y • 78% F	Baseline (at diagnosis)	GFD 1 y +/- 1mos			Poor concordance between biopsy and serology after GFD for one year. GFD led to a significant decrease in majority.
			Biopsy 6 (6%) Marsh 2; 95 (95%) Marsh3	12 (12%) normal; 51 (50%) Marsh 1; 38 (38%) Marsh 2 or 3			
			AGA n/a	n/a			
			EMA n/a	Concordance (both biopsy/EMA + or both) 48%			
			tTG n/a	Concordance 29%; 65%;14%; 16%; 19%			

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, year, location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
McNichol, 1976, Ireland	**Publication type:** journal **Study design:** cohort **Ethnicity:** n/r **Population type:** Classic CD **Biopsy criteria:** Normal, slight, moderate, severe mucosal damage **Checked IgA def.** n/r **Serologic tests:** Test name: n/a Methodology: n/a Cut-off: n/a	**Celiac Group 1** • 36 children on GFD (mean 6 y) • mean age: n/r; range 2.75-11 y • gender: n/r **Control** • 25 normal siblings • mean age: 9.9; range: 4-18 • gender: n/r	Baseline				IEL count higher in CD with slight damage than no damage (p<.005) or controls (p<.001)
			Biopsy Group 1- 16 (44%) normal. 20 (56%) slight mucosal damage; controls all normal				
			AGA				
			EMA				
			tTG				

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, Year, Location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Pacht 1995, Israel	**Publication type:** journal **Study design:** retrospective cohort **Ethnicity:** n/r **Population Type:** Classic CD **Biopsy criteria:** ESPGAN **Checked IgA def.** n/r **Serologic tests:** Test name: IgA EMA Methodology: ME Cut-off: >1:2.5	**Celiac Group 1** • 39 CD, 22 GCD and 17 GFD • mean age: 10 y; range: n/r • % F: 46% **Celiac Group 2** • median age: (range:) • % F:	Baseline 22 GCD;17 GFD				EMA titre monitored longitudinally in 10 pts correlated with gluten intake. EMA correlated with investigator gluten assessment.
			Biopsy n/r; histology available on 5 on GFD - all normal				
			AGA				
			EMA 22/22 GCD EMA+; 0/17 GFD EMA+				
			tTG				

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, year, location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Sategna-Guidetti, 1996, Italy	**Publication type:** journal **Study design:** prospective cohort **Ethnicity:** n/r **Population Type:** CD: details of diagnosis not reported **Biopsy criteria:** Normal, partial VA, subtotal VA, total VA **Checked IgA def.** Yes, excluded **Serologic tests:** Test name: total AGA; IgA EMA Methodology: n/r; ME Cut-off: n/r	**Celiac Group 1** • 47 pts • mean age: n/r; range: 18-68 y • 55% F	Baseline	GFD 8-30 mos			EMA PPV for abnormal histology 100%, NPV 23%; AGA (total) PPV for abnormal histology 93.8%, NPV 25%
			Biopsy 0 normal; 1 (2.1%) partial VA; 11 (23%) subtotal VA; 35 (74%) total VA	9 (19%) normal ; 23 (49%) partial VA; 13 (28%) subtotal VA ;2 (4%) total VA			
			AGA 39 (83%)	AGA measured in 39; Bx normal: 1/7 AGA+; partial VA: 7/20; subtotal VA: 5/10; total VA: 2/2			
			EMA 47 (100%)	Bx normal: 0 EMA+; partial VA: 5/23; subtotal VA: 3/13; total VA: 1/2			
			tTG n/a				

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, year, location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Scalici, 2003, Italy	**Publication type:** journal **Study design:** prospective cohort **Ethnicity:** n/r **Population type:** Classic CD **Biopsy criteria:** ESPGAN **Checked IgA def.** n/r **Serologic tests:** Test name: IgA EMA Methodology: n/r Cut-off: n/r	**Celiac Group 1** • 61 CD pts • mean age: n/r; age range 2-27 y • %F: n/r **Celiac Group 2** • median age: (range:) • % F:	Baseline (at diagnosis)	GFD for 6 mos			Only 11.1% EMA + if 1 dietary transgression/ mos (after 6 mos GFD). 19% EMA + if 1 or more dietary transgressions per week
			Biopsy n/r	n/a			
			AGA				
			EMA 0 EMA+	2/16 (12.5%) reporting strict GFD EMA+; 5/45 (11.1%) admitting dietary mistakes EMA+			
			tTG				

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, Year, Location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Selby, 1999 Australia	**Publication type:** journal **Study design:** cross sectional **Ethnicity:** n/r **Population Type:** Classic CD **Biopsy criteria:** Normal, partial VA, subtotal VA, total VA **Checked IgA def.** n/r **Serologic tests:** Test name: n/a Methodology: n/a Cut-off: n/a	**Celiac Group 1** • 89 pts CD on GFD mean 8.3 y • mean age: 47 y; range: 20-75 • 82% F	Baseline 39 Codex diet; 50 no detectable gluten				VA persisted at high rates after prolonged GFD (whether Codex diet allowing .03% protein from gluten or on no detectable GFD)
			Biopsy 18/39 (46%) VA; 20/50 (40%) VA				
			AGA				
			EMA				
			tTG				

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, Year, Location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Troncone, 1995 Italy	**Publication type:** journal **Study design:** cohort **Ethnicity:** n/r **Population Type:** CD : details of diagnosis not reported **Biopsy criteria:** Normal, partial VA, subtotal VA, total VA **Checked IgA def.** n/r **Serologic tests:** <u>Test name:</u> IgA EMA <u>Methodology:</u> ME <u>Cut-off:</u> >1:5	**Celiac Group 1** • 4 CD on strict GFD (23 in overall study • mean age: 14.5 y; range 10-19 y • 35% F) **Celiac Group 2** • 6 pts <0.5 g/d gluten • median age: n/r; range: n/r • % F: n/r **Celiac Group 3** • 6 pts 0.5-1.0 g/d gluten • median age: n/r; range: n/r • % F: n/r **Celiac Group 4** • 7 pts>2 g/d gluten • median age: n/r; range: n/r • % F: n/r	Baseline				EMA often did not pick up mild dietary transgressions despite mucosal abnormalities including VA.
			Biopsy Group 1 4/4 normal; Group 2 3/6 high IEL, 1/6 VA; Group 3 5/6 high IEL 3/6 VA; Group 4 7/7 VA				
			AGA				
			EMA Group1 0/4; Group 2 1/6; Group 3 3/6; Group 4 7/7				
			tTG				

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, Year, Location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Vahedi, 2000 France	**Publication type:** abstract **Study design:** cross sectional **Ethnicity:** n/r **Population type:** CD: details of diagnosis not reported **Biopsy criteria:** no description **Checked IgA def.** Yes. Excluded **Serologic tests:** Test name: IgA EMA; IgA tTG Methodology: n/r Cut-off: 1:5; n/r	**Celiac Group 1** • 137 CD on GFD>1 y - median 75 mos • median age: 46 y; range 18-74 y • 76% F	Baseline (39% strict GFD)				EMA and tTG often negative despite dietary transgression. EMA 37% sensitive for minor transgression. tTG 31% sensitive.
			Biopsy n/a				
			AGA				
			EMA 2.5% strict GFD; 37% minor transgression; 86% major transgression				
			tTG 3% strict GFD; 31% minor transgression; 77% major transgression				

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, year, location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Valentini, 1994, Italy	**Publication type:** journal **Study design:** prospective cohort **Ethnicity:** n/r **Population Type:** Classic and atypical CD **Biopsy criteria:** Normal, partial VA, subtotal VA **Checked IgA def.** n/r **Serologic tests:** Test name: IgA EMA Methodology: ME Cut-off: >1:5	**Celiac Group 1** • 33 CD pts on GFD >6 mos- mean 9 mos • mean age: n/r; range: n/r • % F: n/r	Baseline GFD > 6 mos				17/24 EMA negative after 6 mos on a GFD despite VA on biopsy
			Biopsy 8 normal; 21 partial VA; 4 subtotal VA				
			AGA				
			EMA 9 (27%). Of 73% EMA negative, 7 normal,14 partial VA,3 subtotal VA				
			tTG				

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, Year, Location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Valetta, 1990 Italy	**Publication type:** journal **Study design:** prospective cohort **Ethnicity:** n/r **Population type:** CD: details of diagnosis not reported **Biopsy criteria:** N,slight VA, moderate VA, severe VA **Checked IgA def.** n/r **Serologic tests:** Test name: IgA AGA Methodology: n/r Cut-off: >3 SD controls	**Celiac Group 1** • 17 pts given gluten challenge • mean age: n/r; range 3 – 11 y • 59% F	Baseline				With GC, most AGA+ which prompted Bx showing VA in all. Does not necessarily mean compliance could be monitored.
			Biopsy at 20-45 d, all had moderate or severe VA				
			AGA 16/17 (94%) after 15 - 35 d gluten challenge				
			EMA				
			tTG				

Evidence Table 12 (cont'd): Promoting or monitoring adherence to a GFD

Author, year, location	Study Design	Study Population	Outcomes				Comments
			Time 1	Time 2	Time 3	Time 4	
Wahab, 2002, unknown	**Publication type:** journal **Study design:** retrospectve cohort **Ethnicity:** n/r **Population type:** Classic CD **Biopsy criteria:** Marsh **Checked IgA def.** No **Serologic tests:** Test name: n/a Methodology: n/a Cut-off: n/a	**Celiac Group 1** • 158 pts • mean age: 44 y; range 0 – 74 y • 91% F **Celiac Group 2** • median age: (range: n/r) • % F:	Baseline	GFD <2 years	GFD 2-5 years	GFD >5 years	Overall, only 65% recovery within 2 y. 10% with no recovery after 5 y. Recovery defined as no VA (Marsh 0-2).
			Biopsy 59 (37%) Marsh 3a; 59 (37%) 3b; 40 (25%) 3c	88% 3a recovery to 0-2; 58% 3b recovery ; 81% 3c recovery	98% 3a recovery to 0-2; 78% 3b recovery; 81% 3c recovery	98% 3a recovery to 0-2; 88% 3b recovery; 86% 3c recovery	
			AGA				
			EMA				
			tTG				

Listing of Included Studies

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Appendix J. Quality Assessment

Table 1: Celiac 1 diagnostic studies (QUADAS)

Author (Year)	Item 1: spectrum of patients	Item 2: selection criteria	Item 3: reference standard	Item 4: time period	Item 5: sampling	Item 6: test result	Item 7: sampling independence	Item 8a: index test description	Item 8b: reference standard description	Item 9a: reference standard results(1)	Item 9b: reference standard results (2)	Item 10: interpretation	Item 11: uninterpretable/ intermediate test result	Item 12: withdrawal explanation	Total No. of Items = 14	
Altuntas (1998)	yes	yes	yes	yes	yes	yes	yes	yes	yes	no	unclear	yes	yes	yes	**12**	(+) reported
Artan (1998)	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	unclear	yes	yes	yes	**13**	(+) reported
Ascher (1996)	yes	yes	yes	yes	yes	yes	yes	yes	yes	unclear	unclear	yes	yes	yes	**12**	(+) reported
Ascher (1990)	yes	yes	yes	yes	yes	yes	yes	unclear	yes	unclear	unclear	yes	yes	yes	**11**	(+) reported
Bahia (2001))	yes	yes	yes	yes	yes	yes	yes	yes	yes	unclear	unclear	yes	no	no	**10**	(+) reported
Bardella (2001)	yes	yes	yes	yes	yes	yes	yes	unclear	yes	unclear	unclear	yes	yes	yes	**11**	(+) reported
Berger (1996)	no	yes	yes	unclear	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	**12**	(+) reported
Biagi (2001)	no	unclear	yes	no	yes	yes	yes	yes	yes	no	unclear	yes	yes	yes	**9**	(+) reported
Bode (1993)	yes	yes	yes	yes	yes	yes	yes	yes	yes	unclear	unclear	yes	yes	yes	**12**	(+) reported
Bonamico (2001)	no	unclear	yes	no	yes	yes	yes	yes	unclear	no	unclear	yes	yes	yes	**8**	(+) reported

Table 1: Celiac 1 diagnostic studies (QUADAS) (cont'd)

Author (Year)	Item 1: spectrum of patients	Item 2: selection criteria	Item 3: reference standard	Item 4: time period	Item 5: sampling	Item 6: test result	Item 7: sampling independence	Item 8a: index test description	Item 8b: reference standard description	Item 9a: reference standard results(1)	Item 9b: reference standard results (2)	Item 10: interpretation	Item 11: uninterpretable/ intermediate test result	Item 12: withdrawal explanation	Total No. of Items = 14	
Bottaro (1997)	no	no	yes	no	yes	yes	yes	unclear	yes	yes	unclear	yes	yes	yes	**9**	(+) reported
Carroccio (2002)	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	unclear	yes	yes	yes	**13**	(+) reported
Carroccio (1993)	no	yes	yes	yes	yes	yes	yes	yes	yes	yes	unclear	yes	yes	yes	**12**	(+) reported
Carroccio (2002)	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	unclear	yes	yes	yes	**13**	(+) reported
Cataldo (2000)	no	yes	yes	unclear	yes	yes	yes	yes	yes	no	unclear	yes	yes	yes	**10**	(+) reported
Chan (2001)	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	unclear	yes	yes	yes	**13**	(+) reported
Chartrand (1997)	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	unclear	yes	yes	yes	**13**	(+) reported
Chirdo (1999)	yes	unclear	yes	yes	yes	yes	yes	yes	yes	no	unclear	yes	yes	yes	**11**	(+) reported
Chirdo (2000)	no	no	yes	yes	unclear	yes	yes	yes	yes	unclear	unclear	yes	yes	yes	**9**	(+) reported
Dahele (2001)	no	no	yes	yes	yes	yes	yes	yes	yes	yes	unclear	yes	yes	yes	**11**	(+) reported
Di Leo (1999)	no	unclear	yes	unclear	yes	yes	yes	yes	no	no	unclear	yes	yes	yes	**8**	(+) reported
Dickey (2001)	no	unclear	yes	yes	yes	yes	yes	yes	yes	yes	unclear	yes	yes	yes	**11**	(+) reported

Table 1: Celiac 1 diagnostic studies (QUADAS) (cont'd)

Author (Year)	Item 1: spectrum of patients	Item 2: selection criteria	Item 3: reference standard	Item 4: time period	Item 5: sampling	Item 6: test result	Item 7: sampling independence	Item 8a: index test description	Item 8b: reference standard description	Item 9a: reference standard results(1)	Item 9b: reference standard results (2)	Item 10: interpretation	Item 11: uninterpretable/ intermediate test result	Item 12: withdrawal explanation	Total No. of Items = 14	
Falth-Magnus-son (1994)	yes	yes	yes	yes	yes	yes	yes	yes	yes	unclear	unclear	yes	yes	no	**11**	(+) reported
Gillett (2000)	no	no	yes	yes	yes	no	yes	yes	yes	unclear	unclear	yes	yes	yes	**9**	(+) reported
Gonczi (1991)	yes	yes	yes	yes	no	yes	yes	yes	yes	no	unclear	yes	yes	yes	**11**	(+) reported
Hallstrom (1989)	no	no	yes	unclear	yes	yes	yes	yes	yes	unclear	unclear	yes	yes	yes	**9**	(+) reported
Hansson (2000)	no	no	yes	yes	yes	yes	yes	yes	yes	unclear	unclear	yes	yes	yes	**10**	(+) reported
Iltanen (1999)	yes	yes	yes	yes	yes	yes	yes	yes	yes	unclear	unclear	yes	yes	yes	**12**	(+) reported
Kaukinen (2000)	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	unclear	yes	yes	yes	**13**	(+) reported
Kolho (1997)	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	**14**	(+) reported
Kumar (1989)	yes	yes	yes	yes	unclear	yes	yes	yes	yes	unclear	unclear	yes	yes	yes	**11**	(+) reported
Ladinser (1994)	no	no	yes	unclear	unclear	unclear	yes	yes	yes	unclear	unclear	yes	unclear	unclear	**5**	(+) reported

Table 1: Celiac 1 diagnostic studies (QUADAS) (cont'd)

Author (Year)	Item 1: spectrum of patients	Item 2: selection criteria	Item 3: reference standard	Item 4: time period	Item 5: sampling	Item 6: test result	Item 7: sampling independence	Item 8a: index test description	Item 8b: reference standard description	Item 9a: reference standard results(1)	Item 9b: reference standard results (2)	Item 10: interpretation	Item 11: uninterpretable/ intermediate test result	Item 12: withdrawal explanation	Total No. of Items = 14	
Lerner (1994)	no	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	**13**	(+) reported
Lindberg (1985)	yes	yes	yes	yes	yes	yes	yes	yes	yes	unclear	unclear	yes	yes	yes	**12**	(+) reported
Lindquist (1994)	yes	yes	yes	yes	yes	yes	yes	yes	yes	no	unclear	yes	yes	yes	**12**	(+) reported
Maki (1991)	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	unclear	yes	yes	yes	**13**	(+) reported
McMillan (1991)	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	**14**	(+) reported
Meini (1996)	yes	yes	yes	yes	yes	yes	yes	yes	yes	unclear	unclear	yes	yes	yes	**12**	(+) reported
Pacht (1995)	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	unclear	yes	yes	yes	**13**	(+) reported
Picarelli (2000)	yes	unclear	yes	yes	yes	yes	yes	yes	yes	no	unclear	yes	yes	no	**10**	(+) reported
Poddar (2002)	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	unclear	yes	yes	unclear	**12**	(+) reported
Rich (1990)	yes	yes	yes	yes	yes	yes	yes	yes	yes	no	unclear	yes	yes	yes	**12**	(+) reported
Russo (1999)	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	unclear	yes	yes	yes	**13**	(+) reported

Table 1: Celiac 1 diagnostic studies (QUADAS) (cont'd)

Author (Year)	Item 1: spectrum of patients	Item 2: selection criteria	Item 3: reference standard	Item 4: time period	Item 5: sampling	Item 6: test result	Item 7: sampling independence	Item 8a: index test description	Item 8b: reference standard description	Item 9a: reference standard results(1)	Item 9b: reference standard results (2)	Item 10: interpretation	Item 11: uninterpretable/ intermediate test result	Item 12: withdrawal explanation	Total No. of Items = 14	
Salmaso (2001)	no	unclear	yes	unclear	unclear	unclear	yes	yes	no	unclear	unclear	yes	yes	yes	**6**	(+) reported
Sategna-Guidetti (1995)	no	unclear	yes	unclear	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	**11**	(+) reported
Sblattero (2000)	no	yes	yes	no	yes	no	yes	yes	yes	yes	unclear	yes	yes	yes	**10**	(+) reported
Sulkanen (1998)	no	yes	yes	yes	yes	yes	yes	yes	yes	unclear	unclear	yes	yes	yes	**11**	(+) reported
Tesei (2003)	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	unclear	yes	yes	yes	**13**	(+) reported
Troncone (1999)	yes	yes	yes	yes	yes	yes	yes	yes	yes	unclear	unclear	yes	yes	yes	**12**	(+) reported
Valdimars-son (1996)	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	unclear	yes	yes	yes	**13**	(+) reported
Valentini (1994)	no	no	yes	yes	yes	yes	yes	yes	yes	unclear	unclear	yes	yes	yes	**10**	(+) reported
Vitoria (2001)	no	no	yes	no	yes	yes	yes	yes	no	no	unclear	yes	yes	yes	**8**	(+) reported
Vogelsang (1995)	yes	yes	yes	yes	yes	yes	yes	yes	yes	no	unclear	yes	yes	yes	**12**	(+) reported

Table 1: Celiac 1 diagnostic studies (QUADAS) (cont'd)

Author (Year)	Item 1: spectrum of patients	Item 2: selection criteria	Item 3: reference standard	Item 4: time period	Item 5: sampling	Item 6: test result	Item 7: sampling independence	Item 8a: index test description	Item 8b: reference standard description	Item 9a: reference standard results(1)	Item 9b: reference standard results (2)	Item 10: interpretation	Item 11: uninterpretable/ intermediate test result	Item 12: withdrawal explanation	Total No. of Items = 14	
Volta (1995)	no	unclear	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	**12**	(+) reported
Whelan (1996)	no	unclear	yes	unclear	yes	yes	yes	yes	unclear	unclear	unclear	yes	yes	yes	**8**	(+) reported
Wolters (2002)	yes	no	yes	unclear	yes	yes	yes	yes	yes	no	unclear	yes	yes	yes	**10**	(+) reported
Item % (+) reported	**59%**	**64%**	**100%**	**75%**	**91%**	**93%**	**100%**	**95%**	**91%**	**41%**	**11%**	**100%**	**96%**	**91%**		

NOTE: (+) reported = postively reported; yes = reported; no = not reported

Table 2: Celiac 2 prevalence & incidence studies (cross-sectional checklist)

Author (Year)	Item 1: Source of Information	Item 2: Inclusion/Exclusion Criteria	Item 3: Time Period for Identity	Item 4: Subjects consecutive	Item 5: Evaluators Masked	Item 6: Quality Assurance Assessments	Item 7: Patient Exclusions	Item 8: Confounding assessed/controlled	Item 9: Missing Data	Item 10: Response Rates	Item 11: Follow-up	Total Items Reported (Max. 11)	
Acerini (1998)	yes	no	yes	no	can't tell	no	no	no	yes	yes	yes	**5**	(+) reported
Ackerman (1996)	can't tell	no	yes	can't tell	no	no	no	no	no	yes	yes	**3**	(+) reported
Agardh (2001)	yes	can't tell	no	can't tell	no	no	yes	no	yes	can't tell	no	**3**	(+) reported
Aktay (2001)	yes	no	yes	no	no	no	no	no	no	no	no	**2**	(+) reported
Annibale (2001)	yes	yes	yes	yes	can't tell	can't tell	yes	no	no	yes	no	**6**	(+) reported
Annibale (2003)	yes	yes	yes	yes	no	no	yes	can't tell	no	yes	no	**6**	(+) reported
Arato (2002)	yes	yes	yes	yes	no	no	no	can't tell	no	can't tell	no	**4**	(+) reported
Bao (1999)	yes	no	no	can't tell	no	no	no	no	no	can't tell	no	**1**	(+) reported
Bardella (1991)	yes	no	no	yes	can't tell	yes	no	no	no	yes	no	**4**	(+) reported
Bardella (2001)	yes	no	yes	yes	no	no	no	no	no	yes	no	**4**	(+) reported
Barera (1991)	can't tell	no	yes	can't tell	no	no	no	no	no	yes	no	**2**	(+) reported
Barera (2002)	no	no	yes	yes	no	no	yes	no	no	can't tell	yes	**4**	(+) reported
Bode (1996)	yes	yes	yes	no	can't tell	no	yes	no	no	yes	no	**5**	(+) reported
Bode (1993)	yes	yes	yes	yes	no	yes	can't tell	yes	can't tell	yes	yes	**8**	(+) reported
Book (2003)	yes	can't tell	no	no	no	no	can't tell	no	no	no	can't tell	**1**	(+) reported
Borch (2000)	yes	no	no	no	no	no	yes	can't tell	no	yes	can't tell	**3**	(+) reported
Calero (1996)	yes	no	yes	yes	no	yes	yes	yes	no	no	no	**6**	(+) reported
Carlsson (2001)	yes	yes	yes	no	no	no	no	yes	no	yes	yes	**6**	(+) reported
Carroccio (2002)	can't tell	no	yes	yes	yes	no	yes	yes	no	yes	no	**6**	(+) reported
Catassi (1996)	yes	no	yes	can't tell	no	yes	no	yes	no	yes	no	**5**	(+) reported
Catassi (2000)	yes	yes	yes	no	no	no	yes	yes	no	yes	no	**6**	(+) reported

Table 2: Celiac 2 prevalence & incidence studies (cross-sectional checklist) (cont'd)

Author (Year)	Item 1: Source of Information	Item 2: Inclusion/Exclusion Criteria	Item 3: Time Period for Identity	Item 4: Subjects consecutive	Item 5: Evaluators Masked	Item 6: Quality Assurance Assessments	Item 7: Patient Exclusions	Item 8: Confounding assessed/controlled	Item 9: Missing Data	Item 10: Response Rates	Item 11: Follow-up	Total Items Reported (Max. 11)	
Chan (2001)	yes	yes	yes	can't tell	yes	no	can't tell	no	no	can't tell	no	**4**	(+) reported
Chartrand (1997)	yes	can't tell	yes	can't tell	no	can't tell	no	no	no	yes	no	**3**	(+) reported
Collin (2002)	yes	can't tell	yes	can't tell	no	no	yes	yes	no	can't tell	no	**4**	(+) reported
Collin (1997)	yes	yes	yes	no	no	no	no	yes	no	can't tell	no	**4**	(+) reported
Corazza (1997)	no	no	no	no	no	no	no	no	no	can't tell	no	**0**	(+) reported
Corazza (1995)	no	no	no	yes	no	no	no	no	no	yes	no	**2**	(+) reported
Corazza (1992)	no	no	no	can't tell	no	no	no	no	no	yes	no	**1**	(+) reported
Corrao (1996)	yes	can't tell	yes	no	can't tell	yes	yes	no	no	yes	no	**5**	(+) reported
Cronin (1997)	yes	no	no	yes	no	no	no	no	no	yes	no	**3**	(+) reported
Csizmadia (1999)	yes	can't tell	yes	no	can't tell	no	can't tell	no	no	yes	yes	**4**	(+) reported
Day (2000)	yes	yes	yes	no	can't tell	no	yes	no	no	no	no	**4**	(+) reported
De Block (2001)	yes	no	no	can't tell	no	no	no	no	no	can't tell	no	**1**	(+) reported
De, Vitis (1996)	no	no	no	no	no	no	no	no	no	yes	no	**1**	(+) reported
Dickey (1997)	no	can't tell	no	can't tell	no	no	no	can't tell	no	yes	no	**1**	(+) reported
Dickey (1992)	yes	yes	no	can't tell	no	no	no	no	yes	no	no	**3**	(+) reported
Farre (1999)	yes	yes	yes	can't tell	no	no	no	yes	no	yes	no	**5**	(+) reported
Fasano (2003)	yes	yes	yes	can't tell	no	no	no	yes	no	yes	yes	**6**	(+) reported
Fitzpatrick (2001)	yes	can't tell	no	can't tell	no	no	yes	no	no	yes	no	**3**	(+) reported
Fraser-Rey0lds (1998)	can't tell	can't tell	yes	no	no	no	no	yes	no	yes	yes	**4**	(+) reported
Gillett (2001)	yes	can't tell	yes	no	yes	no	no	yes	no	yes	no	**5**	(+) reported
Gonzalez (2002)	can't tell	can't tell	no	yes	yes	no	can't tell	yes	no	yes	no	**4**	(+) reported
Green (2000)	yes	no	yes	yes	yes	yes	no	can't tell	no	yes	yes	**7**	(+) reported
Grodzinsky (1996)	yes	no	yes	no	no	yes	no	no	no	no	no	**3**	(+) reported

Table 2: Celiac 2 prevalence & incidence studies (cross-sectional checklist) (cont'd)

Author (Year)	Item 1: Source of Information	Item 2: Inclusion/Exclusion Criteria	Item 3: Time Period for Identity	Item 4: Subjects consecutive	Item 5: Evaluators Masked	Item 6: Quality Assurance Assessments	Item 7: Patient Exclusions	Item 8: Confounding assessed/controlled	Item 9: Missing Data	Item 10: Response Rates	Item 11: Follow-up	Total Items Reported (Max. 11)	
Hansen (2001)	no	can't tell	no	yes	no	no	no	no	no	no	no	**1**	(+) reported
Hawkes (2000)	yes	can't tell	yes	no	no	no	no	no	no	no	no	**2**	(+) reported
Hill (2000)	yes	no	no	no	no	no	no	yes	no	yes	yes	**4**	(+) reported
Hin (1999)	yes	yes	yes	yes	can't tell	yes	no	can't tell	no	yes	no	**6**	(+) reported
Hoffenberg (2003)	yes	yes	yes	can't tell	yes	no	can't tell	yes	no	yes	yes	**7**	(+) reported
Hogberg (2003)	yes	no	yes	no	no	no	can't tell	no	no	yes	yes	**4**	(+) reported
Holm (1993)	can't tell	no	no	can't tell	no	no	no	can't tell	no	yes	no	**1**	(+) reported
Hovdenak (1999)	no	no	no	no	no	no	no	no	no	yes	no	**1**	(+) reported
Howard (2002)	yes	yes	yes	can't tell	can't tell	no	yes	no	no	yes	no	**5**	(+) reported
Ivarsson (1999)	yes	can't tell	yes	yes	no	no	no	yes	no	yes	no	**5**	(+) reported
Ivarsson (2003)	yes	yes	yes	yes	no	no	no	yes	no	yes	no	**6**	(+) reported
Jaeger (2001)	no	can't tell	no	no	no	no	no	can't tell	no	yes	no	**1**	(+) reported
Jansen Th (1993)	yes	yes	yes	no	no	can't tell	no	no	no	yes	no	**4**	(+) reported
Johnston (1998)	yes	can't tell	can't tell	can't tell	yes	no	yes	no	can't tell	yes	yes	**5**	(+) reported
Kaukinen (1999)	can't tell	no	yes	can't tell	no	no	no	no	no	yes	no	**2**	(+) reported
Kepczyk (1995)	yes	yes	yes	yes	can't tell	yes	no	can't tell	no	yes	yes	**7**	(+) reported
Kolho (1998)	yes	can't tell	yes	can't tell	no	no	no	no	no	yes	yes	**4**	(+) reported
Kordo0uri (2000)	no	no	no	no	no	no	yes	no	no	yes	no	**2**	(+) reported
Kotze (2001)	yes	no	no	no	can't tell	no	no	can't tell	can't tell	yes	no	**2**	(+) reported
Lampasona (1999)	no	no	no	no	no	no	no	no	no	can't tell	no	**0**	(+) reported
Li Voon Chong (2002)	no	no	yes	no	no	no	no	yes	no	can't tell	no	**2**	(+) reported
Lindh (1992)	no	no	no	yes	no	no	no	no	no	yes	no	**2**	(+) reported

Table 2: Celiac 2 prevalence & incidence studies (cross-sectional checklist) (cont'd)

Author (Year)	Item 1: Source of Information	Item 2: Inclusion/Exclusion Criteria	Item 3: Time Period for Identity	Item 4: Subjects consecutive	Item 5: Evaluators Masked	Item 6: Quality Assurance Assessments	Item 7: Patient Exclusions	Item 8: Confounding assessed/controlled	Item 9: Missing Data	Item 10: Response Rates	Item 11: Follow-up	Total Items Reported (Max. 11)	
Lopez-Rodriguez (2003)	yes	can't tell	yes	yes	no	yes	no	no	no	yes	no	**5**	(+) reported
Lorini (1996)	no	no	no	no	no	yes	no	no	no	yes	no	**2**	(+) reported
Magazzu (1994)	yes	can't tell	yes	no	can't tell	no	no	no	no	yes	no	**3**	(+) reported
Maki (1988)	yes	no	yes	yes	can't tell	no	no	no	no	no	no	**3**	(+) reported
Maki (2003)	yes	can't tell	yes	can't tell	no	no	yes	yes	no	yes	no	**5**	(+) reported
Mather (2001)	no	no	no	yes	no	no	can't tell	no	yes	yes	no	**3**	(+) reported
Mazzetti (1992)	yes	yes	no	no	no	no	no	no	no	no	no	**2**	(+) reported
McIntyre (1993)	yes	no	yes	yes	no	no	yes	no	no	yes	yes	**6**	(+) reported
Mustalahti (2002)	no	can't tell	no	can't tell	no	yes	yes	no	no	yes	no	**3**	(+) reported
Ot (1998)	can't tell	no	no	no	can't tell	no	no	yes	no	can't tell	no	**1**	(+) reported
Ot (2001)	yes	no	yes	yes	no	no	no	no	no	yes	no	**4**	(+) reported
Nuti (2001)	yes	yes	yes	yes	can't tell	yes	can't tell	no	can't tell	yes	yes	**7**	(+) reported
Oxentenko (2002)	yes	can't tell	yes	no	no	no	yes	can't tell	no	yes	no	**4**	(+) reported
Page (1994)	yes	yes	yes	yes	yes	no	yes	yes	no	no	yes	**8**	(+) reported
Pittschieler (1996)	yes	no	no	no	no	no	no	no	no	no	no	**1**	(+) reported
Pittschieler (2003)	yes	can't tell	yes	can't tell	no	no	can't tell	can't tell	no	can't tell	yes	**3**	(+) reported
Polvi (1996)	yes	yes	no	yes	no	no	yes	no	yes	yes	yes	**7**	(+) reported
Ransford (2002)	no	no	no	yes	no	no	yes	no	no	yes	no	**3**	(+) reported
Rensch (1996)	yes	yes	yes	yes	can't tell	yes	yes	no	yes	yes	no	**8**	(+) reported
Riestra (2000)	can't tell	can't tell	yes	no	no	yes	no	yes	no	yes	no	**4**	(+) reported
Robinson (1971)	yes	no	no	yes	no	no	no	no	no	no	no	**2**	(+) reported
Roldan (1998)	yes	no	yes	no	no	yes	no	no	no	no	no	**3**	(+) reported

Table 2: Celiac 2 prevalence & incidence studies (cross-sectional checklist) (cont'd)

Author (Year)	Item 1: Source of Information	Item 2: Inclusion/Exclusion Criteria	Item 3: Time Period for Identity	Item 4: Subjects consecutive	Item 5: Evaluators Masked	Item 6: Quality Assurance Assessments	Item 7: Patient Exclusions	Item 8: Confounding assessed/controlled	Item 9: Missing Data	Item 10: Response Rates	Item 11: Follow-up	Total Items Reported (Max. 11)	
Rolles (1974)	yes	yes	no	no	no	no	no	no	no	yes	can't tell	**3**	(+) reported
Rossi (1993)	yes	can't tell	yes	no	no	no	no	no	no	yes	no	**3**	(+) reported
Rostami (2000)	no	no	no	no	yes	no	no	no	no	yes	no	**2**	(+) reported
Rostami (1999)	yes	no	yes	no	no	no	no	no	no	yes	no	**3**	(+) reported
Rutz (2002)	can't tell	no	no	no	no	yes	no	yes	no	yes	yes	**4**	(+) reported
Sanders (2003)	yes	no	yes	no	no	no	no	yes	no	yes	yes	**5**	(+) reported
Sategna-Guidetti (1994)	yes	no	no	no	no	no	no	no	no	yes	no	**2**	(+) reported
Saukkonen (1996)	yes	no	yes	yes	no	yes	yes	can't tell	no	yes	yes	**7**	(+) reported
Schober (2000)	yes	no	no	no	no	no	no	yes	no	no	no	**2**	(+) reported
Sigurs (1993)	yes	yes	yes	no	no	no	yes	no	no	yes	no	**5**	(+) reported
Sjoberg (1999)	yes	can't tell	yes	yes	can't tell	no	can't tell	yes	no	yes	no	**5**	(+) reported
Sjoberg (1994)	yes	can't tell	yes	yes	no	no	yes	no	no	yes	no	**5**	(+) reported
Sjoberg (1998)	yes	no	yes	no	no	yes	yes	no	no	yes	no	**5**	(+) reported
Spiekerkoetter (2002)	no	no	yes	can't tell	can't tell	no	no	no	no	yes	yes	**3**	(+) reported
Stokes (1976)	yes	no	no	can't tell	no	no	can't tell	yes	no	yes	yes	**4**	(+) reported
Talal (1997)	no	yes	no	can't tell	no	no	yes	yes	no	yes	yes	**5**	(+) reported
Talley (1994)	yes	yes	yes	no	no	can't tell	yes	yes	no	yes	no	**6**	(+) reported
Thomas (1992)	yes	no	yes	can't tell	can't tell	can't tell	no	no	no	yes	no	**3**	(+) reported
Trevisiol (1999)	yes	can't tell	yes	can't tell	no	no	no	no	no	no	yes	**3**	(+) reported
Tursi (2003)	no	no	no	can't tell	yes	no	no	no	no	no	no	**1**	(+) reported

Table 2: Celiac 2 prevalence & incidence studies (cross-sectional checklist) (cont'd)

Author (Year)	Item 1: Source of Information	Item 2: Inclusion/Exclusion Criteria	Item 3: Time Period for Identity	Item 4: Subjects consecutive	Item 5: Evaluators Masked	Item 6: Quality Assurance Assessments	Item 7: Patient Exclusions	Item 8: Confounding assessed/controlled	Item 9: Missing Data	Item 10: Response Rates	Item 11: Follow-up	Total Items Reported (Max. 11)	
Unsworth (2000)	yes	can't tell	yes	can't tell	no	no	no	yes	no	yes	yes	**5**	(+) reported
Valerio (2002)	yes	can't tell	yes	can't tell	no	no	no	no	no	yes	no	**3**	(+) reported
Van Mook (2001)	yes	yes	no	no	can't tell	no	yes	no	no	yes	no	**4**	(+) reported
Ventura (2001)	can't tell	yes	can't tell	can't tell	no	no	yes	can't tell	no	yes	no	**3**	(+) reported
Vitoria (1994)	no	can't tell	no	can't tell	no	no	can't tell	no	no	can't tell	no	**0**	(+) reported
Vitoria (1998)	yes	no	no	no	no	no	no	no	no	no	yes	**2**	(+) reported
Volta (2001)	can't tell	yes	yes	no	no	no	no	no	no	yes	no	**3**	(+) reported
Weile (1993)	yes	yes	yes	no	no	no	no	no	no	yes	no	**4**	(+) reported
Weile (2001)	yes	no	no	yes	no	no	no	yes	no	yes	no	**4**	(+) reported
West (2003)	yes	can't tell	yes	no	no	no	yes	yes	no	yes	no	**5**	(+) reported
% Items (+) reported:	**71%**	**27%**	**60%**	**29%**	**8%**	**16%**	**28%**	**27%**	**5%**	**73%**	**25%**		

Table 3. Celiac 3 cohort studies (Ottawa-Newcastle Scale)

Author (Year)	Item 1: selection of exposed	Item 2: selection of non-exposed	Item 3: ascertainment of exposure	Item 4: outcome missing data at initiation	Item 5: control factors (2*'s)	Item 6: assessment of outcome	Item 7: adequacy of follow-up length	Item 8: accountability for follow-up	
Askling (2002)	1	1	1	1	2	1	1	1	9 (*) awarded
Collin (1996)	1	1	1	1	2	1	1	0	8 (*) awarded
Corrao (2001)	1	1	1	0	2	1	1	1	8 (*) awarded
Cottone (1999)	1	1	1	1	2	1	1	1	9 (*) awarded
Green (2003)	1	1	1	1	2	1	1	0	8 (*) awarded
Holmes (1989)	1	1	1	1	2	1	1	1	9 (*) awarded
Logan (1989)	1	1	1	0	2	1	1	1	8 (*) awarded
Nielsen (1985)	1	1	1	1	2	1	1	0	8 (*) awarded
Selby (1979)	1	1	1	1	2	1	1	0	8 (*) awarded
Item % (+) reported	**100%**	**100%**	**100%**	**78%**	**100%**	**100%**	**100%**	**56%**	

NOTE: a maximum of 9 stars* may be awarded per study

Table 4: Celiac 3 case-control study (Ottawa-Newcastle Scale)

Author (Year)	Item 1: case definition	Item 2: representativeness of cases	Item 3: selection of controls	Item 4: definition of controls	Item 5: control factors	Item 6: ascertainment of exposure	Item 7: method of ascertainment for cases and controls	Item 8: non-response rate	
Delco (1999)	1	1	1	1	2	0	1	0	7 (*) awarded

NOTE: a maximum of 9 stars* may be awarded per study

Table 5. Celiac 4 cohort studies (Ottawa-Newcastle Scale)

Author (Year)	Item 1: selection of exposed	Item 2: selection of non-exposed	Item 3: ascertainment of exposure	Item 4: outcome missing data at initiation	Item 5: control factors (2*s)	Item 6: assessment of outcome	Item 7: adequacy of follow-up length	Item 8: accountability for follow-up	
Amin (2002)	1	1	1	0	1	0	1	0	5 (*) awarded
Greco (1997)	1	1	0	0	1	1	1	0	5 (*) awarded
Harewood (2001)	1	1	0	0	1	0	1	1	5 (*) awarded
Holmes (1976)	1	1	1	1	1	0	1	0	6 (*) awarded
Johnston (1998)	1	1	1	1	2	1	0	1	8 (*) awarded
Poddar (2002)	1	1	1	1	0	0	1	0	5 (*) awarded
Item % (+) reported	100%	100%	70%	50%	50%	33%	83%	33%	

NOTE: a maximum of 9 stars* may be awarded per study

Table 6. Celiac 4 case-control study (Ottawa-Newcastle Scale)

Author (Year)	Item 1: case definition	Item 2: representativeness of cases	Item 3: selection of controls	Item 4: definition of controls	Item 5: control factors (2*'s)	Item 6: ascertainment of exposure	Item 7: method of ascertainment for cases and controls	Item 8: non-response rate	
Ciacci (1996)	1	0	1	1	0	1	1	0	4 (*) awarded
Fabiani (1996)	1	1	1	1	1	1	1	0	7 (*) awarded
Thomason (2003)	1	0	1	1	2	0	1	1	6 (*) awarded
Westman (1999)	1	0	1	1	2	0	1	0	5 (*) awarded
Item % (+) reported	100%	25%	100%	100%	62.5%	50%	100%	25%	

NOTE: a maximum of 9 stars* may be awarded per study

Table 7: Celiac 4—Quality assessment not applicable to those studies identified as 'Other' in design

Author (Year)
Annibale (2001)
Arato (2002)
Atkinson (1997)
Bai (1997)
Bardella (2000)
Bardella (1985)
Barera (2000)
Boersma (2002)
Corrao (2001)
Fabiani (2000)
Kemppainen (1999)
Kemppainen(1998)
Mora (2001)
Mustalahti (1999)
Rea (1996)
Sategna-Guidetti (2000)
Sategna-Guidetti (2001)
Saukkonen (2002)
Valdimarsson (2000)
Zaccari (1996)

Table 8: Celiac 4—Quality assessment pending

Author (Year)
Smecuol (1997)
Valdimarsson (1996)

Table 9: Celiac 5 case-control studies (Ottawa-Newcastle Scale)

Author (Year)	Item 1: case definition	Item 2: representativeness of cases	Item 3: selection of controls	Item 4: definition of controls	Item 5: control factors (2*'s)	Item 6: ascertainment of exposure	Item 7: method of ascertainment for cases and controls	Item 8: Non-response rate	
Bardella (2001)	1	1	1	1	1	1	1	1	8 (*) awarded
Fabiani (2001)	1	1	1	0	1	0	1	1	6 (*) awarded
Anson (1990)	1	1	1	0	1	0	1	1	6 (*) awarded
Fabiani (2000)	1	1	1	1	1	0	1	1	7 (*) awarded
Item % (+) reported	100%	100%	100%	50%	50%	25%	100%	100%	

NOTE: a maximum of 9 stars* may be awarded per study

Table 10. Celiac 5 cohort studies (Ottawa-Newcastle Scale)

Author (Year)	Item 1: Selection of exposed	Item 2: selection of non-exposed	Item 3: ascertainment of exposure	Item 4: outcome missing data at initiation	Item 5: control factors (2*'s)	Item 6: assessment of outcome	Item 7: adequacy of follow-up length	Item 8: accountability for follow-up	
McNicholl (1976)	0	1	1	0	1	1	1	1	6 (*) awarded
Pacht (1995)	1	1	0	0	1	1	1	1	6 (*) awarded
Sategna-Guidetti (1996)	1	1	1	1	0	1	1	1	7 (*) awarded
Troncone (1995)	1	1	1	0	0	1	1	1	6 (*) awarded
Hogberg (2003)	1	1	1	1	1	1	1	1	8 (*) awarded
Fabiani (1996)	1	1	1	1	1	0	1	1	7 (*) awarded
Item % (+) reported	83%	100%	83%	50%	33%	83%	100%	100%	

NOTE: a maximum of 9 stars* may be awarded per study

Table 11. Celiac 5 non-comparative case series checklist

Author (Year)	Item 1: Intervention	Item 2: Inclusion/exclusion criteria	Item 3: follow-up as an inclusion	Item 4: sample size determination	Irem 5: sample size calculations	Item 6: method & length/accumulation of cases	Item 7: sources of participants	Item 8: method of outcome assessments	Item: 9: blinding	Item 9: primary and secondary measures	Item 11: timing of outcome assessment	Item 12: follow-up schedule	Item 13: maintaining follow-up	Item 14: compliance with follow-up	Item 15: method of data collection	Item 16: exclusions	Item 17: statistical approach for analysis	Item 18: missing data	Item 19: adverse events	Total # of Items = 19	
Burgin-Wolff (1991)	yes	yes	no	can't tell	no	no	yes	yes	no	yes	can't tell	can't tell	yes	yes	yes	can't tell	yes	no	no	9	(+) reported
Burgin-Wolff (2002)	yes	yes	can't tell	can't tell	no	no	yes	yes	no	yes	yes	yes	no	no	yes	no	yes	can't tell	no	9	(+) reported
Dickey (2000)	yes	yes	no	yes	no	yes	yes	yes	no	yes	yes	yes	can't tell	yes	yes	yes	yes	yes	no	14	(+) reported
Fotoulaki (1999)	yes	yes	no	yes	no	yes	yes	yes	no	yes	yes	yes	no	no	yes	no	yes	no	no	11	(+) reported
Kaukinen (2002)	yes	yes	no	yes	no	no	no	yes	no	yes	yes	yes	no	no	yes	no	yes	no	no	9	(+) reported
Lee (2003)	yes	yes	no	yes	no	yes	yes	yes	no	yes	yes	yes	can't tell	yes	yes	yes	yes	yes	no	14	(+) reported
Martini (2002)	yes	yes	no	yes	no	yes	yes	yes	no	yes	yes	yes	can't tell	no	yes	no	yes	no	no	11	(+) reported
Scalici (2003)	yes	yes	can't tell	can't tell	no	no	can't tell	can't tell	yes	yes	yes	yes	can't tell	can't tell	yes	can't tell	yes	no	no	8	(+) reported
Selby (1999)	yes	yes	no	yes	no	yes	yes	yes	no	yes	yes	yes	can't tell	yes	yes	can't tell	yes	no	no	12	(+) reported

Table 11. Celiac 5 non-comparative case Series checklist (cont'd)

Author (Year)	Item 1: Intervention	Item 2: Inclusion/exclusion criteria	Item 3: follow-up as an inclusion	Item 4: sample size determination	Irem 5: sample size calculations	Item 6: method & length/accumulation of cases	Item 7: sources of participants	Item 8: method of outcome assessments	Item: 9: blinding	Item 9: primary and secondary measures	Item 11: timing of outcome assessment	Item 12: follow-up schedule	Item 13: maintaining follow-up	Item 14: compliance with follow-up	Item 15: method of data collection	Item 16: exclusions	Item 17: statistical approach for analysis	Item 18: missing data	Item 19: adverse events	Total # of Items = 19	
Valentini (1994)	yes	yes	no	no	no	no	can't tell	yes	no	yes	yes	yes	no	no	yes	no	yes	no	no	7	(+) reported
Valletta (1990)	yes	yes	no	no	no	no	no	yes	no	yes	yes	yes	can't tell	no	yes	no	yes	no	no	8	(+) reported
% of items (+) reported	**100%**	**100%**	**0%**	**55%**	**0%**	**45%**	**64%**	**91%**	**9%**	**100%**	**91%**	**91%**	**9%**	**36%**	**100%**	**18%**	**100%**	**18%**	**0%**		

NOTE: yes = reported; no = not reported

Table 12: Celiac 5—Quality assessment not applicable to the following study designs

Author (Year)	Study type	
Jackson (1985)	questionnaire	
Lamontagne (2001)	questionnaire	
Ljungman (1993)	questionnaire	
Vahedi (2000)	abstract	

Table 13: Celiac 5—Quality assessment pending

Author (Year)
Baker (1975)
Bartholomeusz (1990)
Ciacci (2002)
Ciacci (2002)
Fabiani (1996)
Hogberg (2003)
Johnston (1998)
Kotze (2001)
Mayer (1991)
Skerritt (1991)
Vahedi (2003)
Volta (1990)

Appendix K. Additional Acknowledgements

The UO-EPC gratefully acknowledges the following individuals who served on our Technical Expert Panel (TEP). Acknowledgement does not reflect endorsement of this report.

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Appendix L. Listing of Excluded Studies

Objective 1 – Sensitivity and Specificity of Tests for CD

Abdulkarim A S, Murray J A. Review article: The diagnosis of coeliac disease. Alimentary Pharmacology & Therapeutics 2003;17(8):987-995. Not sensitivity or specificity of an identified test

Abdulkarim Ahmad S, Burgart Lawrence J, See Jacalyn et al. Etiology of nonresponsive celiac disease: results of a systematic approach. American Journal of Gastroenterology 2002;97(8):2016-2021. Not sensitivity or specificity of an identified test

Abdullah A M. Aetiology of chronic diarrhoea in children: Experience at King Khalid University Hospital, Riyadh, Saudi Arabia. Ann Trop Paediatr 1994;14(2):111-117. Not sensitivity or specificity of an identified test

Abdullah A M, Elrab M G, Al Herbish A et al. Serum antigliadin antibody as a marker of coeliac disease in children with chronic diarrhoea in Saudi Arabia. Med Sci Res 1994;22(3):229Not sensitivity or specificity of an identified test

Abe K K, Michinaga I, Hiratsuka T et al. Association of DQB1(*)0302 alloantigens in Japanese pediatric patients with steroid-sensitive nephrotic syndrome. Nephron 1995;70(1):28-34. Not sensitivity or specificity of an identified test

Abele M, Burk K, Schols L et al. The aetiology of sporadic adult-onset ataxia. Brain 2002;125(Pt 5):961-968. Not sensitivity or specificity of an identified test

Abele M, Schols L, Schwartz S et al. Prevalence of antigliadin antibodies in ataxia patients. Neurology 2003;60(10):1674-1675. Not sensitivity or specificity of an identified test

Abenoza P, Lillemoe T. CD34 and factor XIIIa in the differential diagnosis of dermatofibroma and dermatofibrosarcoma protuberans. American Journal of Dermatopathology 1993;15(5):429-434. Not sensitivity or specificity of an identified test

Ablin R J, Whyard T C. Identification and possible biological relevance of spermatozoal transglutaminase. Experientia 1991;47(3):277-279. Not sensitivity or specificity of an identified test

Acerini C L, Ahmed M L, Ross K M et al. Coeliac disease in children and adolescents with IDDM: clinical characteristics and response to gluten-free diet. Diabetic Medicine - a Journal of the British Diabetic Association 1998;15(1):38-44. Not sensitivity or specificity of an identified test

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Adany R, Glukhova M A, Kabakov A Y et al. Characterisation of connective tissue cells containing factor XIII subunit a. Journal of Clinical Pathology 1988;41(1):49-56. Not sensitivity or specificity of an identified test

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Adleff V, Racz K, Szende B et al. Coexpression of p53 and tissue transglutaminase genes in human normal and pathologic adrenal tissues. Journal of Steroid Biochemistry and Molecular Biology 1998;66(1-2):27-33. Not sensitivity or specificity of an identified test

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Agardh Daniel, Borulf Stefan, Lernmark Ake et al. Tissue transglutaminase immunoglobulin isotypes in children with untreated and treated celiac disease. Journal of Pediatric Gastroenterology and Nutrition 2003;36(1):77-82. Improper control group

Agrawal S, Gupta A, Yachha S K et al. Association of human leucocyte-DR and DQ antigens in coeliac disease: a family study. Journal of Gastroenterology and Hepatology 2000;15(7):771-774. Improper control group

Agreus L, Svardsudd K, Tibblin G et al. Endomysium antibodies are superior to gliadin antibodies in screening for coeliac disease in patients presenting supposed functional gastrointestinal symptoms. Scandinavian Journal of Primary Health Care 2000;18(2):105-110. Improper control group

Aguirre J M, Rodriguez R, Oribe D et al. Dental enamel defects in celiac patients. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontics 1997;84(6):646-650. Not sensitivity or specificity of an identified test

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Ahvazi Bijan, Kim Hee, Chul Kee et al. Three-dimensional structure of the human transglutaminase 3 enzyme: binding of calcium ions changes structure for activation. Embo Journal 2002;21(9):2055-2067. Not sensitivity or specificity of an identified test

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Objective 2 – Prevalence and Incidence of CD

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Objective 5 – Promoting or Monitoring Adherence to a GFD

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