Pathways to Discovery: Multidisciplinary Translational Research

10th RCMI International Symposium

Oral Health Session

Can Socio-Economic Disparities Explain the Relations between Oral Inflammation and Cardiovascular Disease?

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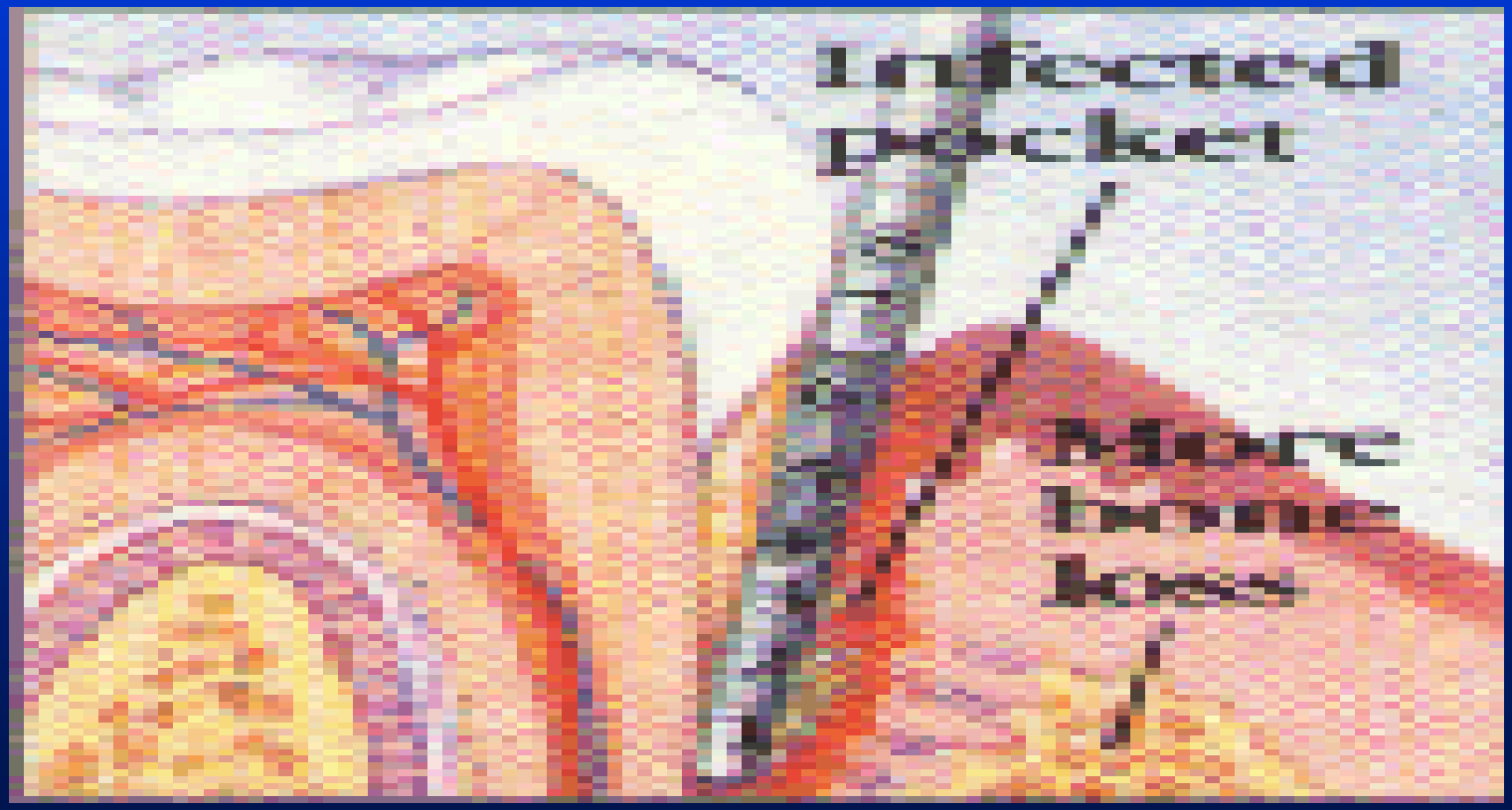
Cardiovascular Disease (CVD)

- Coronary heart disease (CHD)
- Peripheral arterial disease (PAD)
- Ischemic stroke
- Hemorrhagic stroke
- Congestive heart failure (CHF)
- Hypertension
- Sub-acute bacterial endocarditis

Periodontal Disease



Pathways Relating Oral-CVD Associations

Dental Exposures

Poor Oral Hygiene → Periodontal/Caries → Dental Procedures → Tooth loss
Microbes other Oral Infections

Possible Causal Pathways

Bacteremia

Psychosocial factors

Diet Nutrients Weight Change

Inflammation/Vascular Injury

Non-Causal Pathway

Confounding/Shared Factors:

- Age
- Smoking/Alcohol
- Health Behavior/Habits
- Genetic Pre-disposition
- Socio-economic status
- Stress, Obesity, Diet
- Physical Activity
- Access to Care
- Diabetes

Potential CVD Outcomes

- Sub-acute Bacterial Endocarditis
- Coronary Heart Disease
- Ischemic Stroke
- Hemorrhagic Stroke
- Peripheral Vascular Disease
- CVD Mortality

Adapted from: Compendium, 2000
Joshipura et. al.

Causal Inference Criteria

- **Strength of the association**
- **Dose-response relationship**
- **Time-sequence**
- **Consistency**
- **Independence from confounding***
- **Specificity**
- **Biologic Plausibility**

Adapted from Causal Criteria, Hill AB, 1966
****Compendium 2000: Joshipura, Ritchie & Douglass***

Strength of the Association

Stronger the association, more likely that the relationship is not due to chance or confounding

Periodontal Disease and CHD

prospective Studies	Number	Population	Years of Follow-up	Exposure	Outcome	RR
DeStefano*	10,000	NHANES	14	Perio. Disease	CHD	1.2
Joshipura	44,119	Health profess.	6	Perio. Disease	CHD	NS
Beck	1147	Veterans	18	Perio. Disease	CHD	1.5
Genco†	1372	Native Americans	10	Perio. Disease	CHD	NS
Morrison	10,368	Canadian	20	Perio. Disease	Fatal CHD	1.4
Hujoel	8,032	NHANES	21	Perio. Disease	CHD	NS
Howell	22,037	Physicians	12	Perio. Disease	CHD	NS
Tuominen	6,527	Finnish Registry	12	Perio. Disease	Fatal CHD	NS
Mattila	214	Finland	7	Perio. Disease	CHD	1.2

Subgroup analyses

DeStefano*	Men younger than 50	Perio. Disease	CHD	1.7
Genco†	Men younger than 60	Perio. Disease	CHD	2.7

NS - Not Significant

CHD Nested Case-Control Study

Multivariate RR (95% CI)		
	HPFS (M)	NHS (F)
Periodontal Disease History	1.00 (0.90-1.10)	0.99 (0.69-1.42)
Dental infection or swelling	1.02 (0.92-1.11)	3.91 (1.20-12.8)
Periodontal Bone Loss		
Mean bone loss score (cont.)	1.01 (0.69-1.50)	0.92 (0.55-1.54)
% of sites with bone loss ≥ 3 mm (cont.)	1.01 (0.31-3.27)	0.69 (0.10-4.65)
≥ 1 site with bone loss ≥ 5 mm (binary)	1.26 (0.63-2.50)	1.04 (0.41-2.63)
Extreme tertiles of % of sites with bone loss ≥3mm	1.04 (0.43-2.51)	0.77 (0.20-2.90)

CHD Nested Case-Control Study

	HPFS (M)	NHS (F)
Tooth loss due to periodontal disease	1.57(0.49-5.01)	7.32 (0.81-66.7)
Severe perio disease defined as ≥2 sites with ≥6mm bone loss	4.33(1.02-18.40)	0.35 (0.02-5.30)
Combined severe perio disease and/or tooth loss due to perio disease	2.22(0.81-6.05)	2.49 (0.52-11.9)

Second Coronary Heart Disease

Longitudinal Studies	Number	Population	Years of Follow-up	Exposure	Outcome	RR
Drangsholt	630	NHANES	12	Perio Disease	2nd MI	NS
Mattilla	214	Finland	7	Total Dental Index	2nd MI	p<0.05

Periodontal Disease and other CVD

prospective Studies	Number	Population	Years of Follow-up	Exposure	Outcome	RR
Mendez	1,110	Veterans	25	Perio. Disease	PAD	2.3
Hung	51,529	Health Prof.	12	Perio. Disease	PAD	1.4
Beck	1,147	Veterans	18	Perio. Disease	Total Stroke	2.8
Morrison	10,368	Canadian	20	Perio. Disease	Fatal stroke	1.6
Wu	9,962	NHANES	14	Perio. Disease	Ischemic Stroke	2.1
Howell	2653	Physicians	12	Perio. Disease	Non-Fatal Stroke	NS
Joshipura	51,529	Health Prof	12	Perio. Disease	Ischemic Stroke	1.3
Ajwani	364	Finnish	10	Perio. Disease	Fatal Stroke	2.0

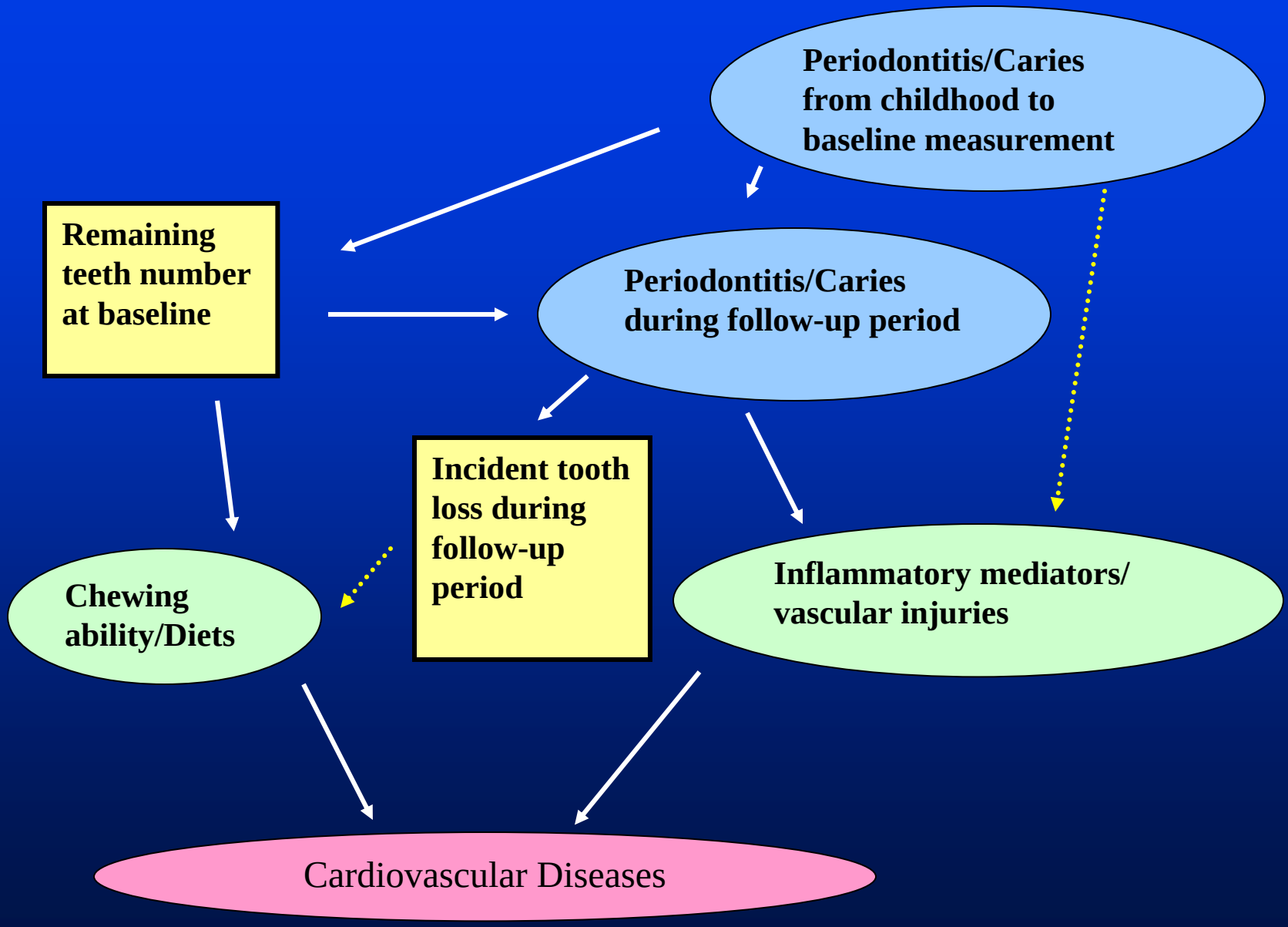
NS - Not Significant

Tooth Loss-CHD

prospective Studies	Number	Population	Exposure	Outcome	RR
Hung	41,407	HPFS	0-10 teeth	CHD	1.4
Hung	58,974	NHS	0-10 teeth	CHD	1.6
Howell	22,037	Physicians	Tooth loss	CHD	NS
Hujoel	4,027	NHANES	0 teeth	CHD	NS
Morrison	4,285	Canadian	0 teeth	CHD	1.9
Subgroup analyses					
Joshipura	Men with perio. disease	HPFS	0-10 teeth	CHD	1.7

NS - Not Significant

Earlier vs. Recent Tooth Loss



Association between number of teeth and CHD *(Hung et al. JPHD 2004)*

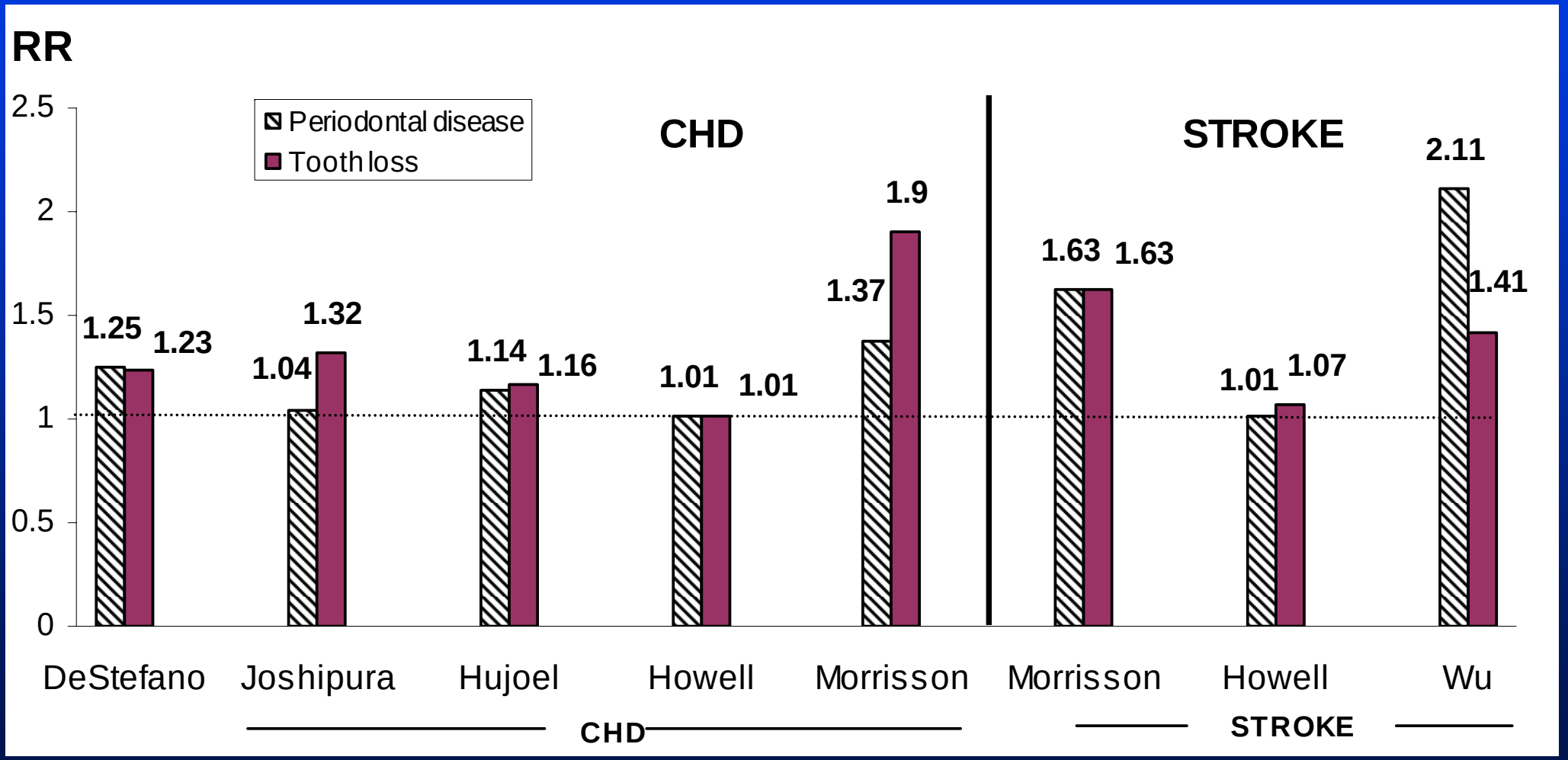
	CHD case number	Multivariate RR and 95% CI	CHD case number	Multivariate RR and 95% CI
	Men		Women	
Baseline number of teeth				
25-32	1222	1.00	241	1.00
17-24	254	1.10 (0.95-1.26)	123	1.14 (0.92-1.42)
11-16	71	1.35 (1.06-1.72)	43	1.34 (0.97-1.87)
0-10	107	1.36 (1.11-1.67)	137	1.64 (1.31-2.05)
Incident Tooth loss				
Incidence in past two years	128	0.86 (0.72-1.04)		
Cumulative incidence	258	0.94 (0.82-1.09)		

Tooth Loss-Other CVD

prospective Studies	Number	Population	Years of Follow-up	Exposure	Outcome	RR
Hung	45,136	Health Prof.	12	Recent tooth loss	PAD	1.4
Hung	45,136	Health Prof.	12	0 teeth	PAD	NS
Morrison	10,120	Canadian	20	0 teeth	Fatal stroke	1.6
Wu	9,962	NHANES	14	0 teeth	Ischemic Stroke	1.4
Howell	22,037	Physicians	12	Tooth loss	Non-Fatal Stroke	NS
Joshipura	44,116	Health Prof	12	0 - 24 teeth	Ischemic Stroke	1.6

NS - Not Significant

Adjusted relative risks for periodontal disease, tooth loss, and CVD by studies



Joshipura et al., JEBDP, 2002

Implications for Individuals and Clinicians Oral - CVD Associations Do Not Call For Extraction of Teeth *(JEBDP, 2002)*

- Presently, there is insufficient evidence that periodontal disease or tooth loss cause CVD
- People who lose teeth may change their diet and this may impact their systemic disease risk
- It is possible that extracting teeth may put some people at an additional risk for cardiovascular and other diseases
- There is no evidence that extraction of teeth, whether periodontally infected or not protect people from CVD
- Clinical decisions for extractions should not be based on a potential association between periodontal disease and CVD

Other Dental Exposures-CVD Cross-sectional Studies

Studies	Number	Population	Exposure	Outcome	RR
Grau	332	Hospitalized	TDI	Stroke	2.6
Jansson	1393	Swedish	OHS	Ischemic Stroke	2.7
Frisk	1056	Swedish	No. of RCT teeth	CHD	NS
Joshipura	34,683	HPFS	RCT	CHD	1.2
Joshipura	34,683	HPFS	Caries	CHD	NS
Caplan	6393	ARIC	≥2 RCT	CHD	1.3

TDI – Total Dental Index includes caries, periapical lesions, periodontitis, and other dental lesions

OHS – Oral Health Score includes a sum of scores for number of missing teeth, apical lesions, caries lesions and marginal bone loss

Endodontic Inflammation and Caries

Multivariate RR for CHD for ≥ 1 RCT vs. 0 RCT and Caries		
	Men	Women
Overall (RCT 1976-1998)	1.21 (1.04-1.40)	1.05 (0.46-2.44)
≤ 24 Teeth	1.19 (0.88-1.62)	1.61 (0.37-7.06)
> 24 Teeth	1.21 (1.03-1.43)	0.70 (0.05-9.92)
Only Dentists	1.38 (1.14-1.67)	-
Other Health Professionals	1.04 (0.83-1.30)	-
Caries (10+ vs. <10)	1.16 (0.89-1.51)	0.44 (0.11-1.78)

Results from Meta-analyses

Study	Type of Studies in Meta-analyses	Exposure	Outcome	RR (95% CI)
Muller, 2002	4 prospective	Perio.	CHD	1.12 (0.95-1.33)
	3 prospective	Perio.	Stroke	1.73 (0.89-3.34)
Janket, 2003*	9 prospective	Perio.	CHD/Stroke	1.19*(1.08-1.32)
	4 prospective	Perio.	CHD/Stroke (≤65 y)	1.44*(1.20-1.73)
	2 prospective	Perio.	Stroke	2.85*(1.78-4.56)
Khader, 2004*	6 prospective + 2	Perio.	CHD	1.15*(1.06-1.25)
	4 prospective + 2	Perio.	Stroke	1.13*(1.01-1.27)

Dose-Response Relationship

Risk increases as degree of exposure increases

Dose-Response Relationship

CHD

- **Beck, 1996 VADLS (prospective)**
  - **Increasing levels of bone loss was associated with higher risk of CHD/angina**
- **Geerts, 2004 (case-control)**
  - **Increasing scores of periodontal risk of infectiousness index (deep pockets and furcation involvement) had a significant dose-response relationship with CHD**

Stroke

- **Elter, 2003 ARIC (cross-sectional)**
  - **Increasing attachment loss was associated with increasing risk for stroke/TIA**
- **Beck, 1996 VADLS (prospective)**
  - **No consistent dose response for stroke with increasing levels of bone loss**

Time-Sequence

Exposure of interest (periodontal disease) precedes the outcome (CVD)

Time-Sequence

- **Results supported by several cohort studies where exposure clearly preceded outcome**
- **CHD – 3/9**
- **PAD – 2/2**
- **Stroke – 5/6**

Consistency

Similar results from number of studies by different investigators

Consistency

Study	RR	Study	RR
CHD		**PAD**	
Beck	1.5	Mendez	2.3
DeStefano	1.2	Hung	1.4
Genco	NS	**Stroke**	
Howell	NS	Beck	2.8
Hujoel	NS	Morrison	1.6
Joshipura	NS	Wu	2.1
Mattila	1.2	Joshipura	1.3
Tuominen, 2003	NS	Howell	NS
Morrison	1.4	Ajwani	2.0

Reasons for Inconsistency

- **Inconsistent results for perio - CHD associations can be explained by:**
  - **Chance variation**
  - **Limitations of measures in studies**
    - **Exposure measures**
    - **Outcome measures**
  - **Differences in population characteristics**
    - **Effects may vary by subgroups**

      e.g. effect may be present only among smokers or low SES groups
  - **Control of confounding**
    - **Confounding by SES and related factors eg. Smoking**

Effect Modification

- Smoking (may alternately indicate residual confounding by smoking)
- Age (higher association in younger but needs further confirmation)
- Socio-economic status (higher association among low SES groups)
- Other (no consistent pattern)

Independence from Confounding

The association is independent of confounders or known risk factors for the disease

Confounding

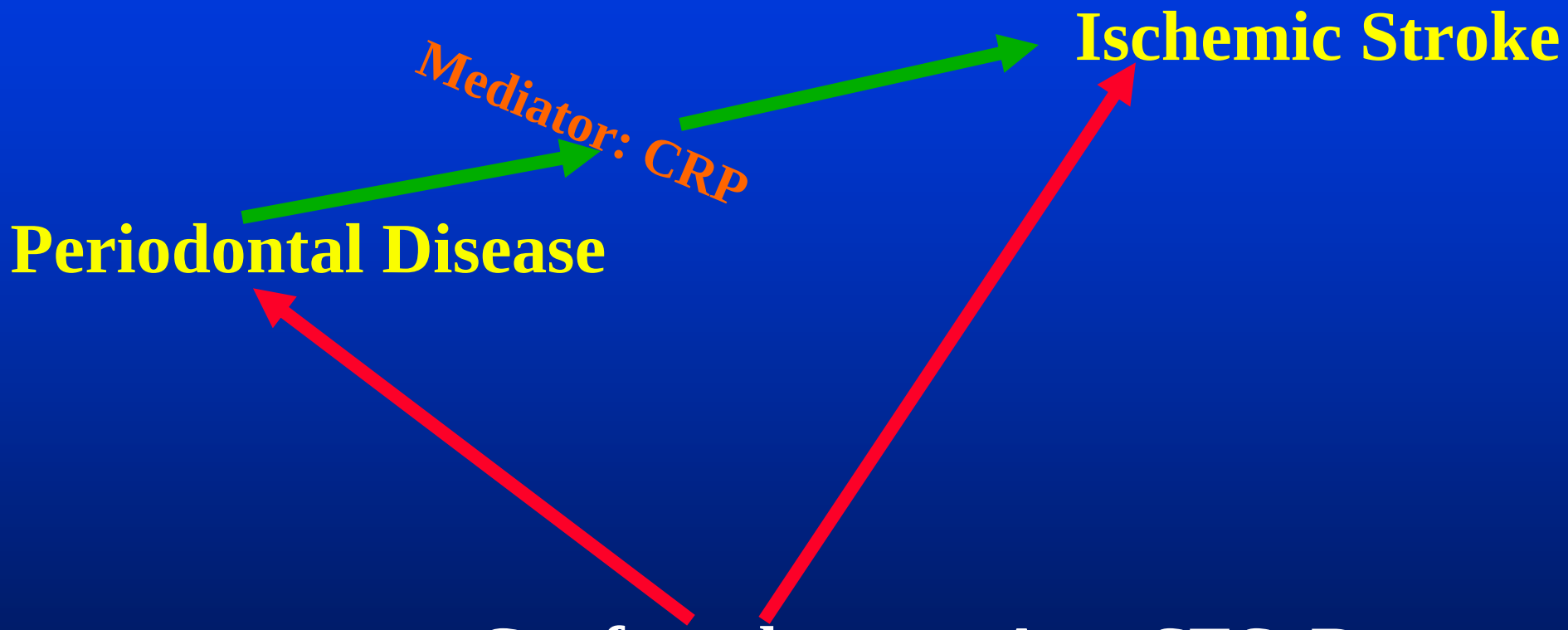
- **Confounders:** **Risk factors common to exposure and outcome leading to a deceptive observed association**
- **Common risk factors/confounders for oral-CVD:**
  - **Age**
  - **Smoking/Alcohol**
  - **Diabetes**
  - **Stress, Obesity, Diet**
  - **Socio-economic status**
  - **Physical Activity**
  - **Access to Care**
  - **Genetic predisposition**
- **Methods to control confounding:**

  Design (restriction, matching or randomization)

  Adjusted in the analyses (included in the model)

Confounding

A confounder is an extraneous factor that leads to an apparent association between the exposure and outcome that is different from the true association



Over-control = Controlling for Mediators

Residual Confounding

Perio-CHD Studies	Confounders Controlled		
	Smoking	Health Awareness	SES
Beck, 1996	No	No	No
DeStefano, 1993	No	No	No
Genco, 1997	No	No	No
Howell, 2001	Yes	Yes	Yes
Hujoel, 2000	Yes	No	Yes
Hujoel, 2001	Yes	No	No
Joshipura, 1996	Yes	Yes	Yes
Mattila, 1995	No	No	Yes
Morrison, 1999	Yes	No	No
Tuominen, 2003	Yes	No	No

Adapted from: Hujoel, 2002

SES as a confounding factor

- Socio-economic status is inversely related both with oral and cardiovascular health
- Socioeconomic status is complex and difficult to measure in its entirety
- Main variables: income and education
  - but need to also include culture, social capital and perceived social well being
- Provides knowledge of individual's environment within present SES as well as over the lifetime (including early childhood)

SES influence

- **SES entails the extent to which one:**

  -is aware of health

  -prioritizes health care

  -values preventive care and routine visits

- **Therefore, the impact of Socioeconomic status on the association between oral health chronic disease varies across populations and countries.**
- **Many published studies are confounded by these factors**
- **Association depends on the extent to which SES is measured and controlled**

SES and CVD

- People with lower education have about twice the risk for CVD (Marins 1996)
- Lower income groups also have a more adverse CVD risk factors profile
- Inverse relation between social capital and levels of total cholesterol, LDL, triglyceride, CRP, ICAM-1, fibrinogen, and homocysteine in women. (Albert, 2006)
- Strong associations between income and CVD (Toivanen, 2006)
- Individuals in the lowest income quartile had 3.6 (prevalence) and 2.1 (mortality) times higher risk of CVD compared to those in the highest income quartile (Toivanen, 2006)

SES and Oral Health

- **Tooth loss has consistently been shown to be strongly associated with SES**

  (Nikias, 1975; Haan, 1987; Caplan, 1993; Drury, 1999)

- **4.5% of high SES persons in NHANES III were edentulous vs. 32% of low SES persons, aged ≥35** *(Drury et al., 1995)*

Courtesy: Monik Jimenez

Race/Ethnicity and Oral Health

- **Blacks and Hispanics to exhibit the more periodontal disease, tooth loss and caries than Whites.**

  (Surgeon General Report on Oral Health, 2000)

- **Blacks are less likely to be offered an alternative to extraction.**

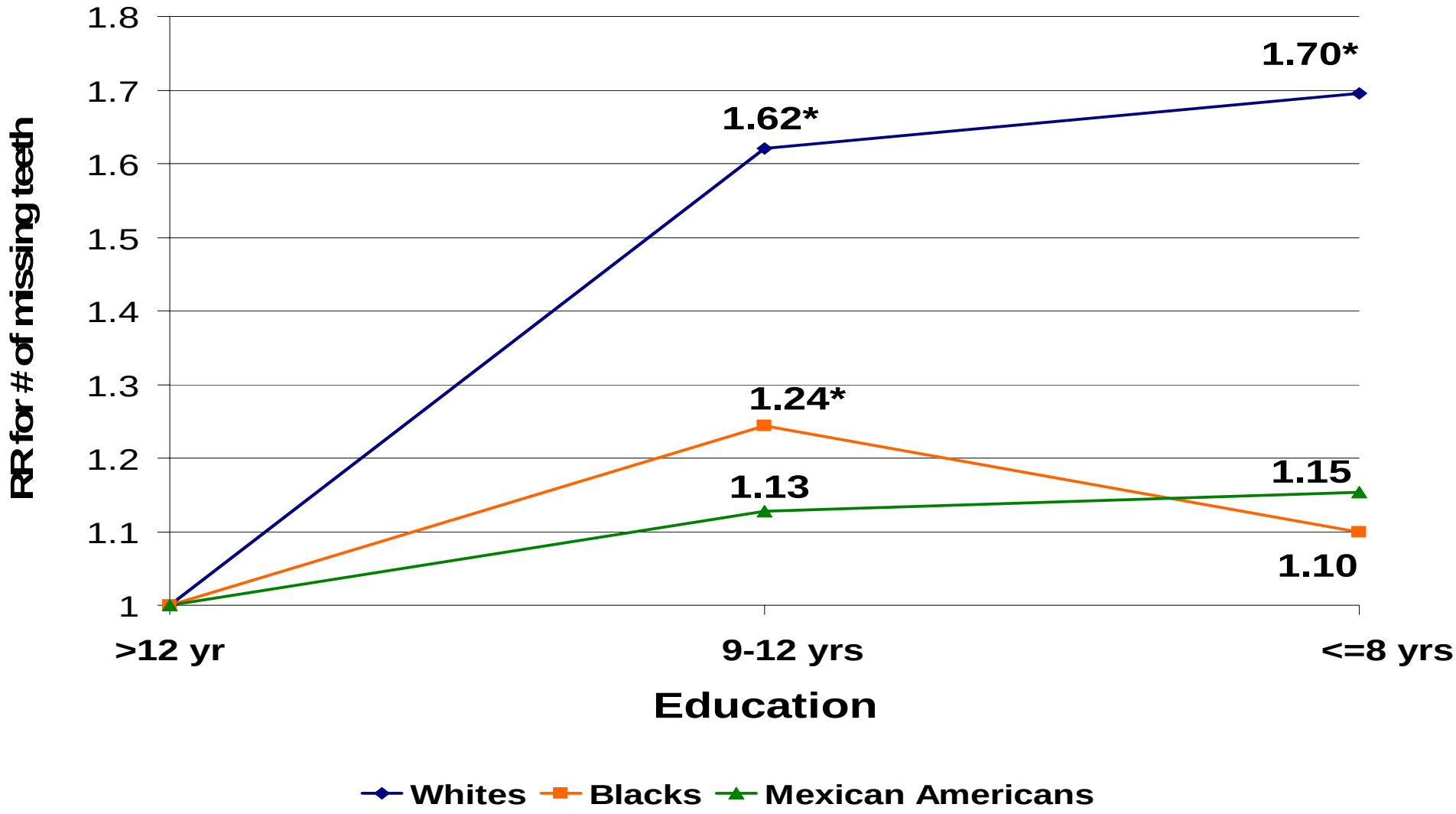
  (Gilbert, 2003)

- **Black dentists were 58% more likely to experience tooth loss over f/u than whites.**

  (Joshipura et al., unpublished)

Courtesy of Monik Jimenez

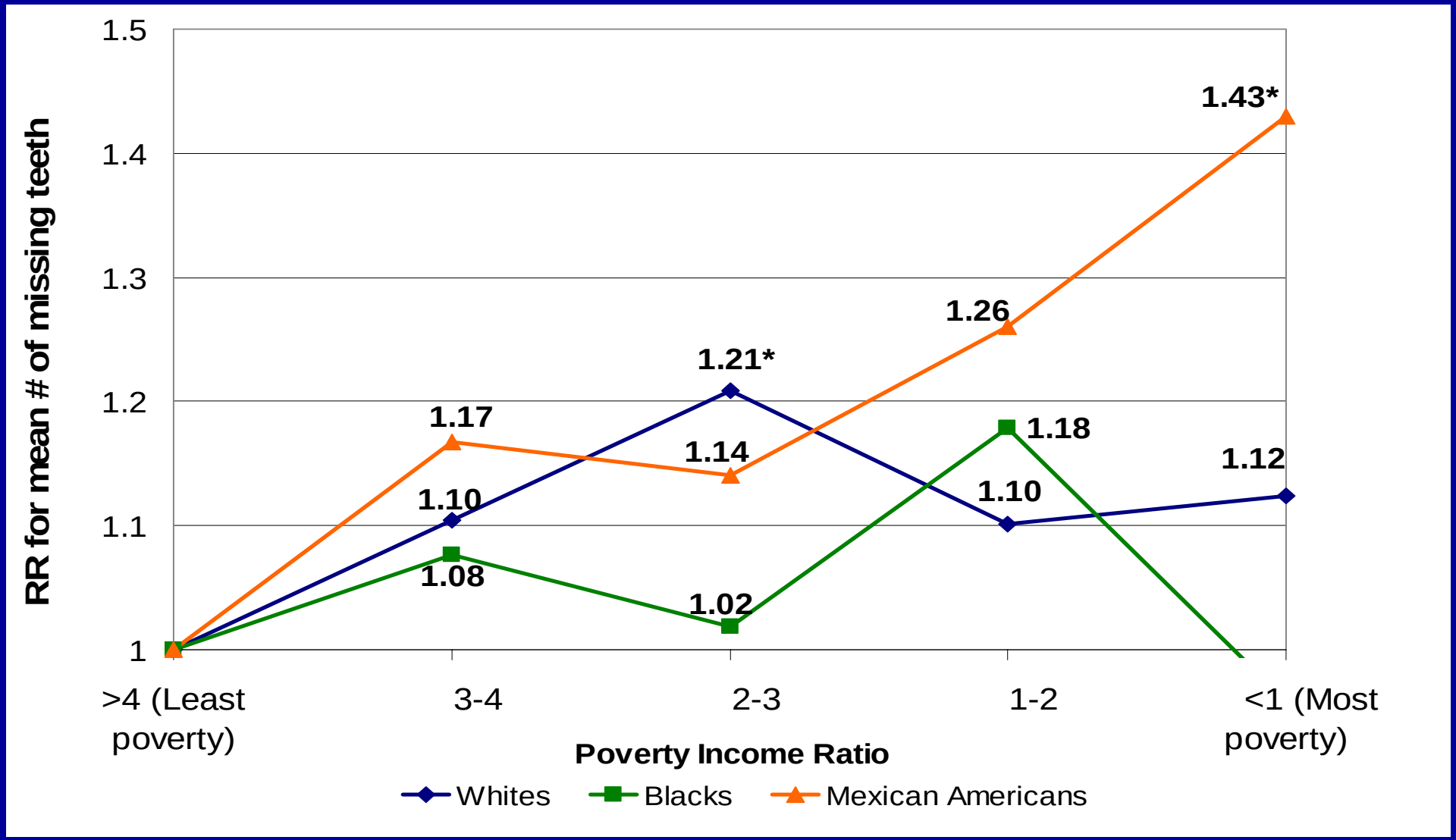
Association between Education & Missing Teeth by Race



*Adjusted for all other SES variables and gender.

* p-value<0.05

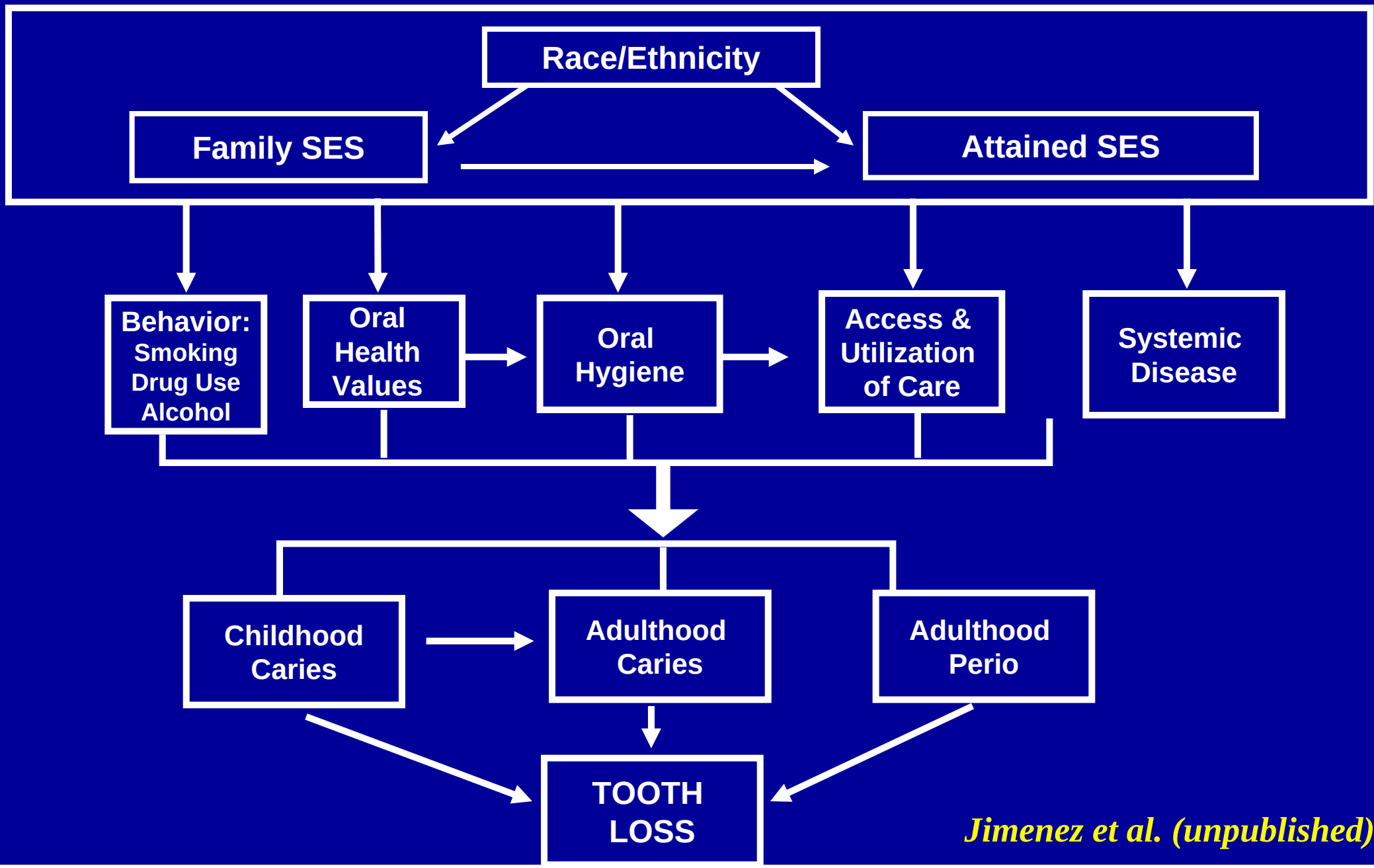
Association between PIR & Missing Teeth by Race



*All SES variables and gender mutually adjusted for one another.

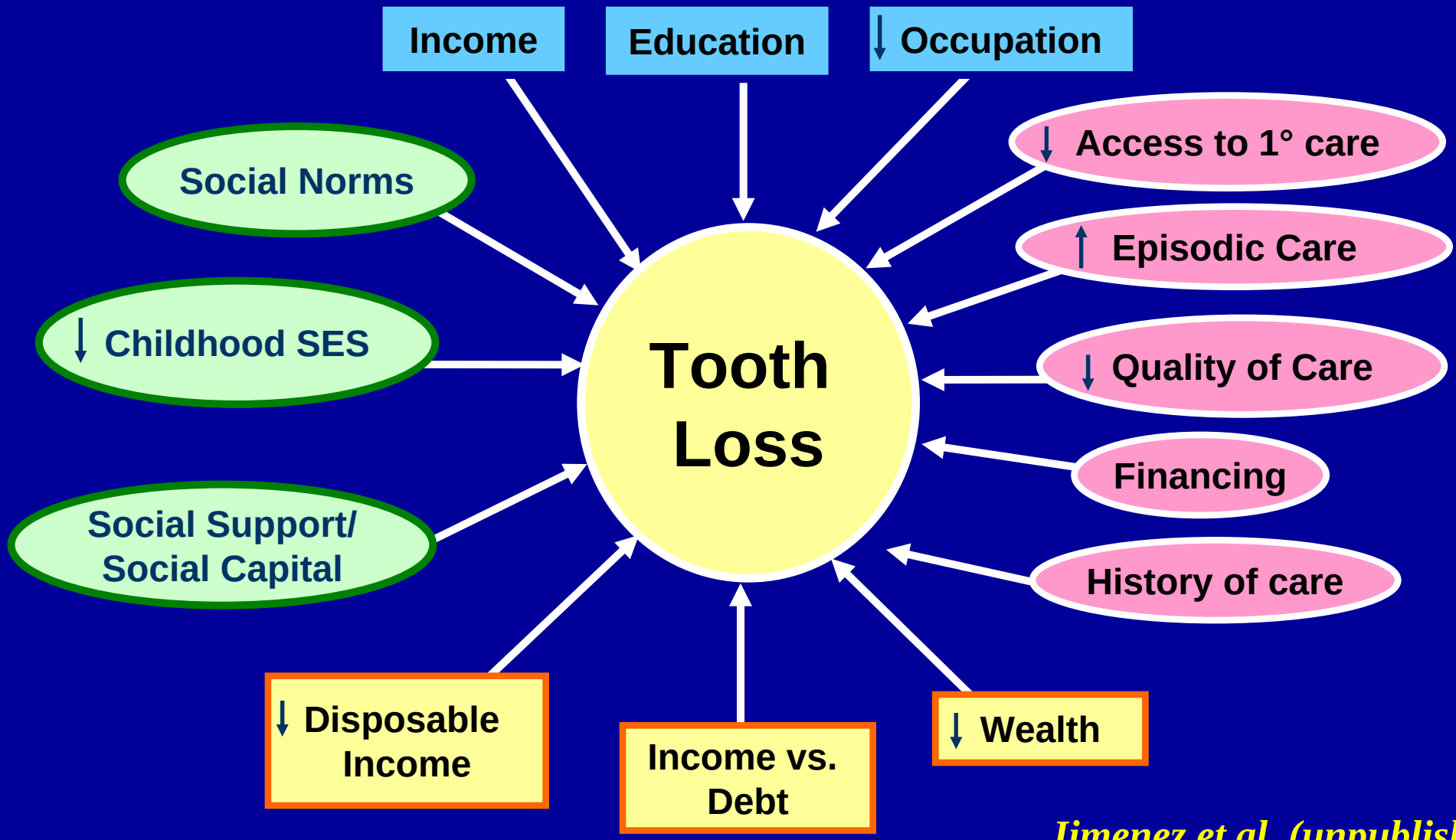
* p-value<0.05

Pathways for Race/Ethnicity & Tooth Loss



Jimenez et al. (unpublished)

Tooth Loss and SES



Jimenez et al. (unpublished)

SES and Oral Health by Race

***May not be true for all Racial/Ethnic Groups*:**

- **Low education and SEI associated with attachment loss in elderly Whites,**

(Elter 1999 et al., J Clin Perio)

- **Age 50+ NHANES III:**
- **Whites: ↓ perio ↑ education & income.**
- **Mexican Americans: no effect of income.**
- **Blacks: ↑ perio with ↑ income.**

(Borrell et al. 2004, AJPH)

Our Work on CVD

Health awareness and behavior are difficult to measure and control

Restriction to high SES group of health professionals provides a unique opportunity to overcome this concern

Our cohorts include:
52,529 males followed (Health Professionals Follow-up Study)
121,700 Nurses followed (Nurses Health Study)

Confounders adjusted: age, smoking, gender, SES, diabetes, alcohol, BMI, physical activity, family history of MI, multivitamin supplement use, vitamin E use, profession, history of hypertension & hypercholesterolemia

Summary of our work in HPFS (Males) and NHS (Females) relating perio and CVD

Diseases	Stroke (M)	PAD (M)	CHD (M)	CHD (F)
Baseline Teeth	Multivariate Relative Risks			
25-32	1.0	1.0	1.0	1.0
17-24	1.6*	1.2	1.1	1.1
11-16	1.8*	1.4	1.4*	1.3
0-10	1.8*	1.1	1.4*	1.6*
Periodontal Disease	1.3*	1.3*	1.0	1.0
Incident Tooth Loss				
During follow-up	1.3*	1.4*	0.9	-

Specificity

Specific agent produces a specific effect

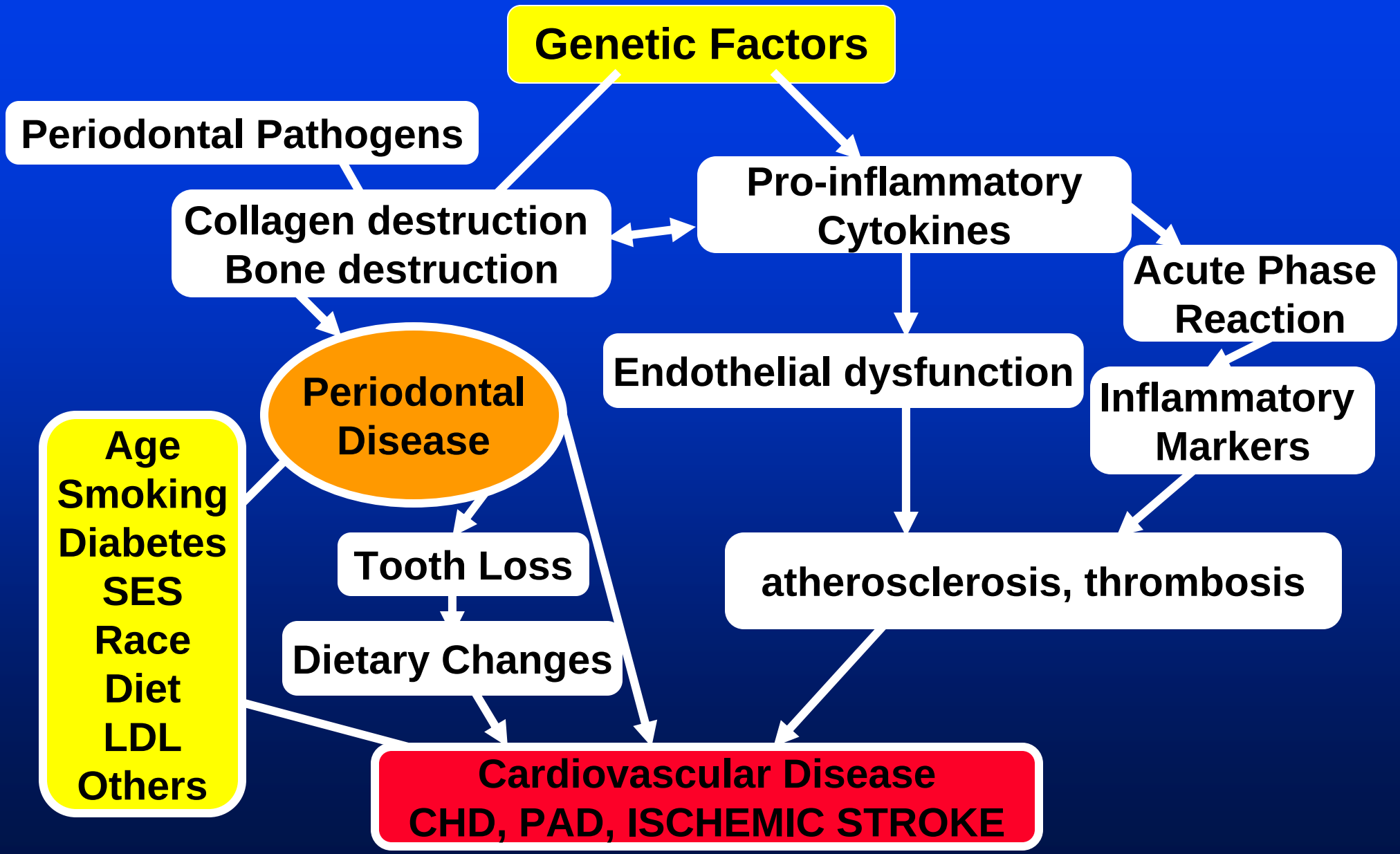
Specificity

- Established when a single putative cause produces a specific effect
- Specificity provides additional support for causality
- Absence of specificity (multiple causes) as in CVD does not negate a causal relationship

Biologic Plausibility

A known biological mechanism that could explain the association

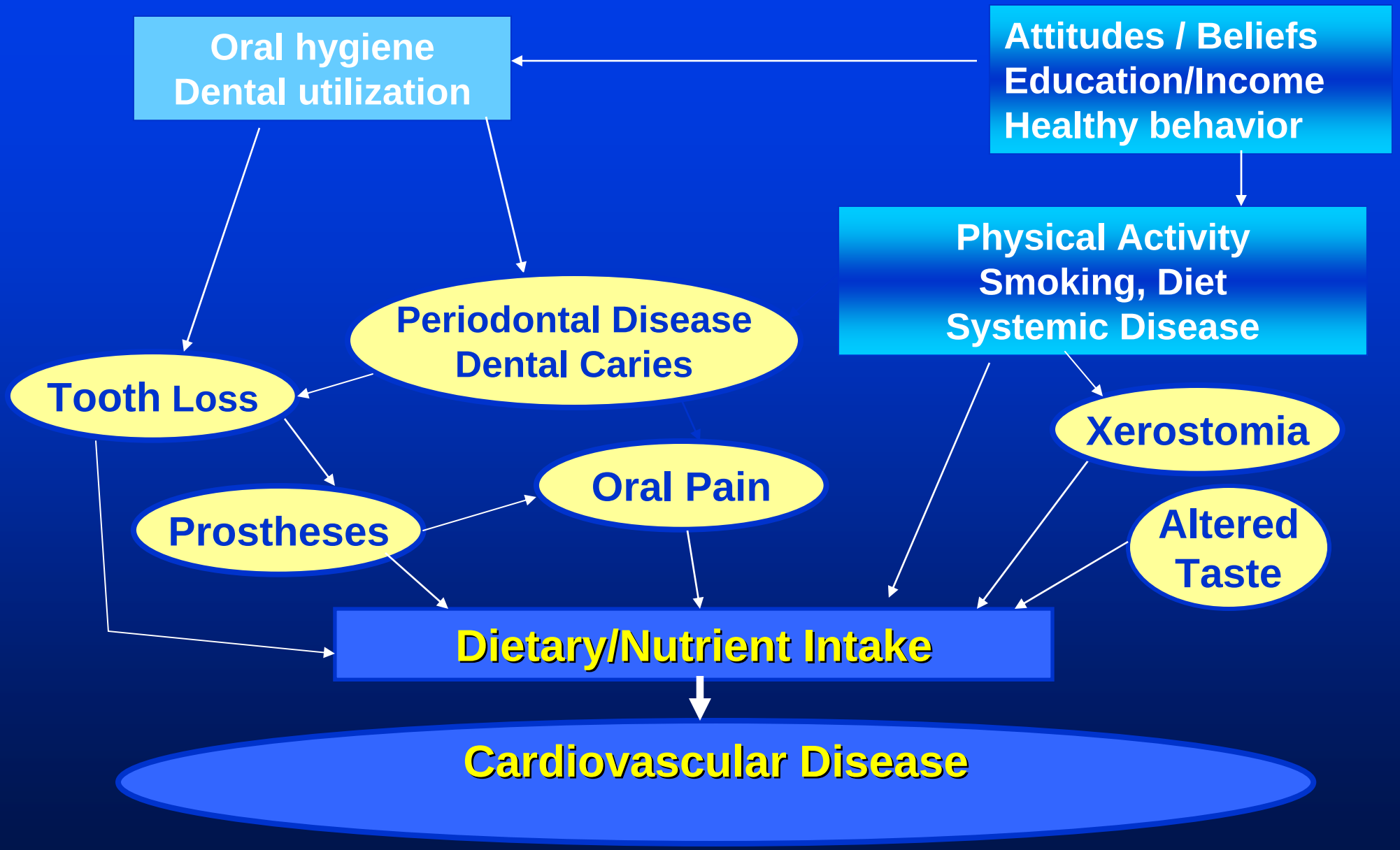
Biologic Plausibility



Role of Inflammation

- Periodontal disease leads to increased inflammatory cytokines which are associated with various inflammatory biomarkers.
- Previous studies show a relation between periodontal disease and CRP, fibrinogen, WBC, ICAM, LDL, triglycerides.
- Biomarkers of inflammation and dislipedemia may mediate Oral - CVD associations

Role of Nutrition



Overall Strength of Evidence

Satisfaction of Criteria for Causal Inference

Exposure	Periodontal Disease		
Systemic Outcome	Cardiovascular Disease		
	CHD	PAD	Ischemic Stroke
Specificity	-	-	-
Strength of Association	+	+	++
Dose-response	+	+	++
Time sequence	+	+	+
Biologic Plausibility	++	++	++
Consistency	-	+	++
Independence from confounding	+	++	++
Overall Consensus	Suggestive	Suggestive	Highly Suggestive

Updated from Joshipura, Ritchie & Douglass., Compendium, 2000

CVD Conclusions

- Common genetic factors need to be ruled out
- Additional well conducted prospective studies are needed
- Evidence linking periodontal disease and CHD is weaker and inconsistent
- If further studies show consistent associations after ruling out confounding, periodontal disease may be an independent risk factor for CVD
- Nutritional factors and markers of inflammation, hemostasis, dislipedemia and endothelial dysfunction markers may be some part of the pathway

CVD Future Directions

- Additional populations including developing countries
- Clinical trials where feasible but clinical trials only assess impact of periodontal treatment not periodontitis
- Additional longitudinal studies evaluating the association between oral conditions and ischemic stroke and PAD.
- Evaluate whether the association between oral conditions and cardiovascular disease may be explained by common genetic factors.
- Evaluate exposures, outcomes and mediators (inflammatory and hemostatic factors and serum lipids) in the same models .
- Determine the extent to which periodontal microorganisms are associated with cardiovascular disease.
- Evaluate additional CVD outcomes.

HPFS Methods

- 18,225 health professionals provided blood; Diet and life style factors similar between men who provided blood and those who did not
- Sample for the biomarker analyses was selected for a study on alcohol; stratified by alcohol intake
- Excluded 8922 men with missing data on diet, smoking, alcohol, and physical activity
- Excluded 208 men with CVD, diabetes, gastric or duodenal ulcer, and cancer except non-melanoma skin cancer

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HPFS Methods

- Final sample: 468 men, 47 to 83 years of age
- Cross-sectional study
- Analysis of Covariance: biomarkers outcome, periodontal disease predictor
- Periodontal disease history: ever report of professional diagnosis
- Adjusting for categorical variables for age, smoking, BMI, and alcohol intake

HPFS Multivariate Results

	No periodontal disease N =377	Periodontal disease N = 91	% difference	p-value
CRP	0.47	0.61	30	0.02*
Fibrinogen	286.5	288.4	1	0.61
Factor VII	99.7	101.0	1	0.36
IL -6	1.5	1.7	11.8	0.44
TNFR1	1129	1092	-3	0.22
TNFR2	1656	1610	-3	0.31
t-PA	15.9	17.6	11	0.001*
vWF	109.0	121.4	11	0.001*
LDL	108.5	120.0	11	0.001*
ApoB	117.6	129.0	9.7	0.002*
HDL	56.5	55.2	-2.4	0.68
Cholesterol	224.9	246.3	9.5	<0.001*

Joshipura et al, JDR 2004

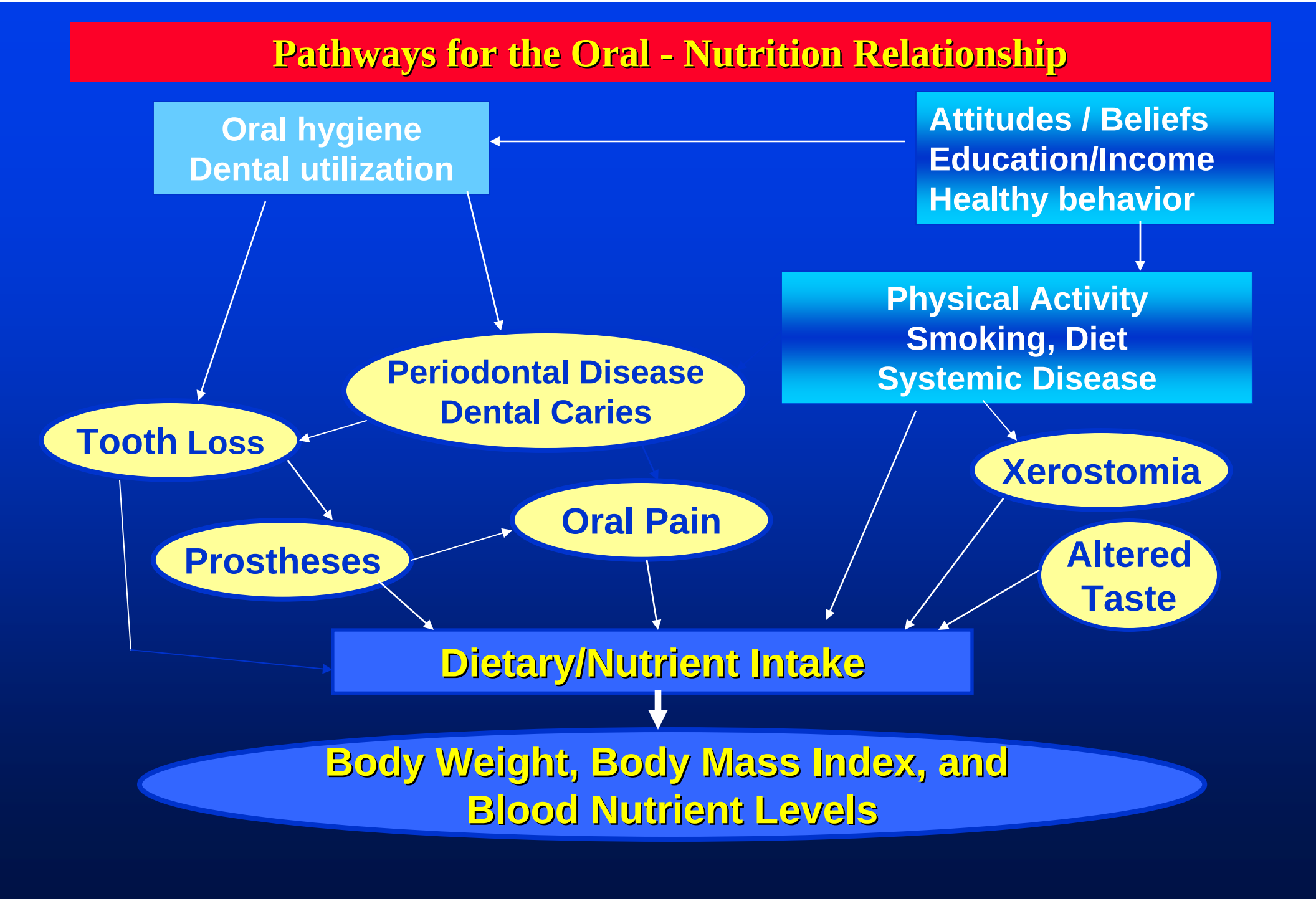
NHS Methods

- CHD nested case control study
- Linear Regression: biomarkers as outcome (log transformed)
- Periodontal disease as exposure: $\geq$ 1 site with bone loss $\geq$ 5 mm or women who lost one or more teeth to periodontal disease.

NHS Results

	No periodontal disease N =229	Periodontal disease N = 152	% difference	p-value
CRP	0.19	0.26	35.8	0.01*
ICAM	332	356	7.4	0.02*
VCAM	579	596	2.9	0.26
E-selectin	44	51	17.0	<0.001*
Fibrinogen	295	307	4.0	0.17
IL -6	1.7	1.7	1.5	0.84
TNFR1	1222	1258	2.9	0.40
TNFR2	2248	2369	5.4	0.07
LDL (mg/dl)	133	144	8.2	0.04

Adjusting for age, smoking, BMI, physical activity, aspirin use, alcohol use, CHD case-control status and diabetes


Pathways for the Oral - Nutrition Relationship
Oral hygiene
Dental utilization
Attitudes / Beliefs
Education/Income
Healthy behavior
Physical Activity
Smoking, Diet
Systemic Disease
Periodontal Disease
Dental Caries
Tooth Loss
Xerostomia
Oral Pain
Prostheses
Altered
Taste
Dietary/Nutrient Intake
Body Weight, Body Mass Index, and
Blood Nutrient Levels

The Role of Nutrition

Confounder:

- **Nutritional factors (alcohol, vitamin C, sugar, etc.) and body weight, may be related to oral conditions, and to CVD independently**

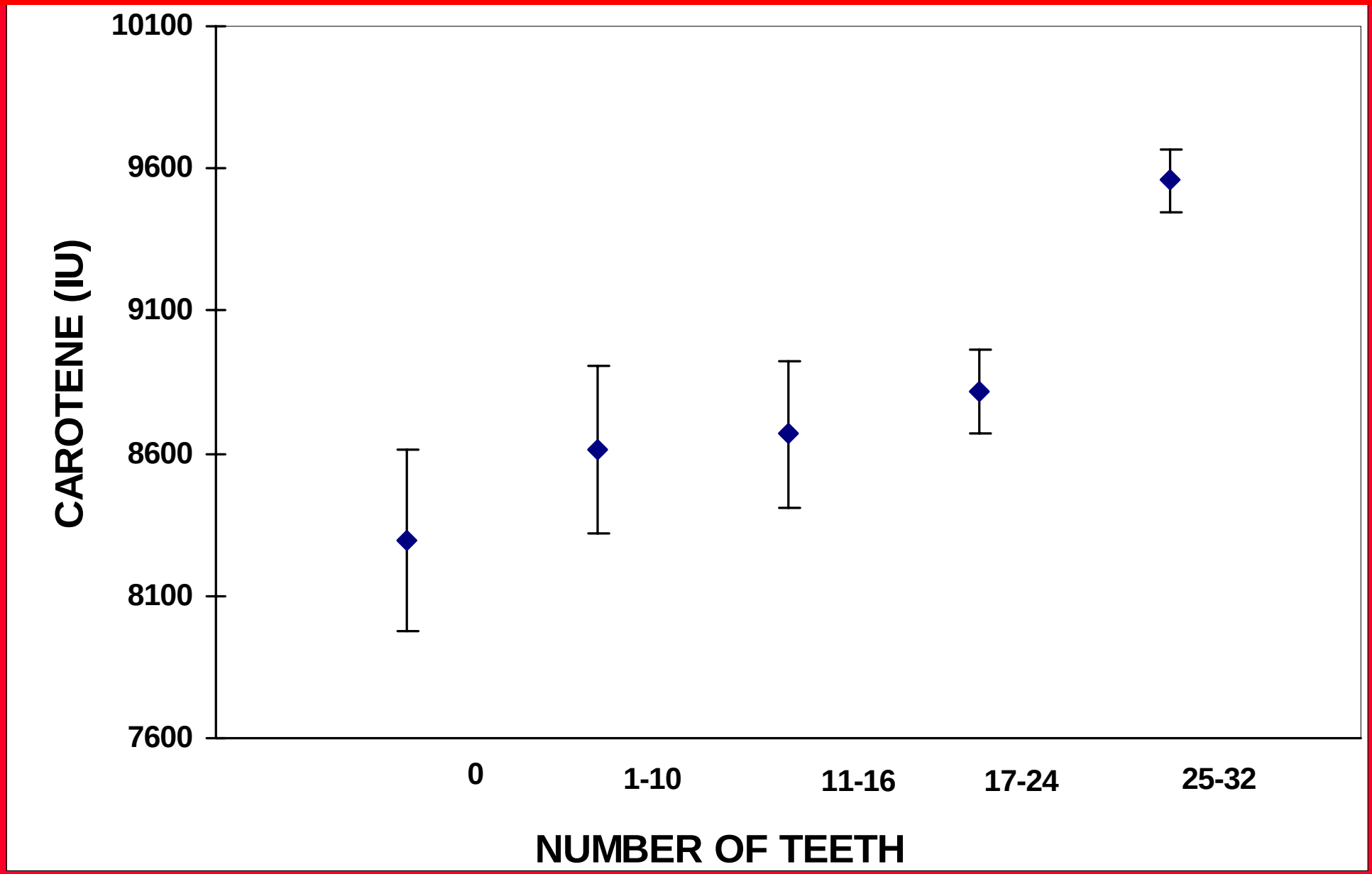
Mediator:

- **Tooth loss → reduced masticatory ability → altered food selection patterns → nutrient intake → CVD**

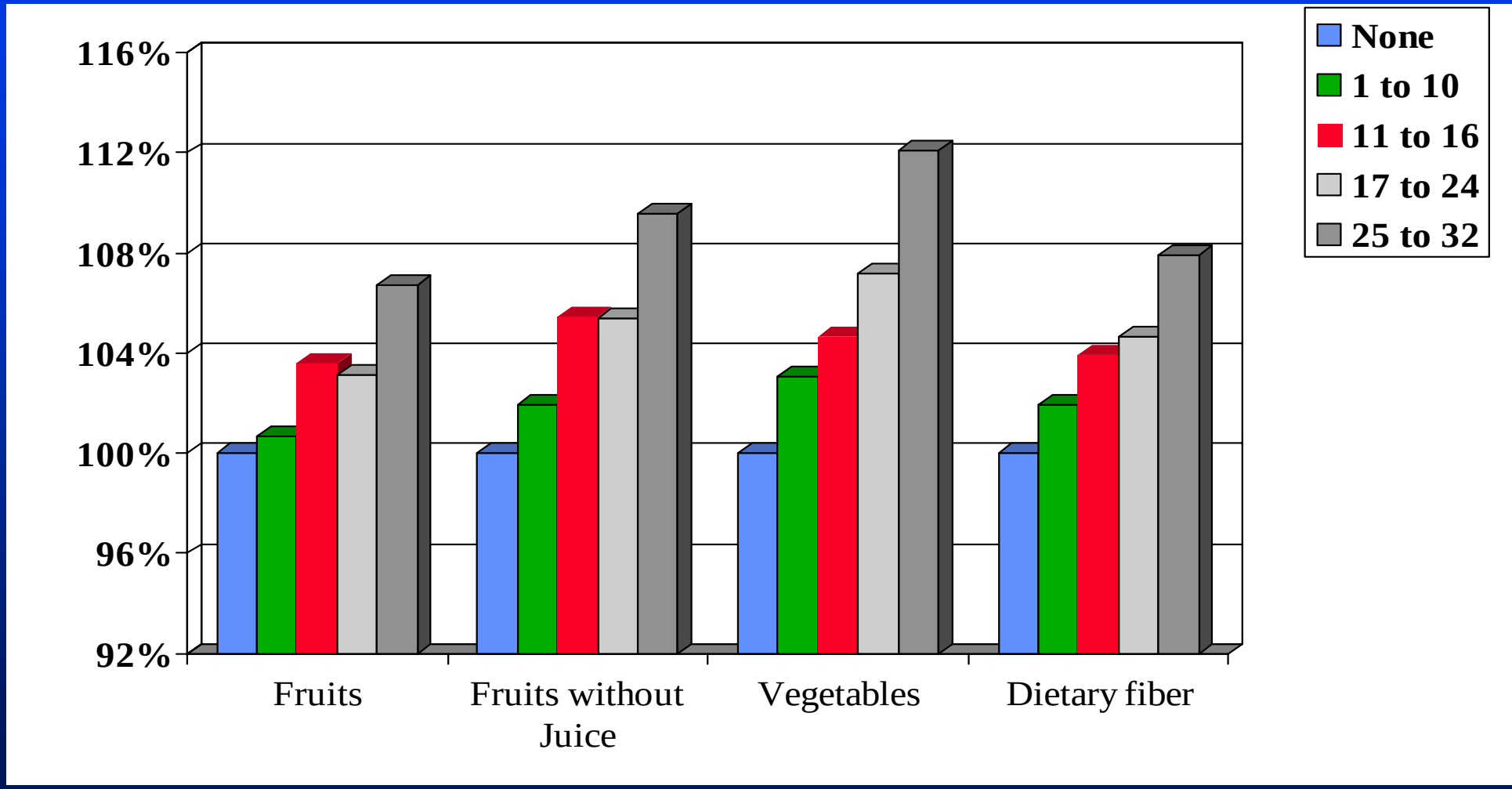
The Role of Nutrition

- Tooth loss impacts dietary quality and nutrient intake in a manner that may increase risk for several systemic disease
- Impaired dentition may be related to weight change depending on age and other population characteristics
- The impact of periodontal disease on nutritional intake and weight warrants further research
- Nutritional mediators may interfere with inflammatory cytokine activity, effect immune function and inflammatory markers, and may affect both oral health and systemic disease
- Nutrition may be a confounder of the perio - CVD relation

Tooth loss and Diet *JADA 1996*



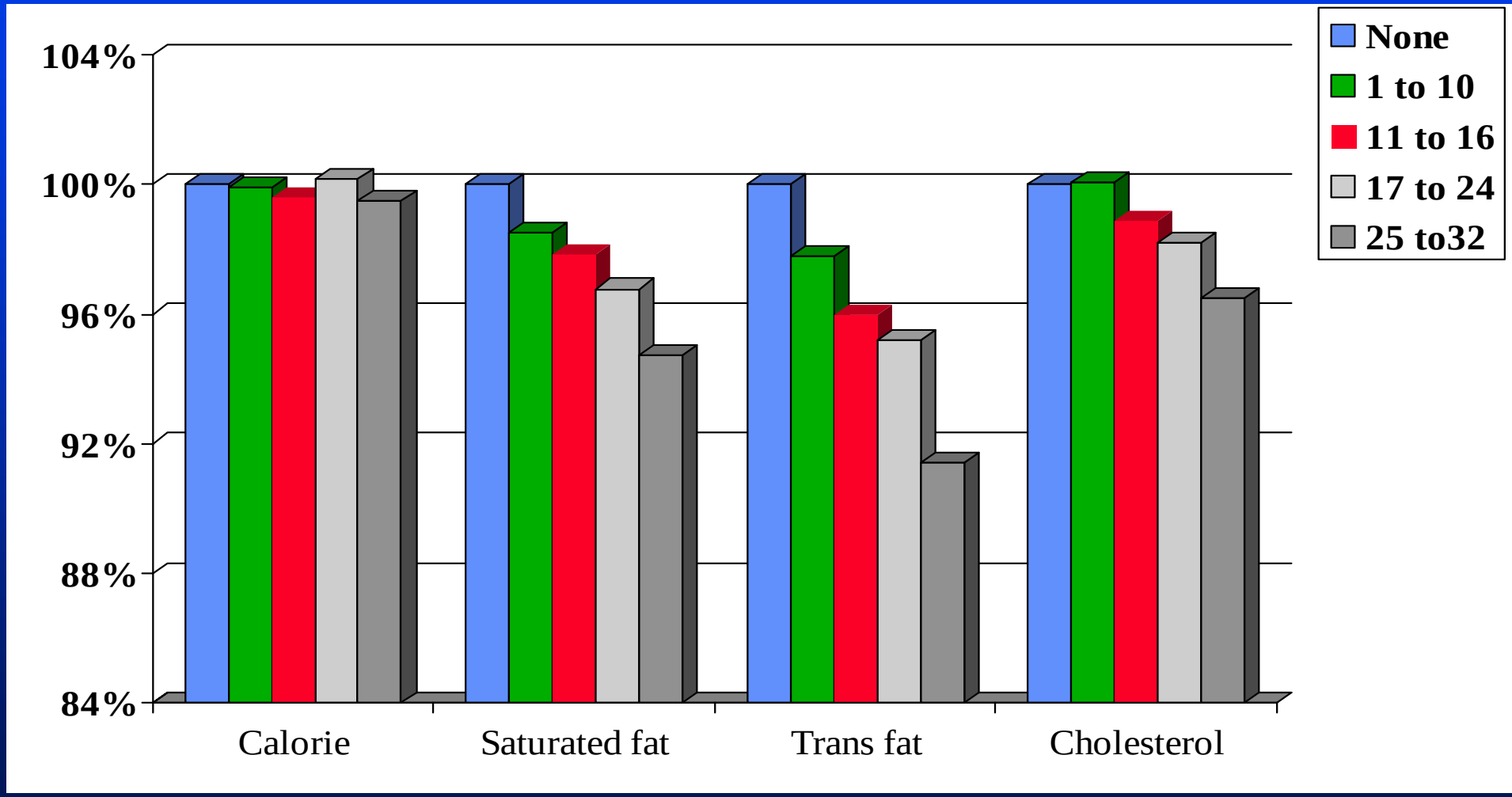
Percentage of Edentulous Subjects' Dietary Intake



(Adjusting Age, Smoking and Physical Activity)

Hung, CDOE In Press

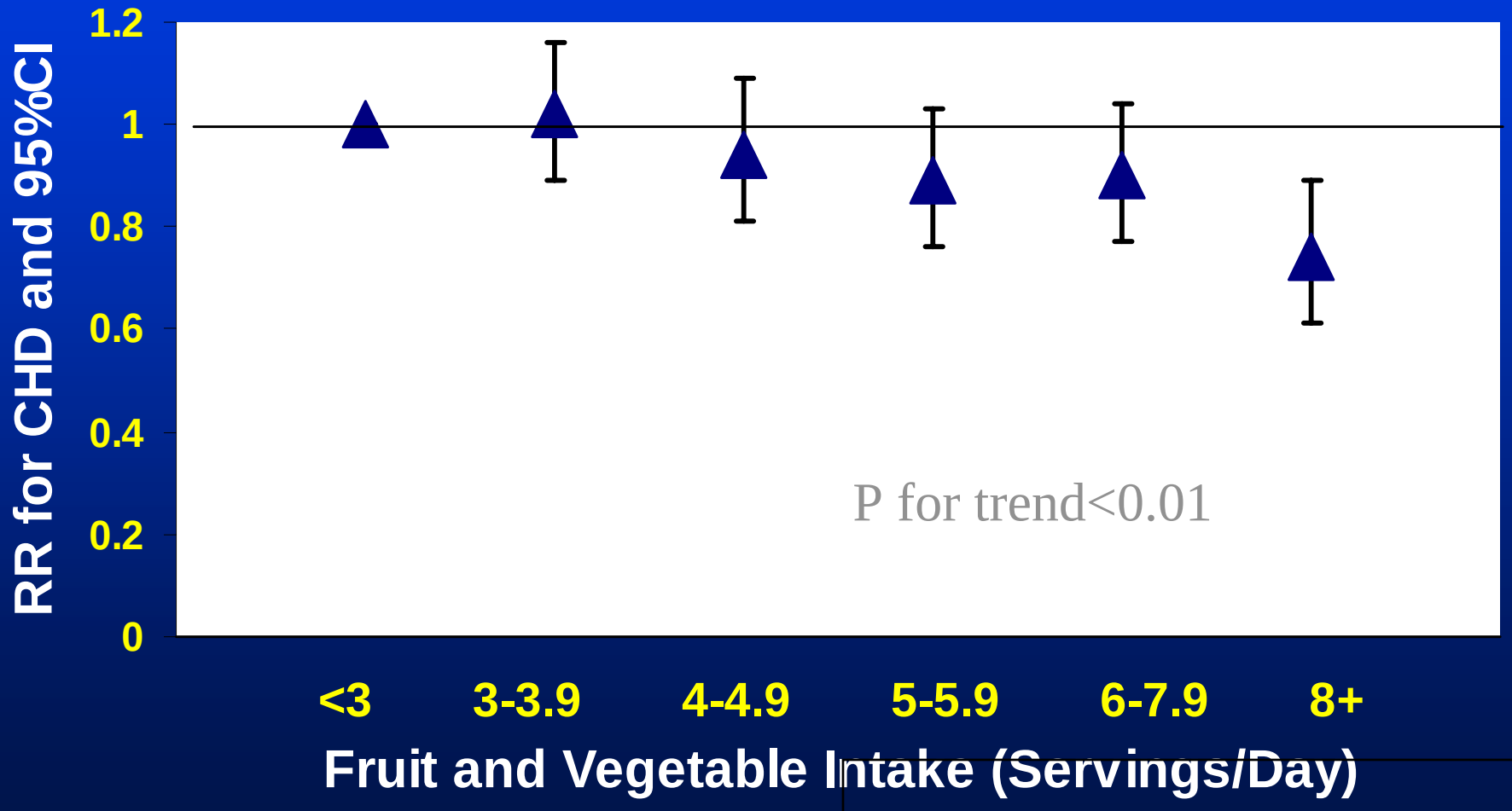
Percentage of Edentulous Subjects' Dietary Intake



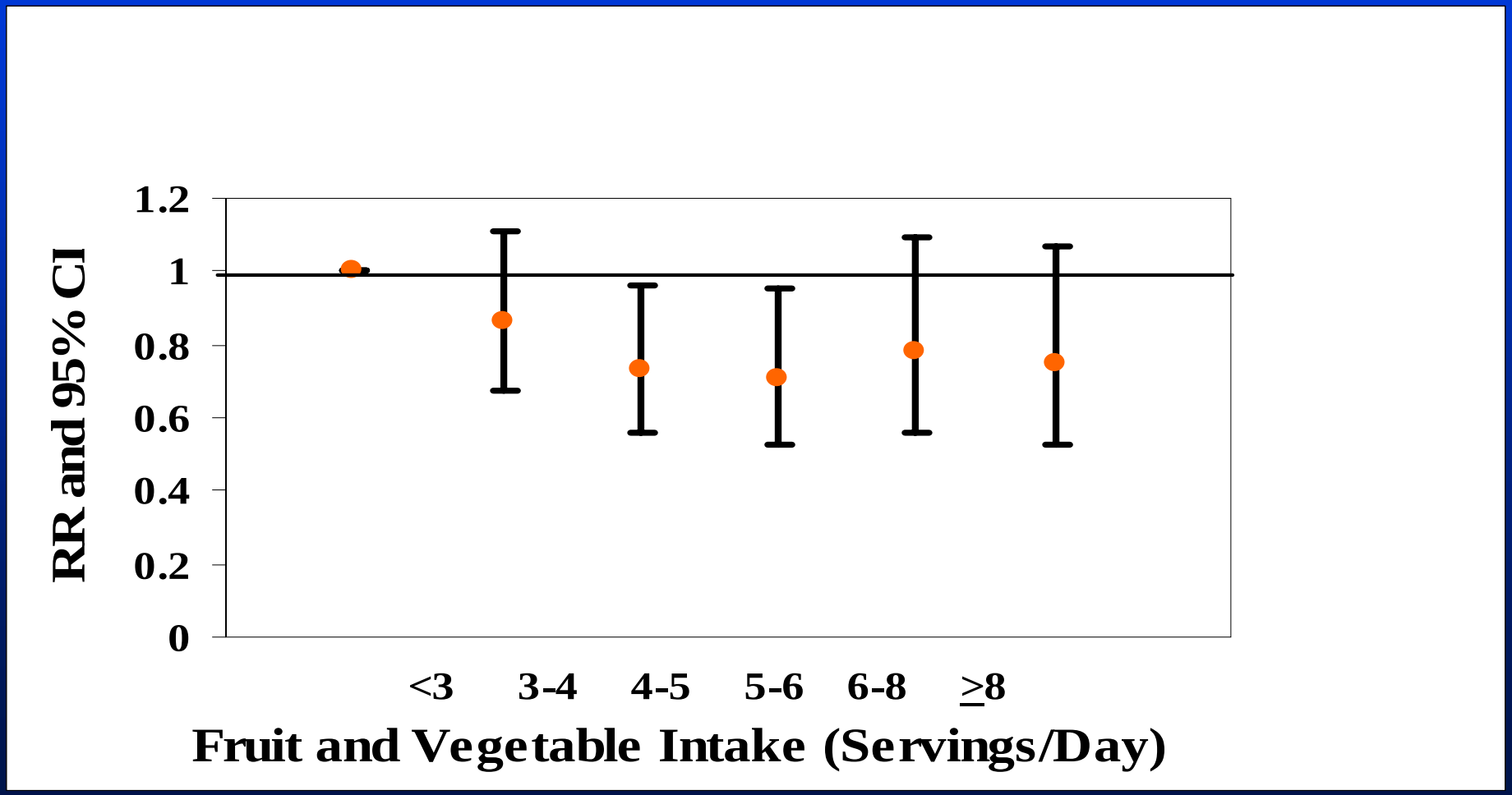
(After Adjusting Age, Smoking and Physical Activity)

Hung, CDOE In Press

Fruit and Vegetable Intake & Risk of CHD in HPFS and NHS *(Annals of Int. Med. 2001)*



Intake of total fruits and vegetables and risk of Ischemic stroke in the NHS and HPFS *(JAMA, 1999)*



Change in Diet after Tooth Loss among health professionals

	Tooth loss		
	0	1-4	≥5
% of subjects consuming banana in 1994 among those who consumed banana 1+ times/week in 1986.	87.0	86.6	85.1
% of subjects consuming cantaloupe in 1994	49.2	50.7	49.8
% of subjects consuming apple or pear in 1994*	79.3	77.9	69.9
% of subjects consuming cooked carrot in 1994	49.7	53.1	56.7
% of subjects consuming raw carrot in 1994*	70.2	62.5	55.5

Subjects with 10 or fewer teeth in 1986 excluded.

Hung, JADA, 2003

Prostheses Do Not Seem to Improve Diet

Dentures (partial / RPD)

- Improvement in perceived chewing ability
- No change in dietary intake

Jepson et al 1999 In Press
Moynihan et al 1999 In Press

Dentures (full)

- Perceived improvement in chewing efficiency
- No change in dietary pattern

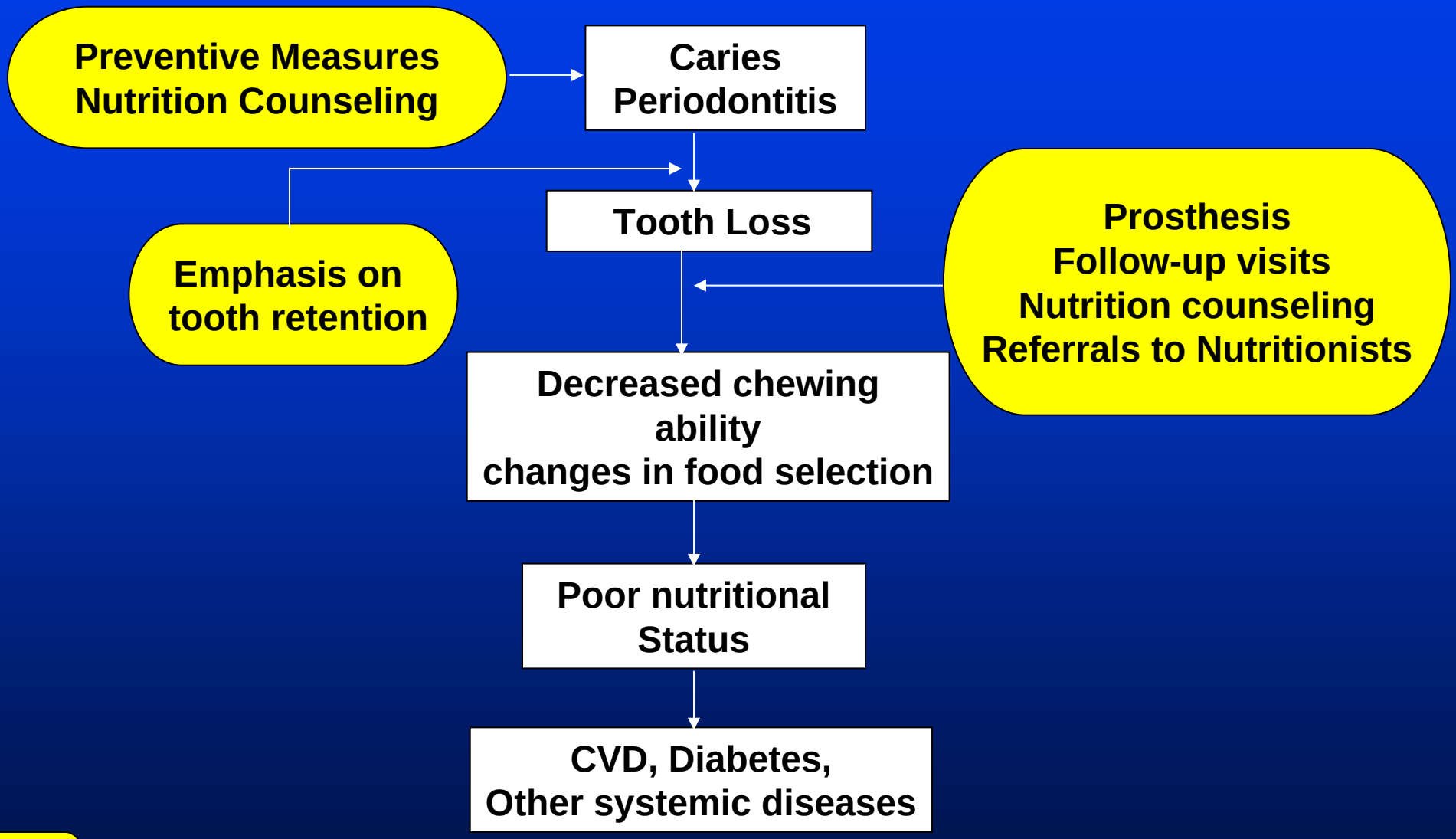
Ettinger R L. Special Care in Dentistry 1998
Gunne H-S J, Wall A.K. Acta Odont Scand 1985

Dentures (implant-retained)

- Perceived improvement in chewing efficiency
- No change in dietary pattern

Garrett N et al J Prosthet Dent 1997

Role of Clinicians in Improving Nutrition



Yellow boxes indicate when and where the clinician can play a role

JCDA, 2005