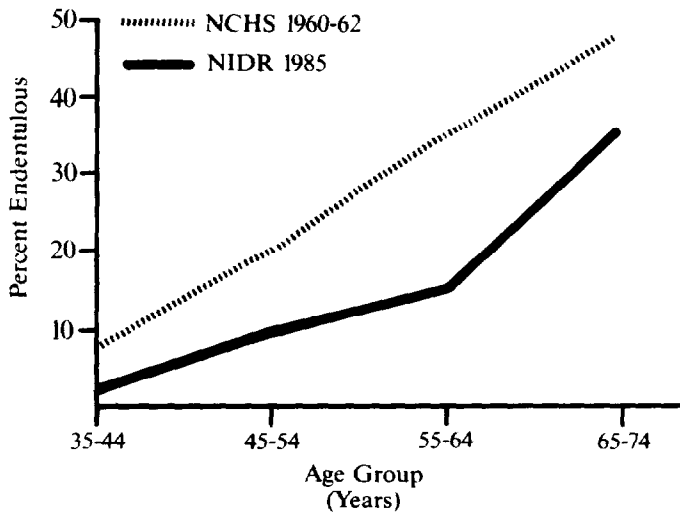Dental Diseases 🔲

Figure 8-3. Percent of persons by severe loss of periodontal attachment (pocket depths measuring 4 mm or more) and age groups as determined from the NIDR survey of employed adults and seniors.

Source: National Institute of Dental Research 1987.

Figure 8-4. Comparison of the percent of edentulous persons in the 1985-86 NIDR survey to that reported from the NCHS survey of 1960-62.

Source: National Institute of Dental Research 1987.

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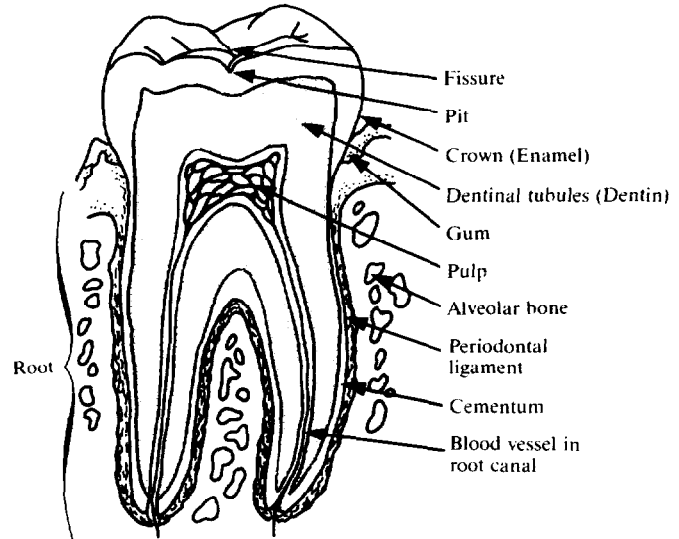
Figure 8-5. **Schematic cross-section of a typical mandibular tooth. The gums recede with age, exposing the cementum of the tooth root.**

dental organ that produces the tooth enamel and a dental papilla that produces the tooth pulp and the dentin. Cells from the dental follicle form the cementum and periodontal ligament after the tooth has formed.

The deciduous teeth begin forming at about 6 weeks *in utero*, when cells in the primitive oral cavity differentiate to form the dental lamina—the site of development of the tooth buds. The dental lamina is active from the second month of embryonic development, when the first buds for the deciduous teeth are formed, until about the age of 5 years, when the buds for the last permanent molars are initiated.

The formation of the tooth crown begins with the secretion of a dentin matrix containing collagen fibrils. Mineral ions then enter the matrix to form small crystals on or between the fibrils. Successive layers of dentin are formed. Enamel formation begins as soon as the first dentin layer has been laid down. Cells of the enamel organ secrete successive layers of a matrix that is chemically different from dentin matrix. Mineral crystals appear in each layer as it is secreted and grow larger as more mineral enters the tissue; matrix proteins and water are removed. This so-called maturation of the enamel continues after the full thickness of enamel matrix has

been laid down. These events occur at different times for the various deciduous and permanent teeth, beginning from a few months before birth until late adolescence.

As the tooth erupts into the mouth, it loses the layers of cells and blood vessels that covered the enamel. The enamel, however, continues to mature during the period immediately after tooth eruption and to incorporate minerals (including fluoride) into its structure from saliva, food, and drinking fluids. Otherwise, the enamel of fully erupted teeth is not known to have any metabolic dependence upon nutrients consumed in the diet. The inner surface of dentin adjacent to the pulp remains lined with the cells that formed it, and these cells continue to form secondary dentin at a very slow rate throughout the life of the tooth.

Thus, there are different periods in the developmental history of the tooth. During the preeruptive period when the crowns are forming in the jaws (which, for the wisdom teeth, continues into late adolescence), the developing enamel and dentin of the crowns are subject to nutritional deficiencies or imbalances in the same way as other tissues. In fact, in the enamel the preeruptive period can be divided into two phases, the secretory and the maturation stage. Nutritional deficiencies or excesses may affect either stage separately or both. Hypoplastic lesions in the enamel reflect disturbances affecting the secretory process. Hypomineralized defects, such as the white spot lesions of fluorosed enamel, give evidence of interference with the maturation process. After eruption, when the crown of the tooth has emerged from the jaw and the enamel is bathed instead by the saliva as well as exposed to micro-organisms and their byproducts, sloughed cells, and food debris, nutritional deficiencies or excesses and dietary habits may affect teeth in a totally different manner.

The development and maintenance of the soft tissues and bone that anchor and support the teeth (the periodontium) are also subject to nutritional deficiencies. The periodontium comprises the gingiva (the tissue that covers the alveolar bone process and surrounds the top of the tooth), the periodontal ligament (the soft connective tissue that surrounds the roots of the teeth and joins the root cementum and the alveolar bone), the root cementum (a specialized calcified tissue that covers the tooth root), and the alveolar bone (the bone that forms and supports the sockets of the teeth). The bony tissue of the alveolar process is dependent on the presence of teeth. The alveolar bone grows in response to dental eruption, is modified by dental changes, and resorbs when the teeth are lost as a result of

advanced dental caries and/or periodontal disease. The resorption of al-
veolar bone ridges (residual ridge resorption) has been described as an oral
disease entity (Atwood 1971).

Throughout life, the oral soft tissues undergo rapid rates of turnover and
repair, and the continued presence of optimum levels of nutrients is neces-
sary to maintain proper oral health and to resist disease.

Dental Caries

Once a tooth is fully erupted, it becomes subject to the influence of
chewing, acids in the mouth, and bacterial plaque. Dental caries begins
with dissolution of the mineral surface of the tooth by acid produced from
fermentable carbohydrate (e.g., sugar) by dental plaque bacteria. Next, the
dissolution advances further into the enamel, first appearing as a white
spot, and subsequently into the dentin and eventually into the pulp. If left
untreated, the result is destruction of the crown and root system, tooth
loss, and resorption of the surrounding bone (Newbrun 1978). The decay
that begins in pits and fissures, on the smooth surfaces, or on exposed roots
of the teeth is invariably due to the combined effects of bacterial infection
and host and dietary factors over time (Wei, Fomon, and Anderson 1977;
Shaw 1978, 1987). Patients with reduced salivary flow are also at increased
risk (Mandel 1983).

Of the many bacteria found in tooth plaque, *Streptococcus mutans* is
considered the primary etiologic agent in coronal caries (Loesche 1986;
Shaw 1987). This bacterium is unique in that it produces enzymes that
convert table sugar (sucrose) into long sticky polysaccharides. The poly-
saccharides promote firm attachment and accumulation of the bacteria in
dental plaque (Gibbons and van Houte 1978). If not removed, the bacteria
metabolize sugars (including sucrose) and produce acids that dissolve
minerals from the teeth and begin the decay process. Root caries, decay of
tooth root cementum following recession of gum tissues, has been regarded
as an independent type of caries (Nikiforuk 1985). However, a relationship
between root caries and coronal caries has been reported recently (Vehk-
alahti 1987). Decay also occurs around restorations where plaque is diffi-
cult to remove.

Whereas sugars facilitate the action of cariogenic bacteria, saliva is a major
impediment to the pathogenesis of these bacteria (Vogel 1985; Mandel
1986). This fluid, secreted by the major and minor salivary glands, coats the
tissues of the mouth and provides protection against bacteria that cause
disease. The saliva contains molecules that when adsorbed to the tooth can
influence attachment of bacteria to this surface and, when present in the

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fluid, can clump bacteria together such that they cannot attach to the teeth but rather are cleared from the mouth by swallowing. Saliva also contains enzymes and proteins with antibacterial activity that inhibit bacterial growth or kill bacteria. In addition to these defense mechanisms, saliva can contain antibodies that attack specific kinds of bacteria. Another important function of saliva is that it supplies the calcium and phosphorus, minerals important in the remineralization of tooth surfaces that have lost mineral due to acid attack.

Several types of interventions might prevent tooth decay. The most important diet-related interventions are fluoridation of drinking water and control of sugar intake, but other approaches such as oral hygiene, application of plastic sealants, use of topical fluorides, and various chemotherapeutic agents have also been shown to be effective (Navia 1985). The efficacy of a caries vaccine that would prevent infection by cariogenic bacteria is under investigation (Navia 1985; Krasse, Elmilson, and Gahnberg 1987).

Periodontal Disease

Periodontal disease encompasses pathologic changes of the gingiva, periodontal ligament, cementum, and alveolar bone that make up the attachment and supporting tissues of the teeth (Lindhe 1983). The destruction of these tissues (the periodontium) results in the loosening and loss of the teeth. The etiology of this disease, like that of dental caries, is multifactorial, involving bacterial infection, host factors, diet, and duration of infection. The accumulation of bacterial plaque near the gum line can cause a nondestructive inflammation of the gingival tissue (gingivitis). Early studies (Löe, Theilade, and Jensen 1965; Theilade et al. 1966) showed that dental plaque caused clinical gingivitis and that when the plaque was removed the disease was reversed or prevented. If the inflammation persists, the disease can progress and destroy the gingival connective tissue and underlying tissues to form a subgingival or periodontal pocket. As the pocket depth increases, the alveolar bone (bone supporting the tooth) is resorbed and the tooth is lost. The primary etiologic agents for periodontal disease are subgingival bacteria (Socransky 1977).

The bacteria make and release toxic substances, destructive enzymes, and antigenic molecules that cooperate in the destruction of periodontal tissues. Large amounts of bacterial products can directly damage tissues or can cause overstimulation of the immune system with resulting tissue damage; under normal oral conditions, the immune system is protective against bacterial challenge (Nisengard 1977; Seymour 1987). Other factors that can decrease host defense mechanisms and, therefore, promote periodontal disease are immunologic defects, metabolic problems, endocrine

dysfunctions, and nutritional deficiencies. Reduced salivary flow is also a risk factor (Mandel 1983).

Key Scientific Issues

- Role of Diet in Tooth Decay
- Role of Diet in Periodontal Disease
- Role of Diet in Other Dental Conditions
- Effect of Tooth Loss on Nutritional Status

Role of Diet in Tooth Decay

Although diet is indicated as one of the main direct determinants of dental caries, it is also linked to each of the other determinants of causation, prevention, and treatment. Dietary factors are important for the host's normal tooth development, host resistance, and salivary composition; for selection and maintenance of the microbial population; and for the amount of time teeth are in contact with sugars and their acid byproducts (Alfano 1980). Sugars are the most important dietary factors in the causation of dental caries; fluoride is most important in its prevention. The role of other dietary components such as fats, protein, and minerals in modulating these effects is also of much current interest.

Role of Sugars

Despite the complex etiology of dental caries, the causative role of dietary sugar—especially sucrose—is well established (Keyes and Jordan 1963). Caries-producing bacteria have a rather high need for a range of simple sugars (glucose, fructose, lactose, maltose, and sucrose) that they readily metabolize into acids that demineralize teeth. The unique role of sucrose, however, depends on its ability to be converted by mouth bacteria into extracellular polymers of glucose or fructose that adhere firmly to the tooth surfaces (Gibbons and van Houte 1978; Doyle and Ciardi 1983). Together with the micro-organisms that secrete them, these extracellular products facilitate the further attachment to teeth and proliferation of bacteria.

Food sugars, such as glucose and fructose that diffuse into this plaque, nourish the micro-organisms. Food starches are converted to a limited extent by salivary enzymes to soluble glucose that can be used by plaque bacteria. Although saliva readily neutralizes organic acids, it has limited access to the acids generated at the tooth surface beneath the plaque.

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Recent reviews of extensive animal studies and human epidemiologic and clinical observations during the past 30 years have thoroughly documented the role of sucrose and other sugars in tooth decay (Newbrun 1982; Glinsmann, Irausquin, and Park 1986). Studies in rats, for example, have demonstrated a direct relationship between the amount of sucrose in the diet and caries incidence that reaches a plateau when the sucrose concentration is 8 to 40 percent by weight. Such studies have also demonstrated the importance of the consistency and composition of the diet. Sucrose has been shown to have a greater cariogenic effect than glucose, fructose, or other simple sugars. Caries incidence increases directly with the frequency of sugar consumption (Bowen et al. 1983).

Despite methodological difficulties in assessing population-wide sugar intake, human epidemiologic studies have generally indicated an association between sugar intake and tooth decay (Sreebny 1982) as well as increasing rates of tooth decay among populations whose sugar intake is increasing (Burt and Ismail 1986). Human clinical studies have demonstrated a virtual absence of tooth decay and much lower concentrations of cariogenic bacteria in the mouths of children with inherited metabolic disorders (e.g., fructose intolerance) that prevent sucrose consumption (Newbrun 1982).

Other clinical studies show the importance of frequency of sugar consumption and the form in which it is consumed. Sugars in solution are more readily cleared from the mouth than sugars in solid foods and, therefore, are more likely to have only a transient influence on plaque acidity. Foods that adhere to the teeth are more cariogenic than those that wash off quickly (Gustafsson et al. 1954). In addition, people eat foods in different ways. It appears that any increase in the length of time food remains in the mouth is likely to increase the initiation and progression of caries (Newbrun 1978).

Metabolism of sugars by mouth bacteria results in acid production concomitant with a pH drop in dental plaque from a level near neutral to about 5. This effect lasts 20 to 30 minutes until buffering agents in saliva restore the normal pH. Recurrent exposures to such episodes for extended time periods lead to dissolution of the enamel. The presence of calcium, phosphate, fluoride, and other minerals in the saliva permits remineralization when the pH is neutral. Thus, the tooth is exposed to alternating cycles of dissolution and remineralization. If the balance favors dissolution, clinical carious lesions occur, and if it favors remineralization, early lesions are repaired and advanced caries does not develop.

Research into the cause of caries has not entirely overcome problems in measurement of sugar intake, the confounding effects of other dietary components, and secular trends in dental hygiene and fluoride consumption. Evidence exists that sugars as they are consumed in the average American diet contribute to the development of dental caries. Current average daily intake levels in the United States are 62 to 143 g, or 18 to 32 percent of total caloric intake (Glinsmann, Irausquin, and Park 1986). Reduction of dietary sugar would be expected to reduce levels of tooth decay in the population, in particular at the time of tooth eruption (Loesche 1985).

Nursing Bottle Caries

A special example of the importance of the duration of contact between fermentable sugars and teeth is nursing bottle caries (Gardner, Norwood, and Eisenson 1977; Ripa 1978; Dilley, Dilley, and Machen 1980; Kelly and Bruerd 1987). This unique pattern of dental caries, where the upper incisors become badly decayed, has been described in young children. In severe cases, the chewing surfaces of the molars become affected, but the lower incisors rarely develop lesions. The lesions on the upper incisors begin as a generalized demineralization on the third of the crown next to the gums and progress so that the tooth is encircled by a ring of decay that may become so deep that the crown breaks off just above the gum.

This condition occurs when children are put to bed with a bottle of milk, formula, juice, or sweetened beverages. These practices allow sugars in milk or other fluids to remain in contact with the tooth surfaces for extended periods. The position of the upper incisors makes them especially vulnerable, while the lower incisors are usually protected by the tongue. These carbohydrates are metabolized to acids by the oral micro-organisms that are always present on tooth surfaces, and this action is compounded by the infrequent swallowing and reduced salivary flow that occur during sleep.

Role of Fluoride

The efficacy of fluoride in the prevention of tooth decay is well established. It is generally assumed to prevent caries by decreasing the solubility of tooth enamel, promoting remineralization of the enamel surface, modifying the chemical interactions in plaque, or inhibiting lactic acid production by cariogenic bacteria (Navia 1985).

Since 1945, when fluoride was first added to community water supplies, more than 35,000 published scientific papers have attested to the efficacy,

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safety, and cost-effectiveness of fluorides in prevention of tooth decay (Richmond 1985). Many of these studies demonstrate reductions in dental caries of 50 percent or more when fluoride is added to a community water supply.

One focus of research in the 1930's and 1940's was to determine the optimal level of fluoride in drinking water. This concentration is about 1 ppm for temperate regions. Current recommendations for optimum fluoride concentrations vary from 0.7 to 1.2 ppm, depending on regional variation in prevailing air temperature because the amount of water consumed tends to be less in colder climates. Caries incidence increases when drinking water contains less than the optimum level of fluoride, but mild forms of fluorosis (the mottling of tooth surface) can begin to occur at about twice the optimum level. Fluorosis occurs only when developing teeth are exposed to excess fluoride, generally over a period of months; once a tooth is fully formed, continuing fluoride exposure may promote the remineralization process.

For children living in areas with inadequate concentrations of fluoride in the water, dental authorities recommend fluoride supplements at dosages that depend on the natural fluoride content of the local water supply and the age of the child. The dosages currently recommended by the Council on Dental Therapeutics of the American Dental Association and the American Academy of Pediatrics are provided in Table 8-1. The effectiveness of prenatal fluoride supplementation, however, is uncertain because clinical studies of its effects on subsequent caries incidence have been equivocal (Driscoll 1981).

Table 8-1
Supplemental Fluoride Dosage Scheduled
(in mg F/day[a]) According to
Fluoride Concentrations of Drinking Water

	Concentration of Fluoride in Water (ppm)		
Age (years)	Less than 0.3	0.3 to 0.7	Greater than 0.7
Birth to 2	0.25	0	0
2 to 3	0.50	0.25	0
3 to 13	1.00	0.50	0

[a]2.2 mg of sodium fluoride contain 1 mg of fluoride.

Source: Council on Dental Therapeutics 1984.

Another area of uncertainty concerns current levels of fluoride intake. When the optimal level of about 1 ppm was originally established, the primary source of fluoride for human consumption was the water supply. Today, fluoride is also available from toothpastes, mouth rinses, and soft drinks and foods prepared with fluoridated water. These sources have increased the availability of fluoride beyond that originally envisioned, perhaps to levels that might induce fluorosis (Horowitz et al. 1984). Several recent studies have observed an increased incidence of mild (barely noticeable) tooth mottling among children taking fluoride supplements or living in communities where water is fluoridated. Although there is no evidence that this condition is harmful, these observations have led some authorities to suggest reevaluation of previously accepted standards for optimal fluoride use (Navia 1985; Leverett 1982).

Controversy about water fluoridation is not new. Despite the preponderance of community studies conclusively demonstrating the effectiveness of both naturally occurring and purposefully supplemented fluoride in drinking water, opposition has been raised on grounds of effectiveness, safety, and individual rights (Taves 1979; Watson and Schrotenboer 1983). However, studies have failed to show any relationship between fluoride and increased risk for heart disease, brain lesions, liver disease, allergies, kidney disease, mental retardation, cancer, or mutagenicity (Richmond 1985). Indeed, studies of one chronic disease, osteoporosis, suggest a protective effect for fluoride (Simonen and Laitinen 1985; see chapter on skeletal diseases). Based on the scientific evidence, the courts have consistently ruled that States and municipalities have the right to implement fluoridation as a public health procedure (Block 1986; Löe 1986).

Role of Other Dietary Factors

Protein. Experimental data show certain critical periods in the development of the teeth of young rats, during which a marginal protein deficiency exerts striking effects (Nakamoto, Mallek, and Miller 1979a, 1979b). When low-protein diets were fed to rats at any time throughout pregnancy and lactation, the molars of their offspring were altered in shape and were significantly smaller than the molars of offspring from control rats (Shaw and Griffiths 1963; Holloway, Shaw, and Sweeney 1961). Although the change in tooth size occurred without a reduction in enamel thickness, minor cusps of the third molars of the offspring from these protein-deficient females frequently did not develop, suggesting that nutritional deficiencies can prevent normal tooth development. Caries incidence was higher in the offspring of protein-deficient females than in control offspring when both were fed the same caries-producing diet after weaning. The offspring from

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protein-depleted females had abnormalities of the salivary glands (decreased DNA, RNA, and protein concentrations, and decreased net weight) and the saliva (greatly reduced salivary flow and small increase in salivary protein concentration), which may partially explain their increased caries incidence (Menaker and Navia 1973a, 1973b, 1974).

Supplementing the diet of pregnant and lactating rats with 1 percent DL-methionine (the limiting amino acid of casein) prevented almost all of the abnormalities, but supplementation begun at weaning was ineffective (Shaw and Griffiths 1963). That the deficient nutrient was protein, but not calories or other nutrients, was demonstrated when protein supplements alone prevented these abnormalities (Menaker and Navia 1973a, 1973b, 1974).

In humans, protein-energy malnutrition is often associated with poor dental health. Delayed tooth eruption and impaired tooth development are common findings among children in developing countries where protein, calories, and other nutrients are inadequate (Steggerda and Hill 1942; Sweeney 1966). Abnormal lesions on the teeth of younger children in less developed nations have been reported to be highly susceptible to tooth decay even when children consume lesser amounts of sugar-containing foods and consume foods less frequently than children in more highly developed nations (Sweeney et al. 1969; Sweeney, Saffir, and de Leon 1971). Nevertheless, the permanent teeth in older children and adults in developing countries are not more susceptible to caries than those in older children and adults in industrialized countries (Russell 1966). At present, there is no evidence to indicate that an increase in caries incidence accompanies delayed tooth eruption. Little information on the influence of protein deficiency on the rate of salivary flow or on saliva composition in humans is available (Vogel 1985).

Fat. In animal studies, increasing the concentration of fat in a diet with the carbohydrate concentration held constant reduces the caries-producing potential of the diet (Shaw 1950). This may have to do with the sequence in which foods are eaten. When dietary sugar is followed by a food such as cheese (which is high in fat), the usual acid-forming response is blunted and less acid is present in plaque (Edgar 1981; Edgar et al. 1982; Schachtele and Jensen 1983). The significance of these observations for human tooth decay, however, has not yet been established.

Vitamin A. Studies of the continuously erupting incisor of the rat showed that a severe deficiency of vitamin A impairs tooth formation (Wolbach and

Howe 1925, 1933; Boyle and Bessey 1941; Schour, Hoffman, and Smith 1941). The observed changes resembled the effects of vitamin A deficiency on tissues of ectodermal origin elsewhere in the body. Bones formed entirely during a period of vitamin A deficiency contain reduced amounts of calcium, suggesting an effect on bone mineralization and the resulting bone structure. A deficiency of vitamin A for a limited time before tooth eruption has been shown to increase the susceptibility of rat molars to caries (Harris and Navia 1980; Navia and Harris 1980). Other studies, in which the third molars of rats were cultured, showed that vitamin A deficiency delays tooth formation and interferes with mineralization of both enamel and dentin (Navia et al. 1984). Vitamin A deficiency has also been observed to disrupt normal differentiation of rat tooth-forming cells and to alter the distribution of certain molecular components of dentin and pulp (Punyasingh et al. 1984).

Vitamin D and Calcium. In early studies of rickets, deficiencies of a fat-soluble vitamin, later identified as vitamin D, resulted in the softening and discoloration of the enamel of developing teeth, delayed tooth eruption, and an irregular tooth arrangement in the jaw (Mellanby 1918; Mellanby 1919). These defects were also seen in the offspring of pregnant animals given a vitamin D-deficient (rickets-producing) diet but not in animals that received daily supplements of cod liver oil (Mellanby 1928). Calcium deficiency resulted in less severe defects in the enamel and dentin, but deficiencies of both calcium and vitamin D resulted in very poorly formed enamel and dentin (Mellanby 1923). These early studies indicated the need during tooth development for adequate dietary intake of calcium and vitamin D.

In a series of clinical trials in England in which children received vitamin D supplements to their regular diet, modest reductions in dental caries were observed both for secondary teeth that had erupted before the trials began and for teeth that erupted during the trial (Committee for the Investigation of Dental Disease 1936). Despite evidence for a beneficial role of vitamin D in tooth development, clinical trials to determine whether vitamin D supplements reduce dental caries have yielded varied results (Shaw 1952), and this area remains uncertain.

A long-term study of dental abnormalities in children showed that the inherited conditions of vitamin D-dependent rickets or hypoparathyroid syndrome, both of which reduce levels of plasma calcium, are associated with abnormal lesions in tooth enamel but normal dentin. Children with low plasma levels of phosphorus but normal levels of plasma calcium developed normal enamel but abnormal dentin. Those with low plasma levels of both

calcium and phosphorus developed abnormalities in both enamel and dentin (Nikiforuk and Fraser 1979). The implications of these observations for dietary intake in normal children are unknown.

Phosphate. An increasing amount of phosphate in the diet reduces experimental caries incidence in rats (Nizel and Harris 1964), but supplementation of the diet of children with phosphate has not been shown to produce benefits (Stralfors 1964; Ship and Mickelsen 1964; Averill and Bibby 1964). Phosphate supplementation of chewing gum is reported to produce a reduction in caries incidence (Baron and Bilotti 1979; Finn et al. 1978; Jamison 1979; Richardson 1979).

Trace Elements. Certain epidemiologic surveys and experimental animal studies have suggested that some trace elements such as molybdenum, vanadium, strontium, and iron can inhibit caries development and that other elements such as selenium, magnesium, and cadmium can accelerate caries development. None of these studies has demonstrated either an independent action of these elements or an action that either augments or decreases the influence of fluoride (Curzon 1983; Navia 1970).

Other Food Components. Foods containing salts or acids can stimulate salivary flow, alter its buffering capacity, or decrease microbial acid production. Food components such as lectins (Gibbons and Dankers 1981, 1982), tannins (Kashket, Paolino, and van Houte 1985), and other less well-characterized substances (Madsen 1970) can inactivate bacteria or influence their metabolism and reproductive ability and, in these ways, may affect caries incidence.

Alternative Sweeteners. Sucrose and other simple carbohydrates can be replaced by polyols or non-nutritive sweeteners in many kinds of foods. The benefits of such products must take into account the quantities of cariogenic sweeteners that must be replaced to achieve significant caries reduction, the relationship of a food's overall chemical composition and physical properties to its cariogenicity, and the possibility of other nutritional or health effects (Hoskins 1978).

Role of Diet in Periodontal Disease

Periodontal disease is an infection of the gums and supporting structures caused mainly by the microbial flora that adhere to dental plaque. Like dental caries, the etiology of this disease includes specific microbiologic, environmental, and systemic components. The gingival sulcus (the space

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between the gums and the teeth) harbors bacteria that directly and indirectly affect the formation of periodontal lesions and the later destruction of periodontal tissues and their cellular components. Environmental influences on the gums include dietary sugars and other factors that promote the formation of plaque on tooth surfaces, entrapment of food in periodontal lesions, and poor dental hygiene. Systemic influences on the initiation and progression of periodontal lesions are less well defined and more difficult to investigate, although there is considerable evidence that persons who are diabetic, immunosuppressed, or genetically susceptible are especially prone to periodontal disease. In general, only severe nutritional deficiencies or extreme endocrinologic imbalances appear to adversely affect the resistance of the gums. Diet may influence periodontal disease not only through its effects on plaque formation, but also through stimulation of salivary flow, antigenic stimulation of immune mechanisms, and enhancement of host repair and defense mechanisms (Alfano 1976; Navia 1985); however, its precise role is as yet undefined.

Role of Calcium

Calcium is a necessary mineral in the formation of all calcified tissues, including those of the periodontium. In adult dogs, a low-calcium diet produces radiologic evidence of loss of alveolar bone within 2 months and a periodontal syndrome within 1 year (Henrikson 1968). When the diet was supplemented with calcium, the periodontal disease reversed within 6 months.

It has been proposed that periodontal disease is caused by internal resorption of bone due to an imbalance of dietary calcium-to-phosphorus ratio (Krook et al. 1972). A more recent study (Svanberg et al. 1973) using similar conditions failed to confirm those results. Most scientists believe that periodontal disease is initiated by subgingival bacterial infection. Additional studies are necessary before the role of calcium in the etiology of periodontal disease can be ascertained.

Role of Vitamin C

A diet deficient in vitamin C (ascorbic acid) causes scurvy in humans. Advanced disease produces gingival changes and loosening of the teeth. Scurvy and some types of periodontal disease in humans are similar to experimental scurvy in the guinea pig and in both Old and New World monkeys, with bleeding of the gingiva, destruction of the periodontal membranes, and loosening and ultimate loss of teeth (Alvares et al. 1981; Boyle and Bessey 1941; Glickman 1948). There is ample evidence that ascorbic acid, which is required for the formation of collagen in connective

tissues, is necessary for the repair and maintenance of periodontal tissues. Only severe deficiencies result in aggravating periodontal disease.

Role of Folate

Folate is a member of the vitamin-B complex necessary for the normal production of red blood cells. Folate deficiency is a common type of hypovitaminosis in humans. In experimental gingivitis studies in humans, folate supplementation resulted in significantly less inflammation of gum tissues (Vogel and Alvares 1985). Its role in gingivitis and periodontal disease is under investigation.

Role of Other Nutrients

Various periodontal abnormalities have been produced in experimental animals as a result of nutritional deficiencies of protein (Chawla and Glickman 1951), tryptophan (Bavetta and Bernick 1956), calcium (Becks and Weber 1931), magnesium (Klein, Orent, and McCollum 1935), vitamin A (King 1940), vitamin D (Becks and Weber 1931), and B-complex vitamins (Becks, Wainwright, and Morgan 1943; Tomlinson 1939). However, in these studies, only severe deficiencies resulted in periodontal destruction, and most of these experiments were carried out in grown animals.

On the basis of current knowledge, nutritional deficiencies or imbalances do not seem likely to exert major influences on the prevalence or severity of periodontal disease in the general population. However, the ability of individuals with nutritional abnormalities to resist the progression of periodontal lesions initiated by microbiologic and environmental influences may be reduced. Severe protein malnutrition might alter the immunologic system in such a way as to render the host unusually susceptible to infection.

Role of Diet in Other Dental Conditions

Residual Ridge Resorption

The alveolar ridge (the bony ridge in which teeth are positioned) is vulnerable to the resorption or loss of mineral subsequent to tooth loss. This process results in a progressive change in the shape and a reduction in the size of the ridge that greatly affects the stability and retention of dentures (Atwood 1971, 1979; Tallgren 1972). While some patients adapt to ill-fitting dentures and contend that they can eat anything, many patients adapt by changing their diet (Yurkstas and Emerson 1964).

Resorption of the residual ridge results from an imbalance of bone resorption and bone formation, as does osteoporosis, leading to speculation about a possible relationship between these conditions. The role of calcium in the prevention and treatment of osteoporosis, either alone or in combination with vitamin D, estrogen, or fluoride, has received much attention, and calcium deficiency has been reported to be a factor in both osteoporosis and residual ridge resorption (Barzel 1979; Menczel et al. 1982). A study of 44 toothless patients found a relationship between a history of reduced dietary calcium intake and severe accumulated ridge reduction (Wical and Swoope 1974). Denture patients who were given daily supplements of calcium and vitamin D in a 1-year double-blind controlled study had 36 percent less bone loss than the group that received a placebo (Wical and Brussee 1979). However, the wide overlap of bone loss in the treatment and control groups suggested considerable individual variation and the need for further research in this area. Approaches available to minimize residual ridge resorption or restore lost bone are the following: use of implants, bone rebuilding with calcium phosphate preparations, and prevention of tooth loss.

Oral Cancer

In its earliest stages, oral cancer is painless and may present no overt symptoms. Because dentists see many people regularly, and because dentists have access to the oral cavity, they are in a unique position to provide early identification of suspicious lesions and signs of tobacco habits that put people at risk for oral and other diseases.

Although alcohol and chewing tobacco are known to increase risk, the role of nutrition in oral cancer in humans is less well established (see cancer chapter). In part, this is because epidemiologic studies on the relationship of nutrients to cancers elsewhere in the body have not been extended to include oral lesions. However, animal models have been developed for the experimental production of oral cancer that take advantage of the easy accessibility of the tongue and the mucosa of the oral pouch of the hamster for the application of suspected carcinogens and for later observation of their effects (Marefat and Shklar 1977; Shklar 1972).

These models permit the evaluation of nutritional influences in cancer formation. In several studies, animals whose diets were supplemented with derivatives of vitamin A developed oral tumors that were fewer, smaller, and later in appearance on exposure to the carcinogen 9,10 dimethyl-1,2 benzanthracene (DMBA) than those in animals that had not received the supplement (Shklar, Marefat, et al. 1980; Shklar, Schwartz, et al. 1980;

Burge-Bottenbley and Shklar 1983). A similar test of vitamin E (DL-L-tocopherol) produced fewer and smaller tumors in the animals that received it (Shklar 1982; Schwartz et al. 1985; Odukoya, Hawach, and Shklar 1984). More study of these issues is needed. Similarly, results from small studies on the influence of nutrients on the course of head and neck cancer suggest the need for further investigation (Dwyer et al. 1986).

Effects of Tooth Loss on Nutritional Status

The loss of teeth in adulthood should not be considered a normal or an inevitable event, but a pathologic result of disease or trauma that affects the normal physiologic process of the jaws, teeth, muscles, and nerves of the mouth.

Chewing Ability

The loss of natural teeth as a result of dental caries, periodontal disease, or trauma significantly reduces the ability to chew foods, as demonstrated by a variety of chewing-efficiency tests (Manly and Vinton 1951; Yurkstas, Fridley, and Manly 1951; Kapur, Soman, and Yurkstas 1974; Rissin et al. 1978; Kapur and Garrett 1984; Perez, Kapur, and Garrett 1985). Other studies suggest that tooth loss leads to reduced force in chewing such foods as carrots, meats, raw vegetables, and peanuts; markedly lower chewing performance (Manly and Vinton 1951; Kapur and Garrett 1984); and avoidance of certain hard-to-chew foods (Yurkstas and Emerson 1964). Patients with reduced salivary flow may be unable to wear removable dental prostheses due to lack of lubrication (VA 1986).

Swallowing Ability

Dentures can cause a loss of sensation in the mouth that can lead to problems in swallowing. In a study of 584 individuals who had to have a foreign object removed from the esophagus, the absence of natural teeth was considered a major factor (Ray and Vinson 1958). Dentists are urged to warn all patients provided with partial or complete dentures about this hazard and about the need for care in the preparation and chewing of food. Of 16 patients seen during 1 year because of impaction of a foreign body in the throat, only 3 had normal teeth (Wengraf 1969).

Choking on food was reported to be the sixth most common cause of accidental death in the United States in 1979, with 700 of 2,900 fatalities occurring among people 75 years or older (NSC 1979). Studies of choking victims have found that many have poor dental status (Haugen 1963;

Anderson 1977), and poorly fitting dentures are frequently suspected to be a factor in the blockage of airways with inadequately chewed and swallowed food.

Effect on Food Selection

In addition to reduced chewing efficiency, various studies have demonstrated that loss of natural teeth, even when replaced with dentures, can cause the following impairments: diminished ability to discriminate food particle size and texture, such as ability to differentiate between soggy and crisp wafers (Kapur and Collister 1970); increased flow of stimulated salivary gland secretion (Kapur, Collister, and Fischer 1967; Kapur and Garrett 1984); and diminished ability to perceive subtle differences in the sweet taste of certain solid foods (Giddon et al. 1964).

Effect on Nutritional Status

Some early studies reported that inadequate dental function as a result of toothlessness or inadequate prosthetic devices contributed to the establishment and maintenance of nutritional deficiencies (Mann, Mann, and Spies 1945; Greene, Dreizen, and Spies 1947; Ruikka, Sourander, and Kasanen 1967; Makila 1968, 1969a, 1969b). However, dietary intake can be adequate in an individual with reduced chewing provided that food is selected carefully and prepared appropriately (Geissler and Bates 1984).

Implications for Public Health Policy

Dietary Guidance

General Public

Dietary factors of principal interest in dental diseases are sugars and fluoride. Frequent consumption of sugars, especially sucrose, promotes formation of dental plaque, the key predisposing cause of both caries and periodontal disease. In the United States, the daily intake of sugars ranges on average from 62 to 143 g, or 18 to 32 percent of total caloric intake. Evidence exists that sugars as they are consumed in the average American diet contribute to the development of de ital caries, suggesting that the general public should reduce its sugar consumption.

The role of fluoride in prevention of tooth decay is also well established from animal studies and from human epidemiology and clinical trials. Although fluoride is present in foods, the most efficient source of this nutrient for the general public is community drinking water that naturally

contains fluoride at an optimal level or to which fluoride is added to achieve the optimal level. Most, but not all, water supplies can be fluoridated, and current recommendations for optimum fluoride concentrations vary from 0.7 to 1.2 ppm depending on regional variation according to prevailing air temperature. Conclusive evidence shows that such levels of fluoride are safe.

Although other nutrients such as vitamin A, vitamin C, calcium, and phosphate may also be associated with prevention of dental diseases, evidence is insufficient at this time to recommend changes in dietary patterns on the basis of their relationship to these conditions for the general public.

Special Populations

Persons with diminished salivary flow are at special risk for caries and periodontal disease. They also may be unable to wear removable dental prostheses due to the lack of lubrication by saliva. Artificial saliva preparations containing fluoride and topical fluoride gels help to prevent tooth decay in such persons and can be recommended as an adjunct to sugar-restricted diets and appropriate dietary counseling. Children over the age of 6 months are at risk for nursing bottle caries, and their parents and caregivers should receive guidance in dietary and behavioral approaches to prevent this condition. Evidence related to the benefits of fluoride consumption by pregnant women on subsequent tooth development of the fetus and caries in the offspring is insufficient to recommend fluoride supplementation during pregnancy. Individuals with diabetes are especially prone to periodontal infections and should take special care to use available dietary and therapeutic means to control disease.

Nutrition Programs and Services

Food Labels

The presence and relative amount of added sugars, especially sucrose, contained in processed foods, as indicated by ingredient lists on food labels, should continue to play an important role in identifying dietary factors associated with dental disease.

Food Services

Evidence related to the role of dietary factors in dental disease suggests that food service programs should provide optimally fluoridated drinking water and promote noncariogenic foods, especially in programs for populations at high risk for dental diseases.

Special Populations

Persons with an active history of dental caries or with reduced salivary flow and parents of young children should be provided with counseling and assistance in developing diets low in cariogenic foods and in accessing appropriate sources of fluoride. Persons with diabetes are especially prone to periodontal infections and should take special care to use available dietary and therapeutic means to control their disease (see chapter on diabetes).

Research and Surveillance

Research and surveillance issues of priority related to the role of diet in dental diseases should include investigations into:

- The definition of critical periods of development of dental tissues that may be sensitive to nutrient intake.
- The role of nutritional factors in the maintenance and repair of the periodontium and oral tissues.
- The relationship between nutritional imbalances during tooth development and the formation of tooth lesions or defects that may increase caries susceptibility in children.
- The role of nutrition and nutritional status in the etiology and pathogenesis of dental diseases in older persons and other high-risk populations.
- The relationship between nutrition and both the immune and the nonspecific defense mechanisms of oral tissues and fluids.
- The most effective means to educate the public on the role of water fluoridation, diet, and dental care in preventing dental diseases.
- The mechanisms of fluoride action in the prevention of dental disease or osteoporosis.
- The effect of dietary factors such as vitamin A, vitamin E, and alcohol on the initiation and progression of oral cancers.
- Epidemiologic methods to determine the correlation between malnutrition and dental caries and/or periodontal disease.
- The role of calcium in the etiology and/or prevention of residual ridge resorption and periodontal disease.
- Estimation of the levels of fluoride from all sources in the diets of children.
- Estimation of the extent of dental fluorosis in the population.

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