THE ROLE OF FOODS AND NUTRITION IN DENTAL HEALTH

by

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INTRODUCTION

Oral diseases may be less critical and less to be dreaded than cardiovascular diseases or poliomyelitis, but they have the all important distinction of being universal. Every survey to date has shown that dental decay and periodontal disease, the two major causes of tooth loss, are the most prevalent of all diseases of mankind (1).

In the United States, dental caries has been described as the most frequent, chronic disease. Periodontal disease also is a widespread problem, particularly in the adult population, but to some extent among younger persons as well (2). According to the National Institute of Dental Research there are probably a billion unfilled carious teeth in the United States. There are over 100 million people who have lost teeth because of diseases of the gums and supporting structures (1).

In the National Nutrition Survey of 1969, numerous dental problems were apparent due to nutrient deficiencies of a combination of one or more nutrients such as protein, vitamins, and minerals (3). In addition, Ritchie (4) stated that malnutrition affects many of the developing countries; and among deficiency diseases, dental diseases are a growing problem.

While food and nutrition requirements to maintain general health are well understood by nutritionists, until recently, little was known about the effects of various foods and nutrients on specific tissues of the mouth. Scientists are now studying how foods and nutrition affect tooth development, dental caries, periodontal disease, oral malformations, and other diseases of the mouth (5).

The development of knowledge in oral biology has come about as a result of the application of some of the same research procedures used in the accumulation of knowledge in the science of nutrition. Four nutritional investigation methods used to obtain information concerning the relationship of foods and nutrition to dental health are: animal experiments, clinical trials, epidemiological surveys, and microbiological assays (6, 7).

Roth (1) stated

> it is rare indeed when a patient cannot be aided by proper nutritional guidance, since nutrition is probably the most important environmental factor affecting dental health and disease. The fact is that the same wholesome nutritious foods that are essential for good systemic health are also essential for good oral health. Coupled with good oral hygiene and periodic professional care, a proper diet can help maintain healthy teeth for a lifetime, and avoid other oral problems as well.

The purpose of this paper was to review the literature regarding the effects of foods and nutrition on dental health. Consideration was given to the factors: structure, composition, and development of the teeth and periodontium; dental caries, definition and mechanism, factors which promote dental caries, factors which inhibit or retard dental caries, and the cariogenicity of different foodstuffs; and periodontal disease, definition and mechanism, local factors, and systemic factors as related to foods and nutrition. The diets and nutrition of various age groups were also discussed.

MORPHOLOGY AND FUNCTION OF THE TEETH AND THE PERIODONTIUM

Knowledge of the formation and development of the teeth and periodontium is essential for inadequate nutrient intake may adversely affect these structures and impair their functions.

Tooth formation begins before birth. The tooth buds begin to form in the gum at about the sixth week of prenatal life. Calcification of the tooth bud is in process as early as the fourth to sixth prenatal month and advances rapidly until birth when a considerable portion of the unerupted deciduous teeth are in an advanced stage of development. In addition, calcification of the 4, 6-year permanent molars have also begun to calcify in the jawbones. By 2 1/2 years of age, the deciduous teeth of most children are fully erupted (8, 9).

Calcification of the permanent teeth, with the exception of the 6-year molars, begins between birth and 3 years of age. Eruption takes place between ages 6 and 13, and the enamel is completely formed about 3 years previous to eruption. The roots are completely formed about 3 years after eruption (8-11).

The teeth are hard calcified structures fixed in bony sockets in the upper and lower jaws. A tooth is divided into two parts: a root or roots which anchor it in the jawbone, and a crown, the part that is visible in the mouth (8).

A tooth is composed of 4 tissues: the enamel, covering the crown and functioning to resist abrasive wear; the dentin, forming the body of the tooth; the dental pulp, occupying the pulp chamber in the crown and the root canals in the roots; and the cementum, covering the root and an integral part of the tooth (2, 12).

The periodontium of a tooth includes the gingival tissues (located physically around the tooth), the alveolar bone (providing the bony socket and structure), and the periodontal membrane (consisting of ligaments attached to the alveolar bone and cementum). The periodontium functions to support the teeth in proper relationship to each other and to cushion the shock of the teeth coming together during mastication (2, 13). The structure and components of the tooth are illustrated in figure 1.

CHEMICAL COMPOSITION OF THE ORAL CAVITY

Knowledge of the chemical composition of the hard and soft tissues of the oral cavity is essential in order to better understand how nutritional biochemistry affects the development and preservation of these oral structures and tissues. Changes in the composition of the oral tissues may be related to nutritional problems concerned with dental caries and periodontal diseases (14).

The major inorganic constituents of enamel, dentin, alveolar bone, and cementum are calcium and phosphorous belonging to the apatite class of compounds. The primary organic matter is proteins, consisting mainly of collagen. The dental pulp is a soft tissue which is the remains of the formative organ of the dentin and contains a compact layer of odontoblasts. The gingival tissue is made up of epithelial and connective tissues containing collagen and mucopolysaccharides. The periodontal membrane consists of fibroblasts, collagenous fibers, nerves, and blood vessels (12-17).

DEVELOPMENT OF THE TOOTH

If there is adequate nutrition present, each tooth will undergo successive periods of development during its life cycle (9, 12), (figure 2):

1. Growth
    a. Initiation-beginning of formation of the tooth bud from the oral epithelium.

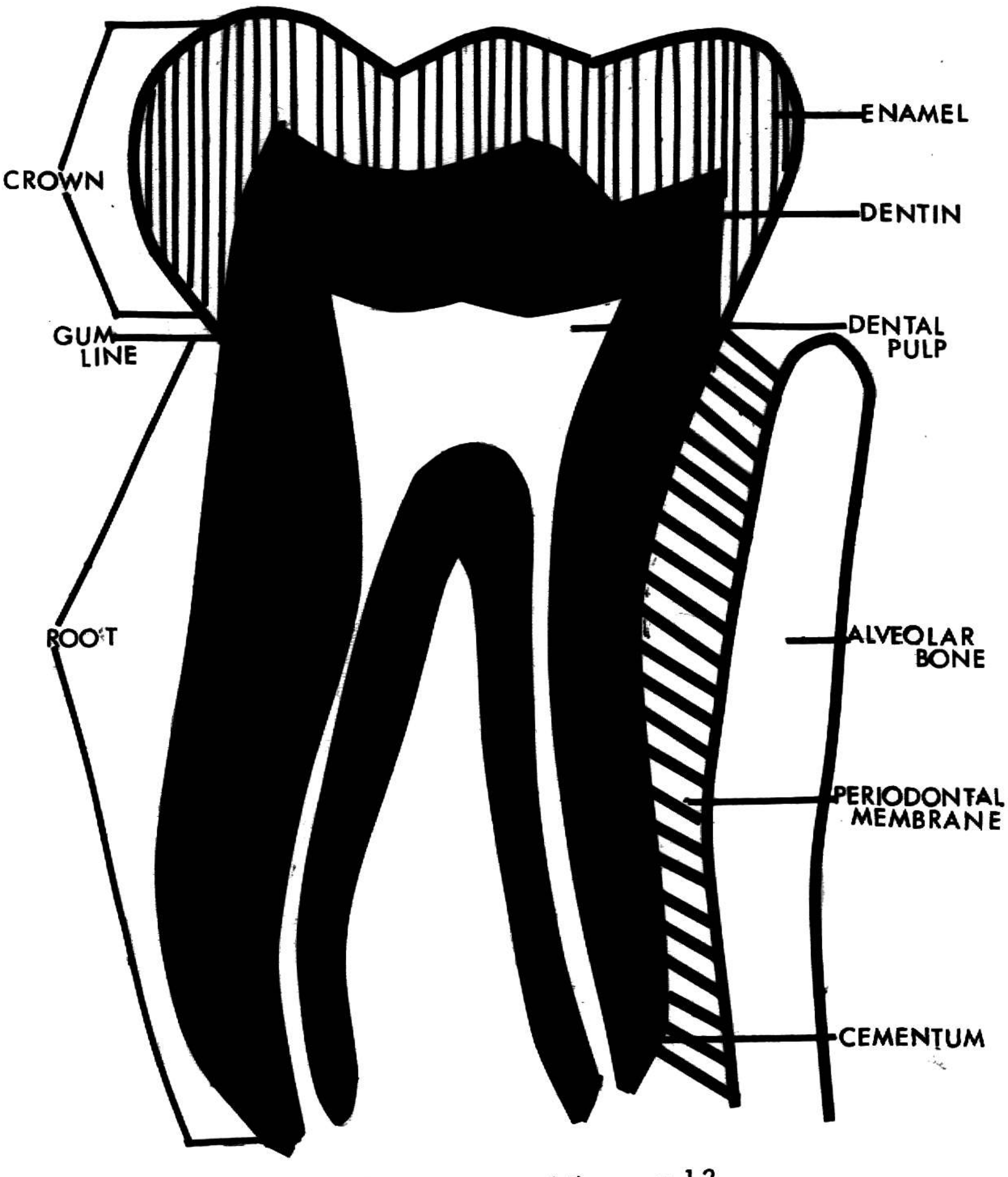

Fig. 1 The structure and components of the tooth.[1,2]

[1] Wilson, E. D., K. H. Fisher and M. E. Fuqua (10).
[2] Finn, S. (11).

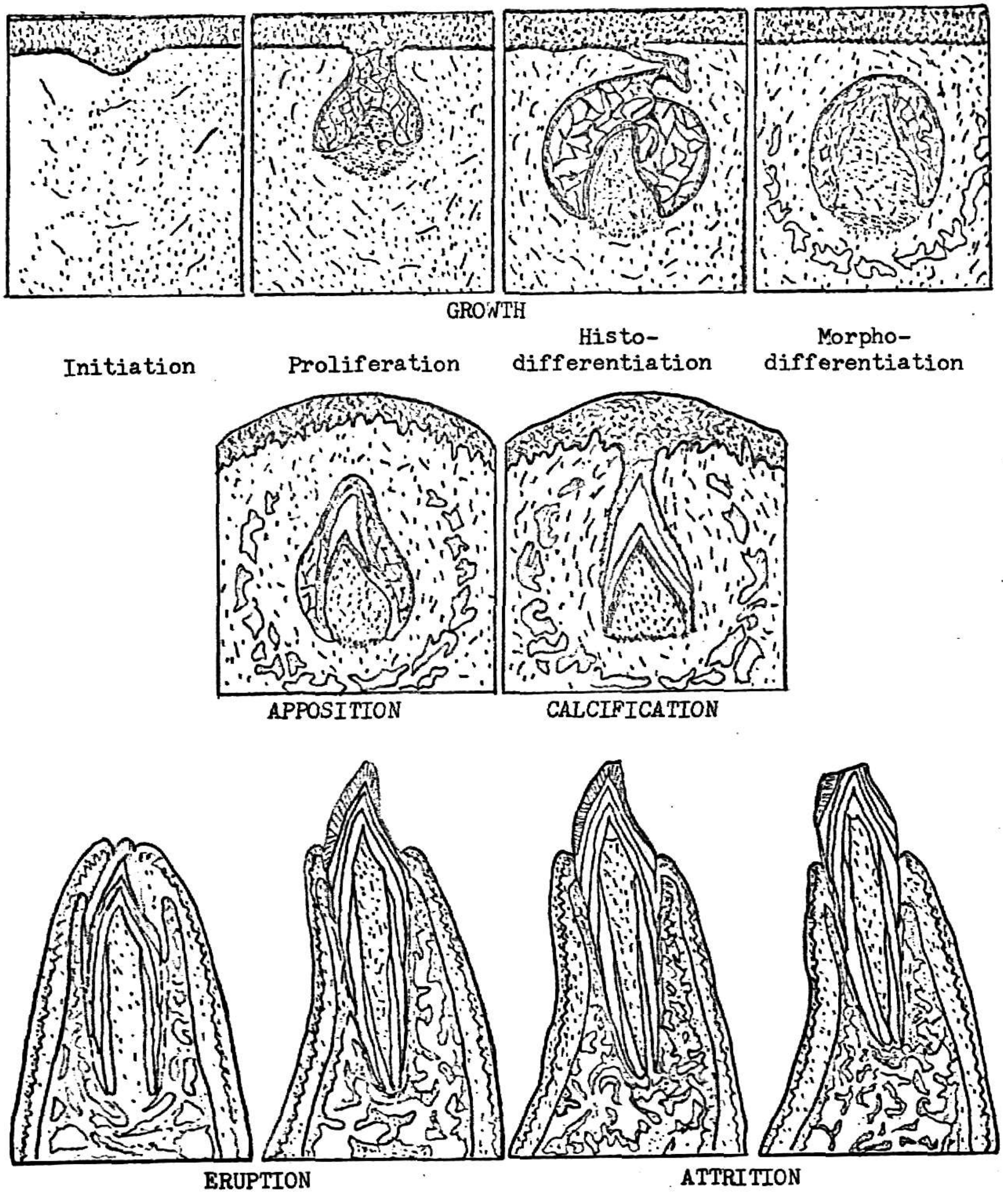

Fig. 2 Periods of tooth development.[1]

[1] Massler, M., and I. Schour (12).

b. Proliferation-multiplication of cells and elaboration of the enamel organ.

c. Histodifferentiation-specialization of cells-the cells of the inner epithelium of the enamel organ become ameloblasts (cells which take part in the elaboration of the enamel prism); the outer cells of the dentin organ (pulp) become odontoblasts (cells which form the outer surface of the dental pulp adjacent to the dentin).

d. Morphodifferentiation-arrangement of the formative cells along the future dentinoenamel and dentinocemental junction so as to outline the size and shape of the future crown and root.

e. Apposition-deposition of the enamel and dentin matrix in incremental layers.

2. Calcification-hardening of the matrix by the precipitation of calcium salts.

3. Eruption-migration of the tooth into the oral cavity.

4. Attrition-wearing of the tooth during function.

5. Resorption-removal of the tooth roots of the primary teeth by action of osteoclasts (cells responsible for the resorption process).

EFFECT OF FOODS AND NUTRITION ON TOOTH AND PERIODONTIUM DEVELOPMENT

Nutrition plays a vital role in proper development of the tooth and dental tissues. It is most important during the period of time when teeth are undergoing matrix formation and calcification. These processes can be influenced by maternal, infant, and childhood nutrition (18). In addition, the adequacy of the nutrients supplied the tooth during the formative periods influences its resistance to decay (10).

Research studies, both animal and human, amply substantiate the beneficial effects of an adequate diet in building a strong, healthy tooth and periodontium (10). As a result of these studies, proteins, calcium, phosphorus, and vitamins D, C, and A, have been implicated as playing a vital role in proper tooth and dental tissue development.

Nutrients

Protein. Alteration of the genetically established tooth structure, a decrease in caries resistance, smaller, missing and/or malformed teeth, and delayed tooth eruption have resulted in the offspring of maternal rats fed diets low or deficient in protein (19-21).

In regard to periodontal tissues, lower animal studies disclose that, in the presence of a protein deficiency in the maternal diet, degeneration of the connective tissues of the gingivae and other periodontal structures, and slowed cementum deposition occur in the offspring (22).

It has frequently been found that infants from mothers with low protein diets during pregnancy show formation of a poorly calcified dentinal and enamel matrix, an irregular predentin layer, alteration and irregularity in tooth structure, and delayed eruption of teeth (23). In children who suffer from protein malnutrition there has been noted rotation of the teeth, due to small or missing teeth, malformed teeth, delayed dentition and eruption, and decreased caries resistance (2, 4, 23).

Calcium, Phosphorus, Vitamin D. An appreciable portion of the primary dentition is formed in utero and, while the developing fetus is in a relatively protected environment, experimental evidence has been reported which indicates clearly that inadequate calcium, phosphorus and/or vitamin D in the maternal diet can have deleterious effects on the fetus (24). Calcium, phosphorus, and vitamin D deficiencies during and preceding the eruption of teeth undoubtedly account for faults in tooth structure (25).

Vitamin D is considered with calcium and phosphorus because one of its actions is to increase the absorption of calcium. After calcium and phosphorus have been incorporated into the teeth, they cannot be withdrawn, nor can new enamel be formed after the formative periods due to the degeneration of the ameloblasts (2).

Studies conducted on animals maintained on diets deficient in these nutrients disclose structural imperfections in enamel and dentin, hypoplastic teeth, and pitted and grooved permanent teeth (26, 27).

Human clinical studies have indicated that inadequate diets of calcium, phosphorus, and/or vitamin D during the mineralization of the teeth often cause defective matrix formation and calcification of deciduous and permanent enamel and dentin, poorly constructed teeth, retardation in eruption, hypoplasia, and slow development of the jaw bones (2, 16, 22, 24, 27, 28). In addition, the alveolar bone is sensitive to these minerals and vitamin deficiencies. It has been reported that the marrow spaces of alveolar bone become filled with uncalcified tissue; the teeth become loosened as a result of alveolar bone resorption and destruction of the periodontal ligament (27).

When too much vitamin D was fed to pregnant animals, it was found that there resulted a disturbance in the way the body handled calcium, and led to badly shaped jaws in the offspring and related faulty bite or malocclusion (28).

In the human, excessive vitamin D intake produced malocclusion, thinning of the enamel and dentin, pulpal calcification, and hypercalcification of alvelor bone, periodontal membrane and gingivae (22).

Vitamin A. Studies concerning Vitamin A deficiencies in both animals and humans attest the importance of this vitamin in tooth and periodontium development.

Animal studies have shown that a lack of vitamin A during the prenatal period changes the shape of teeth and causes delayed eruption (5). In the enamel organ of rats there is an atrophy and degeneration of ameloblasts if the animals are fed a vitamin A deficient diet. This dietary deficiency can slow down and even completely stop the growth of teeth. The odontoblasts are also exceedingly sensitive to vitamin A deficiency; and they, too, atrophy (28). In addition, the growth and development of jaws are affected which is manifest by crowding of the teeth and by roots that are stunted and thickened (29). The alvelor bone forms slowly and may even atrophy. Furthermore, the periodontal membrane forms irregularly, and the cementum is thickened (22).

Vitamin A deficiency in the human may cause atrophy of the formative cells of the enamel and dentin resulting in poor calcification and hypoplasia (22, 28, 30). However, Hypervitiminosis A may produce destructive results. An excess amount has been shown to reduce the rate of formation of the alveolar bone, and active osteoblasts become less prominent with the result that the bone becomes abnormally thin (28).

Vitamin C. It has long been established that ascorbic acid is essential for the proper development of the human dental tissues (16). With a vitamin C deficiency there is produced an irregular dentin or no dentin at all, enamel hypoplasia, and wasting of the alveolar bone. The pulp becomes enlarged, engorged, and dilated; and the pulpal vessels may eventually rupture (22).

After the teeth have been formed, the deficiency symptoms are confined to the periodontal tissues (16). The marginal gingivae become enlarged, enveloping and almost completely concealing the teeth, and hemorrhage on the slightest provocation. There is a lack of periodontal support due to a noteworthy lack of fibroblasts and collagen fibrils and engorged capillaries; the periodontal membrane fibers rupture readily, and the teeth become loose and may even exfoliate spontaneously (31, 32).

Diets of Various Age Groups

Morphologically, teeth cannot be changed once they are formed; however, their structure can be influenced significantly during the formative periods (33). Accordingly, the search for nutritional factors in oral health must focus on the maternal-prenatal diet, infancy, and childhood dietary intakes (5).

Nutritionally speaking, life starts at conception and not at birth. As the tissues of the fetus start to grow and develop they fulfill their individual requirements from the pool of nutrients available through the placenta of the mother, or her mammary gland, and finally through fetal metabolic apparatus. Yet, the importance of nutrition during pregnancy is poorly recognized by a large section of the population (31). Pregnancy makes many demands on the prospective mother, not the least of which are her nutritional needs and those of the unborn infant.

The protein intake must be increased because of its specific contribution to the growth of all tissues, and in particular, to the proper development of the dental tissues. The pregnant woman must also be supplied with calcium and phosphorus in quantities large enough for her own needs and those of the growing fetus for the proper formation of the teeth and bony framework of its body. Foods rich in vitamins must be supplied liberally in the diet of the pregnant woman if she is to meet her own nutritional needs as well as those of the growing fetus. However, special attention must be given to over supplementation of vitamins A and D (31). Dentists have noticed that maloclussion appears to be increasing in frequency and is perhaps more common today than ever before. During pregnancy there appears to be an excessive use of vitamin D beyond the 400 IU per day recommended. Many pregnant women take extra amounts of vitamin D-enriched milk plus vitamin supplements and may consume 2,000-3,000 units per day (5).

In addition, the nutritional status of the mother at the initiation of pregnancy is a critical point. Of concern is the increasing incidence of teenage pregnancies and inadequate diets often associated with this age group. Studies conducted on teenage girls have revealed that calcium, iron, and vitamins A and C are four nutrients often found deficient in their diets (34-36). Hinton et al. (37) found that the foods least consumed by teenage girls were milk, and vitamin C and A-rich fruits and vegetables.

Infants in the United States, both breast and bottle-fed, generally seem to be well nourished (38). Studies indicate that calcium, protein, phosphorus, and the B-vitamins are being adequately met. Vitamins A and D often exceed the RDA for this age group due to supplementations (39). This is of concern because of the effects of Hypervitaminosis on the development of the dental tissues. Vitamin C was found to be variable due to a late and reluctant introduction of citrus fruits and/or juices (38, 39). On the other hand, infants in other countries are not as fortunate as the majority of infants in the United States, and malnutrition is both prevalent and of serious consequence (25).

Another age group of importance with regard to dental development is the one to six year olds. Often calcium, vitamins A, C, thiamine and iron have been found to be below two-thirds the RDA in many studies while protein is quite adequate (40-45). This age group is turning more to table and adult foods, decreasing their food intake because of a decrease in appetite, becoming distracted by intellectual and social interests, and establishing many likes and dislikes (46, 47).

In 1968, nutritionists working with mothers and children in maternal and child health programs were asked to provide some information about current practices regarding food. It was revealed that, by one year of age, the majority of children were on table foods. Large amounts of breads and cereals, fats, and concentrated sweets (jelly, sugar, and soft drinks) as compared to meats, fruits, and vegetables were being consumed. Many children were apparently eating large quantities of foods that provided few of the essential nutrients other than calories (48). More emphasis should be placed on adequate milk consumption and fruits and vegetables.

As in the preschool group, calcium, vitamins A, D and C, and iron are the least well-supplied nutrients in the diets of the pre-teens ages eight through twelve years (36). Snack foods rich in these nutrients should be emphasized instead of free-sugar snacks often consumed.

DENTAL CARIES

Dental caries is probably the most common disease affecting the human race. This disease is increasing among technically underdeveloped nations and is rapidly approaching the severity levels found in affluent societies (33). Authorities estimate that only about 2% of the people of the United States have escaped having at least one dental cavity. Contrary to the regional and the endemic incidence of some other diseases, dental caries is so common that nearly everyone has experienced it (25).

Dental caries affects adults as well as teenagers and children. Accumulated data on the onset of dental caries in the primary dentition indicate that at one year of age 5% of the children have dental caries. This increases to approximately 10% at two years of age. There is a further rise so that by the third and fourth years, 40 and 55% of the children, respectively, have tooth decay. This trend continues and at age five, three out of four preschool children have carious primary teeth (49).

Likewise, a considerable number of surveys have been made on dental caries experience in the permanent dentition. These studies are in general agreement that at age six, 20% of the children have experienced tooth decay in their permanent teeth. This increases rapidly so that at ages eight and ten, 60 and 85% of children, respectively, have been affected by dental caries. At age twelve, when most of the permanent dentition has erupted, over 90% of school children have experienced tooth decay (49).

Therefore, since dental caries was and is the most prevalent chronic disease in man, it can be readily understood that the maximal effort in dental research has been directed and expanded here. It was the first dental disease associated in a scientific fashion with foods and nutrients (50).

Dental caries is a destructive, demineralization process, in the main, of the enamel and dentin of the tooth (50). The primary lesion begins at the tooth surface and, if not arrested or removed, progresses inward, ultimately involving the pulp (51). It is initiated by organic acids formed by the action of bacterial enzymes on fermentable carbohydrates with the acids progressively destroying the portions of the tooth. The bacterial enzymes adhere to the enamel surface of the tooth in the form of dental plaque. The surfaces of the teeth rapidly collect dental plaque which is a glue-like substance comprised principally of microorganisms (50, 51).

Although the dental caries process is not completely understood, there is general agreement that three major factors must be given consideration in order to understand the carious process. The interplay of the various factors associated with the etiology of caries is illustrated in figure 3. Caries depends on the presence and simultaneous operation of a cariogenic microflora (particularly on the tooth surface), a favorable substrate (carbohydrate foodstuffs in general, and sucrose, in particular and in local contact with the tooth), and a susceptible tooth surface. The fermentable carbohydrates and the oral enzymes may be considered as attack forces, the tooth surface as the resistance force (51).

Factors Which Promote Dental Caries

Microbial Factor. As a result of accumulated research concerning the role of microorganisms in dental caries, the Chemico-Parasitic Theory of

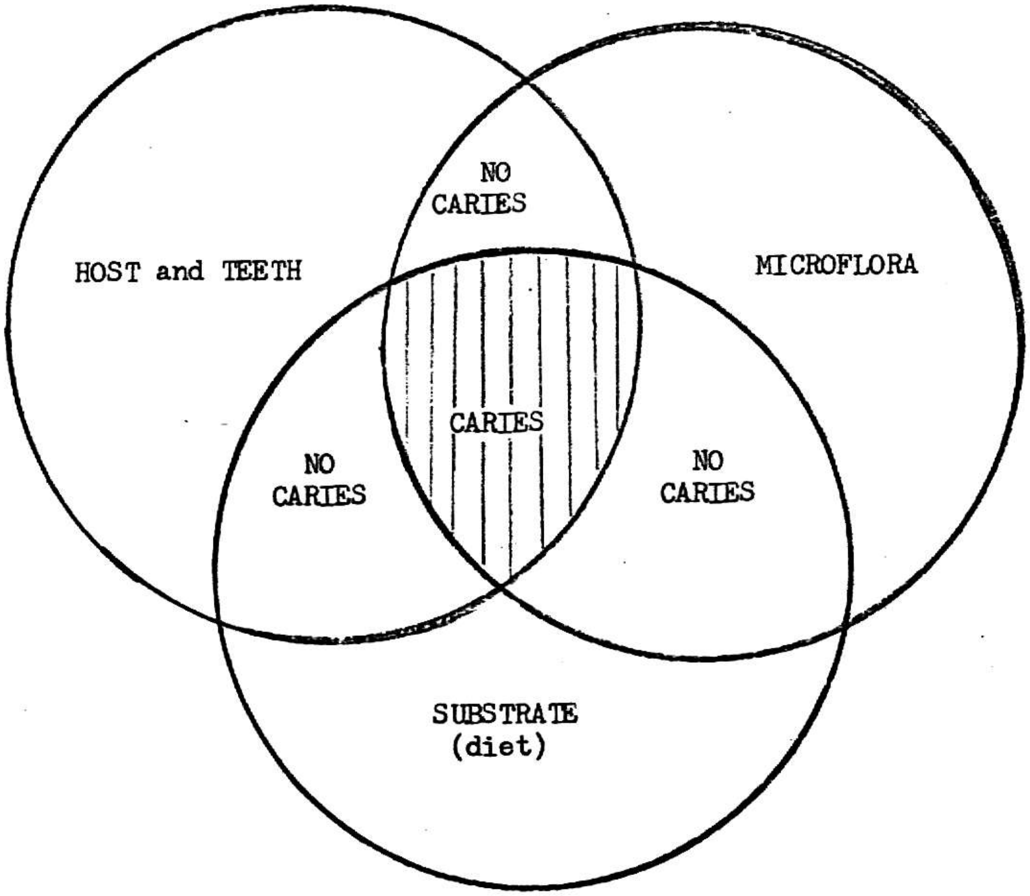

Fig. 3 The interplay of factors in caries.[1]

[1] Volker, J. F., and R. C. Caldwell (51).

dental caries was formulated. This theory states that fermentable carbohydrates are acted on by oral microorganisms to form organic acids which destroy the portions of the teeth (51, 52). However, the assumption cannot be made that all microorganisms are equally important in this connection. The most causative microorganisms seem to be the acidogenic bacteria--lactobacilli, staphylococci, and streptococci (51).

Carbohydrates. Of particular importance to food scientists and nutritionists is the carbohydrate factor. For centuries it has been noted that persons ingesting diets containing appreciable quantities of starches and sugars tend to have moderate to excessive tooth decay. It has also been noted that individuals subsisting on diets that are made up primarily of fats and proteins have little or no dental caries (51). Primitive people, as a rule, show a lower incidence of dental caries than do civilized populations. However, as soon as primitive populations are touched by civilization; and native foods are replaced by processed, sophisticated foods, in general, and refined sugars, in particular, an increase in dental caries follows (25).

The human pattern and trend of carbohydrate consumption are influenced by the ease and cheapness of production of foodstuffs that are rich in carbohydrates and the general affluence of the society. With higher incomes there is a corresponding increase in consumption of refined sugar. In the wealthiest countries, sugar contributes nearly 20% of the total calories consumed. Compared to other food commodities, the world production of sugar is increasing most rapidly. The average consumption of sugar has increased nearly threefold since the beginning of the century (52).

Through epidemiological, clinical, microbial, and experimental studies, it has been established that:

1. A carbohydrate substrate in the diet is necessary for oral bacterial action (6, 52).

2. Actual contact between the carbohydrate and the tooth is necessary for the production of dental decay. This fact indicates clearly that the action of fermentable carbohydrates in producing tooth decay is a local one, disproving the once-held notion that systemic carbohydrates can initiate the carious process (51, 53, 54, 55).

3. The cariogenic effect of carbohydrates is accentuated when its feeding is initiated during early tooth formation, development, and calcification. The carbohydrate may be reducing the protein content of the diet with subsequent damage to tooth structure and enamel or be replacing a protective mineralizing factor present in trace amounts, perhaps, in natural foods which are more beneficial to the tooth during its maturation (4, 56, 57).

4. The chemistry of the carbohydrate, mono-, di- or polysaccharide, is an essential consideration with respect to the degree of its cariogenic action. For example, sucrose or glucose are very active cariogenic agents, but starches and dextrins are not. The reason that starch may not be as cariogenic is that the large starch molecule does not penetrate and diffuse through the dental plaque so readily as the smaller di- and monosaccharides. Starch is more likely to remain on the outer surface of the plaque where it can be washed away and eliminated. On the other hand, the sucrose that has diffused through the plaque will degrade to acid beneath the plaque and react with the tooth surface (52, 58).

5. The higher the carbohydrate content in the diet, the more caries that result (6, 59, 60).

6. The physical form and composition of the carbohydrate influence the extent of caries production because of the factor of food retention in the mouth and the oral clearance time. Sugar in solid form has been shown to be more cariogenic than liquid sucrose. Carbohydrates with a strong tendency to be retained on the tooth are more cariogenic than nonretentive carbohydrates (51, 52, 54, 61).

7. The more frequent the ingestion of carbohydrate foodstuffs, the greater the increase in caries development (2, 51, 52, 61).

Factors Which Inhibit or Retard Dental Caries

Tooth Structure and Composition. Teeth that are well-formed and calcified seem to have a higher resistance to decay than do teeth that are poorly formed because there are not present tooth composition alterations and surface imperfections that favor the accumulation of carbohydrates and microorganisms predisposing the tooth to the destructive action of dental caries (8).

The fully formed and erupted tooth, particularly the enamel, is relatively, but not completely inert. The chemical composition of the calcifying tooth can be modified to a certain extent. The discovery that inclusion of optimal quantities of fluoride in the calcifying tooth reduced caries susceptibility confirmed this fact. Studies with radioactive isotopes support the theory that little replacement of the enamel is accomplished via normal metabolic pathways, i.e., progressively through the pulp and dentin. However, the same techniques revealed that the mineral components of the enamel surface are being constantly replaced or added to by three sources: the normal oral environment; materials that are introduced into the mouth ordinarily in eating and drinking; and materials that are introduced for therapeutic purposes (61).

Probably the most effective nutrient for the prevention of dental caries which can alter tooth composition is fluoride. It is by far the most important preventive nutritional measure available (50, 62).

Fluorides in solution or in rapidly soluble salts are absorbed almost completely from the gastrointestinal tract. During and following its absorption in small quantities, fluoride is in part rapidly excreted by the kidneys and in part stored in bone or developing teeth. As intake of fluoride increases, absorption also increases (63). Because of their relatively small mass, the teeth serve as storage sites for only a small fraction of retained fluoride. However, the teeth have a high affinity for the fluoride ion and are reluctant to mobilize any substantial amounts to circulation. It appears that the fluoride deposited in teeth is not subject to appreciable reabsorption (64).

A general knowledge of the deposition of fluoride in the developing tooth is necessary in order to understand the role of this nutrient in relation to the prevention of dental caries. The unit cell, or smallest repeating unit in the crystals of teeth is $Ca_{10}(PO_4)_6(OH)_2$. All these ions may be partially replaced by other ionic species particularly on the surface. Fluoride can substitute for the hydroxyl groups found in the apatite crystals. When the hydroxyls are completely replaced by fluoride, fluorapatite is formed, $Ca_{10}(PO_4)_6\ F_2 + 2\ OH$. Fluorapatite is less soluble than hydroxyapatite, and its formation may partially explain the cariostatic effect of fluoride (62, 63).

The acquisition of fluoride is confined almost entirely to the surface of the crystals and does not involve the body of the tooth. Thus fluoride is highest in the outermost layer of enamel. Diffusion of fluoride from the surface inward is restricted to the outer portion of the enamel, since the body of the enamel becomes inaccessible as a result of high mineralization. The uptake of fluoride is known to depend on the amount of fluoride ingested and on the time of fluoride exposure. As the fluoride intake increases, such as in water fluoridation, the fluoride concentration in the enamel rises; therefore markedly increasing the deposition of fluoride in the outermost enamel layers (62, 64). The process of fluoride deposition begins while the enamel is calcifying and continues through the pre- and post-eruptive life of the tooth, although to a lesser extent after post-eruption (64).

The mode of action of fluoride and dental caries is yet not fully understood. Since dissolution of mineral is a predominant phase of the carious lesion, and the dissolution is brought about by bacterial metabolites; fluoride could inhibit caries by increasing the resistance of the enamel to demineralization or by exerting antibacterial effects. However, the quantity of fluoride used is much too small to inhibit bacterial metabolism (63). Numerous studies have shown that deposition of fluoride in the enamel will retard dissolution of the enamel mineral and will increase the resistance of the enamel to demineralization. This ability to slow down the dissolution process and to increase enamel resistance is commonly believed to be fluoride's mode of action in inhibiting caries (62-64).

In carious lesions, there is an alternating process of demineralization and reprecipitation. During the formation of the carious lesions, a number of calcium phosphate compounds may form in addition to apatite. They are more soluble than hydroxyapatite and will precipitate in the carious environment; but in the presence of sufficient amounts of fluoride, only apatite can form. Fluoride thus has the ability to induce formation of apatite under conditions which, in its absence, would favor formation of more acid and more soluble compounds. The critical role of fluoride in this environment appears to be that of favoring recrystallization or reprecipitation of hydroxyapatite. Fluoride can also penetrate the partly demineralized enamel of initial carious lesions, hence retarding the growth of the carious lesions (62-64).

Since it appears that dietary fluoride provides its greatest benefit during the period of tooth development, it may be assumed that the child should receive adequate fluoride from infancy until at least 12 to 14 years of age. By this time, the crowns of all teeth, but the third molars, should be completed and more than half of the permanent teeth will have erupted (63). Among the authorities who have studied the problem, it is agreed that the simplest, the cheapest, and the most far-reaching method of ensuring adequate fluoride is through the fluoridation of the drinking water. This procedure will supplement, but not supplant other dental health measures. Controlled water fluoridation has been shown to reduce the incidence of caries by 50 to 70% among young people whose teeth were formed during the period of water fluoridation, and that a level of fluoride which seems to be protective without being harmful is 1.0 parts per million of drinking water (62-64).

Another important nutrient recently found to play an active role in caries prevention is phosphorus (50). During the past decade, more than 150 studies have been conducted, and the results are nearly unanimous in indicating that various types of inorganic and organic phosphates are effective in preventing the development of caries (27, 65). The results are more conclusive in animal studies with regard to the addition of various types of phosphates to otherwise cariogenic diets (27, 65, 66). However, encouraging preliminary human trials of phosphate-fortified breakfast cereals and the dietary addition of an organic phosphate compound, such as casein, resulting in cariostatic properties have been conducted (2, 50). Although the human results are not conclusive, this is a hopeful area of research (2). Even at the present time, Lossee (19) has stated, "the ingestion of fluoride or a high-phosphate diet during tooth development can result in alterations in caries susceptibility."

Present evidence indicates that the action of phosphates is primarily a local one, affecting the surface layers of the teeth, rather than a systemic effect involving the body's digestive system (27, 50). The mechanism of action of phosphorus is believed to be different from that of fluoride; however, the caries controlling actions of these two elements are improved synergestically when administered together (50).

Fats and Proteins. The foods that are available to man are fats, proteins, and carbohydrates. There is reason to believe that both fats and proteins are factors which inhibit or retard dental caries (50). Research is available which suggests fats are associated with caries inhibition, but it is only recently that any substantial scientific information has been accumulated concerning a relationship between proteins and dental caries (61).

Evidence that dietary fats have a limiting influence on dental caries has been noted in both human and animal studies (27, 61, 67). It has been observed that dental caries decrease when increasing amounts of fat are added to the diet (22, 66, 67). Although the mechanism of the action of fats is not conclusively known, it has been suggested that the inhibition mechanism is a local one. Since fat tends to form an oily film on the tooth, its effectiveness may be that it acts as a barrier helping to keep carbohydrates from the bacteria in the plaques on the tooth and also lessening the retention of foods on the surface of the tooth by coating the food particles with an oily film which would prevent their direct contact with either bacteria or acid (12, 61, 67).

Concerning protein, it has been reported that dental caries in rats is accelerated when the diets are heat treated, destroying some of the protein (68). Furthermore, a cariogenic diet may be made cariostatic by the addition of moderate to high amounts of protein (2, 68, 69). Thus the quantity of protein in the diet is important with regard to caries inhibition (2, 69). Nizel and Shulman (50) have postulated that if dietary proteins are ingested in sufficient quantity, there may be enough amines and urea to buffer the acids and prevent the decalcification occurring in the dental caries process.

Saliva. Although oral microorganisms and retained carbohydrates are etiologic factors in dental caries, it must be remembered that each of these exists in an environment in which saliva is always present. Accordingly, it is conceivable that the chemical properties and rate of flow of saliva may influence dental caries susceptibility (2).

The buffering capacity of saliva may have an effect on the acid-carious process (14). The saliva may be capable of neutralizing considerable acid before the H ion concentration is altered to a point at which enamel is likely to be dissolved in appreciable amounts. The buffering capacity of saliva is primarily due to the presence of bicarbonate (51).

Saliva also contains appreciable quantities of calcium and phosphorus. Under certain circumstances, these inorganic ions in saliva may combine to form insoluble precipitates (51). There is general agreement that the inorganic ions in saliva are constantly being exchanged with those of the tooth surface or are being absorbed thereon (14, 51). If the salivary calcium and phosphate are available, they might combine with the tooth surface in such a fashion as to maintain its integrity (5, 51).

The rate of salivary secretion has been noted by some workers to be an important factor in caries etiology (14, 51). Increased dental caries may be related to diminished salivary flow whereas decreased dental caries may be related to increased salivary flow (2).

Although it is not clear how nutrition and the foods consumed affect the chemical composition and rate of flow of saliva, there is a relationship (24, 51).

CARIOGENICITY OF DIFFERENT FOODSTUFFS

Because foods and nutrition are involved in the dental caries process, dental caries; therefore, is of major importance from a nutritional standpoint. Reduction and prevention must be the aim and goal of tomorrow (6). Special attention should be directed towards the nutrient, carbohydrate.

Since it is impracticable and undesirable to eliminate carbohydrate foodstuffs from modern civilized diets, the most realistic approach to reducing their harmful effects and the prevention of tooth decay rests in substituting less destructive carbohydrate-containing foods for those which are most destructive and by reducing the frequency of their ingestion (70).

Two methods of research have been developed to ascertain the cariogenicity of different foodstuffs. These are the Caries Potentiality Index (CPI) and the Decalcification Index (61).

Caries Potentiality Index measures the amounts of food retained in the mouth. Since food retention on the tooth surface is an important part of caries initiation, it is logical that foods which are retained on the surface in the greatest amounts would have the greatest potentialities for caries production. The CPI, therefore, measures the sugar content of the saliva after ingestion of a food and the time necessary for varying amounts of the sugar to be cleared from the saliva. This is done by observing the time in minutes after the food is ingested that the total sugar content in the saliva exceeds 0.02%, 0.2%, 2%, and 20%. The times for each per cent are totaled and designated as the CPI (61). An abbreviated summary of the CPI data collected in a study conducted on various foodstuffs is presented in table 1. The CPI of these foods was predictive of the amount of tooth decay that developed in the experimental subjects when these foods were added to their dietaries (61).

Other studies have been conducted on the CPI of different foods, and there is a similarity in the findings. In general, sticky foods, such as dried fruits or caramels, show greatest retention; and starchy foods, such as breads and potatoes, occupy an intermediate position, with fruit juices, fresh fruits, and beverages being lowest (70).

Since the initial stage of caries is primarily a process of enamel decalcification, the amount of decalcification produced by foods retained on the tooth incubated with saliva has been researched. The product of the quantity of food retained on the tooth multiplied by the quantity of the acid formed after salivary incubation of this quantity of adhering food is designated as the Decalcification Potential (61). The most common finding is that the less refined cereals and sugars produce less decalcification than the more refined forms (70-72).

It is difficult to draw clear-cut conclusions to indicate the comparative caries-producing capacities of foods. However, the most

TABLE 1

Caries Potentiality Indexes of Representative Foods[1]

Food	Total Sugar %	Sugar Concentration in Saliva: Maximum %	Sugar Concentration in Saliva: Av. Clearance Time (Min) Above 0.2%	Caries Potentiality Index
Caramel	64.0	18.8	5	27
Honey + Bread + Butter	19.0	4.6	7.5	24
Chocolate, Light	47.5	10.1	6.25	21
Honey	72.8	5.6	5	18
Sweet Cookies (biscuits)	9.0	1.9	5	18
Danish Pastry	30.0	2.4	2.5	13
Wheaten Bread	12.3	2.8	4	13
Ice Cream	2.4	3.2	2.5	9
Marmalade	65.3	3.5	3.5	10
Marmalade + Bread + Butter	16.3	1.8	2.5	9
Potatoes (Boiled)	0.8	1.6	2	7
Potatoes (Fried)	3.9	0.4	2.5	7
White Bread + Butter	1.5	0.8	2	7
Coarse Bread (rye) + Butter	2.3	1.3	2	7
Milk	3.8	0.6	2	6
Apple	7.5	0.4	1	5
Orange	6.5	0.3	1	3
Fruit Juice	11.5	1.2	1	3
Lemonade	9.3	0.5	0.75	2
Carrot (Boiled)	2.4	0.1	..	1

[1]Volker, J. F. and R. C. Caldwell (61).

consistent findings reveal that sugar in liquid form is less conducive to caries than when used in a solid or semiliquid state; less refined cereals and sugars are less conducive than more refined forms; sticky forms of carbohydrate have more caries potential than less adhesive forms; and foods such as meats, fish, eggs, and fibrous foods such as vegetables, and fruits are less conducive to caries production (52, 61, 70).

It is generally believed that fibrous foodstuffs exert a detergent effect during mastication, resulting in improved oral hygiene (61, 70). The fibrous foods are retained on the teeth in smaller amounts and conceivably can dislodge some food sticking to the tooth (2, 5, 61). Therefore, fresh fruits and vegetables and other fibrous foods are preferable to sticky foods at the end of a meal or between meals.

Much emphasis has been placed on the carious potential of various foodstuffs, however, of equal concern is the frequency of eating sugar and carbohydrate-containing foods. The importance of this factor is validated by numerous studies (70, 71).

Diets and Dietary Habits of Various Age Groups

The diets and dietary habits of children and adolescents are of particular importance with regard to the prevention and reduction of dental caries. Between-meal eating is a characteristic of these age groups; but this practice should not be discouraged, rather, the type of foods eaten between meals must be emphasized (34, 37, 46, 47). Children have been shown to eat four to six times a day and seem to need these dietary additions (46, 47). Also, snack foods make up a large proportion of the adolescents' diets and play an important role in maintaining the nutrients needed for the rapid growth spurt between the ages of 14 to 18 (34). Yet, unnecessary carbohydrate consumption between meals should be eliminated in the diets of children and young adults and stress placed on the detergent foods as snack items. While the impacting foods, those that require little chewing and that tend to cling to the teeth and pack into the fissures and grooves, should be discouraged. Among such foods are cookies, crackers, candies, chewing gum, lollypops, and sandwiches with jams and jellies (2). In addition, the "rewarding" of children with confections at home, at school, and in professional offices presents another hazard. Particular attention should be placed on the rewarding of children with candy at school and at doctors' offices. Both parents and children alike are apt to construe such a gift as professional approval of this form of sugar ingestion (1).

Another characteristic of the adolescent's diet and dietary habits is the frequency of omission of breakfast and other meals (35). A breakfast of high carbohydrate and low-protein content, the omission of breakfast, or the skipping of or eating poorly at other meals can influence the increased consumption of free-sugar snacking during the day, increasing the caries potential (2, 73, 74). A well balanced diet is a major factor in the prevention of caries for when such a diet is eaten in adequate amounts, there is little room or a desire for sweets (74).

Not only should attention be directed towards children and adolescents, but the diets and dietary habits of adults are also of importance with regard to the prevention and reduction of dental caries. One must take into consideration the between-meal snack habit of adults exemplified by the mid-morning and mid-afternoon coffee breaks accompanied with Danish pastry, cake or other forms of carbohydrate-containing foods that are pernicious and productive of caries. Likewise, the use of sucking candies and chewing gum could be very important today because adults are using more of these candies in an attempt to stop the smoking habit (1).

It would be difficult, if not impossible, to eliminate all sugar from the diet. It should be possible, however, to limit the amount of sweets consumed, particularly between meals. Children, adolescents, and adults should be taught that sweets should be eaten with meals and that the teeth should be brushed immediately after meals and between-meal snacks (8). A reduction in the frequency of sugar intake is more important than in the amount consumed since there is a fresh attack upon the teeth by the acid each time sugar is eaten (2).

PERIODONTAL DISEASES

The several tissues, gingivae, periodontal membrane, and alveolar bone, that surround and support the teeth are subject to various disease processes, termed collectively periodontal disease. When periodontal disease occurs, the tissues may be damaged (75).

The processes of periodontal disease are inflammation of the gingival tissues, hyperemia, edema, pain produced by pressure, bleeding, and finally loss of function because of the disruption of normal cellular function (75, 76). In some areas of inflammation, the tissues degenerate to expose the root of the tooth. If the inflammatory condition is both prolonged and severe, the tissues of the gingivae, periodontal membrane, and the alveolar bone are affected by considerable loss of their structures resulting, perhaps, in loss of teeth because of failure of support (75).

The term "periodontal disease" has been associated with the terminal stages of the disease: tooth loss, pus formation, and severe bone loss. These symptoms often are detected only in later years, and this has led to the common assumption that periodontal disease is a degenerative disease of adults over 35 years of age (5, 76). However, periodontal disease does not exist only in adults. The disease is slowly progressive, extending over many years; and the early stages are extremely common in children. Severe periodontal degeneration is unusual in children, but even at this age a few cases present serious problems (1, 76).

Despite numerous clinical and fewer laboratory observations made over many years, there is still much disagreement among clinicians and investigators as to the definition and nomenclature of the various diseases affecting periodontal tissues, as to their etiology and pathogenesis, and consequently, as to the methods for their treatment (75).

In the field of relating foods and nutrients to gingival and periodontal health in man and animals, the state of knowledge is limited. It is at the stage today that dental caries research was 35 years ago. The correlation between nutrition and periodontal disease is only on the threshold of discovery (6).

As of the present, it is believed that systemic conditions, mainly nutritional deficiencies, and general health per se are seldom the entire cause of any periodontal disease; but they may predispose the tissues to disease in the presence of mild, local factors, modifying the reaction of the tissues to the local irritation (75, 76). Periodontal disease in all age groups is primarily caused by local conditions (76). Such unfavorable factors are maloclussion (faulty bite), impacted food and other debris accumulated about the teeth, the physical nature of the diet, accumulation of calculus (hard masses of calcified material adhering to the teeth), and the oral microflora of the immediate environs. All the local factors are believed to act by trauma or irritation of the periodontal tissues, which together with the action of the ever-present microbial flora, produce the periodontal lesions (75). Much attention is focused on the local factors to the etiology of periodontal disease; however, the systemic factor must not be deemphasized for, since nutrition helps to determine the integrity of the periodontal tissues, it is a significant factor in the progression of periodontal disease (76). Burnett (75) stated, "Systemic and local factors cannot exist apart but must necessarily act in concert."

Local Factors

Oral Microflora. The oral microbial flora is the factor, perhaps more than any other, which mediates periodontal disease (75). The oral cavity swarms with bacteria living a precarious existence on the surface of the tongue, mucous membranes, and teeth. With each replenishment of food about the teeth at the end of a meal or between meals, another source of food is available to the bacteria, which multiply accordingly. Teeth and tissues are remarkably resistant to bacterial products; but where excess acid is formed on the tooth surface, the tooth suffers; and where massive accumulations are present about the tissues, the constant supply of bacterial products and by-products cause an irritation and inflammation. Bacteria capable of digesting any type of food which may lodge about the teeth are always present in the mouth, and digestion of debris upon the tissue surface is only one step removed from the digestion and damage of the periodontal tissue themselves (76, 77).

Calculus and Accumulated Debris. Accumulations of debris, consisting mainly of bacteria and epithelial cells, and calculus about the teeth and on tooth surfaces act as irritants on the periodontal tissues causing continual inflammation. Therefore, these accumulations should be removed by proper and regular tooth brushing and by having the teeth cleaned regularly by a dentist (8).

Malocclusion. Faulty bite or considerable protrusion of the upper teeth make it difficult for vigorous chewing of food, affecting the cleanliness of the oral cavity considerably. Hence, debris, food impaction, and calculus about the teeth will often accumulate resulting in irritation of the periodontal tissues and acceleration of microbial action leading to the development of periodontal lesions (75, 76).

Physical Properties of Food. The physical nature of the diet is an important local factor mediating periodontal disease. The gingivae are scrubbed and kept clean from debris by mastication of food. The tissues are perfectly adapted to this heavy function by their position, contour, and structure. However, irritation above that which is tolerated by the tissues often occurs. Adherence of debris about the teeth is the most common cause, and the physical nature of foods is perhaps the most important factor influencing adherence (77).

The texture of the foods eaten greatly influences the effect on oral tissues. Soft foods, which call for a minimal amount of chewing, tend to impact between the teeth and around the gingivae causing irritation and providing the oral microflora the nutrients needed to produce acids and plaque material (75). Impacting foods encourage food retention and do not provide stimulation to the periodontal tissues (77). Therefore, as in dental caries, such impacting foods as soft mixtures, sticky candies, cookies, and crackers should be avoided between meals or be eaten with the meal; and tooth brushing should follow immediately (2).

On the other hand, fibrous and detergent-type foods help maintain a clean oral cavity and exert a beneficial effect on the periodontal tissues (77). They require vigorous and thorough mastication which cleans the teeth, decreases the rate of calculus deposition, removes food particles and debris, and exercises and stimulates the periodontium, maintaining their integrity (2). Fresh, crisp fruits and vegetables and unground meats are to be encouraged at meals and as between-meal snacks and should not be followed by sticky mixtures (76). Unfortunately most modern diets do not stimulate the periodontal tissues sufficiently to maintain them in a healthy state. If they are not maintained, the equilibrium between the tissues and the oral microbial flora will be shifted in favor of the bacteria, resulting in infection and inflammation (75).

Besides the physical nature of foods, carbohydrates may possess periodontal disease-producing potential. The knowledge of the relation of carbohydrates to periodontal disease is still sparse, but the initial experimental results seem to hold some promise for developing a tenable hypothesis. With an increased ingestion of carbohydrate foods, particularly those that are readily retained and easily fermented, the incidence of periodontal disease also increases (52, 75, 78). It is felt that the periodontal disease produced by a carbohydrate regime is a reflection of bacterial activity and the availability of optimal nutrition for the growth of these microorganisms. These microbial agents are less able to operate in a carbohydrate-free or carbohydrate-low oral environment (52, 78).

Systemic Factor

Although periodontal disease is primarily caused by local factors present in the oral cavity, periodontium response to bacterial and physical irritants is profoundly altered by systemic conditions. Tissue cells rely on a constant supply of materials for their metabolism. These include vitamins, minerals, oxygen and other nutrients. An alteration in the level of any of these substances may be the cause of severe local disturbance. Local irritations which are tolerated or which produce only a mild reaction under normal circumstances, may give rise to severe inflammation and destruction if the cells are denied the materials they require for repair. Normal, healthy tissues have ample reserves so that heavy function and local damage produce an unnoticeable reaction; but where deficiencies are present, even light functional stresses may cause considerable local reaction followed by degenerative changes (75-77).

Both animal and human studies indicate that when nutrition is poor, the periodontal tissues succumb more readily to infections and irritations; and once started, periodontal disease progresses rapidly (2, 76, 77). Of the nutrients tentatively observed to be related to the periodontal disease process are proteins, calcium, phosphorus, iron, and vitamins D, A, C, and the B-complex (75).

Proteins. Lower animal studies disclose that, in the presence of a protein deficiency, there is degeneration of the connective tissues of the gingivae and other periodontal structures, slowed cementum deposition, extensive calculus formation, gingival inflammation, and severe absorption of the alveolar bone. Yet when protein is replaced in their diets, a major reduction in periodontal disease occurs (2, 76).

Unfortunately, human studies concerning protein deficiencies are incomplete and inadequate; and hence, no definite statements can be made (75). However, protein deficiency or deprivation affects the activity of matrix-forming cells, namely fibroblasts, osteoblasts and cementoblasts. Therefore, microscopic findings such as atrophic and degenerative changes in the connective tissue of the gingivae and periodontal membrane, increased size of bone spaces, less than normal amounts of osteoblasts, and retardation in deposition of cementum are to be expected (23). Proteins also play a significant part in the reaction of the body to infection. They are essential to preservation of a reserve of phagocytes which ingest and destroy bacteria and thus may affect the local factor so prominent in the process of periodontal lesions, the oral microflora (77).

Calcium, Phosphorus, and Vitamin D. Deficiencies of calcium, phosphorus, and vitamin D affect the alveolar bone and periodontal membrane. The marrow spaces of alveolar bone become filled with uncalcified tissue, and the teeth become loosened as a result of alveolar bone resorption and destruction of the periodontal membrane. Malocclusion may also result (27).

Iron. Iron deficiency anemia may partake in influencing the course of periodontal disease. All oral mucous membranes are atrophied, affecting their proper maintenance (79).

Vitamin A. The primary effect of vitamin A deficiency is on the normal development and functioning of the epithelial tissues lining the mucosa of the oral cavity, causing cell atrophy; and therefore, resulting in irregular periodontal membrane formation, cementum resorption, destruction of the alveolar bone, and hyperplasia of the gingival tissues (23, 28, 75, 77, 78, 80).

B-Complex Vitamins. The B-complex vitamins function in regulating normal processes of the body involving body maintenance and hence periodontal tissue integrity (81). Edema or emaciation of the gingivae tissue may occur in thiamine deficiency; riboflavin deficiency is manifested by a reddening of the mucous membrane and lesions of the mouth. Swelling of the gingivae and reddening of the mucosa are also revealed by a deficiency in niacin. A decrease in leucocytes, which are one of the bodies chief defense mechanisms against microorganisms, occurs with a folicin deficiency. Therefore, a specific or generalized deficiency of the B-complex vitamins may weaken the oral tissues to infection and cause hyper-sensitivity of the oral mucosa (81).

Vitamin C. Of all the vitamins, ascorbic acid is the one most likely to affect the periodontal tissues for it is essential in the normal metabolism of connective tissue fibers (77). In vitamin C deficiency, an enlargement of the marginal gingivae occurs, enveloping and almost completely concealing the teeth. The gingivae are soft, hemorrhage on the slightest provocation, and show a lack of fibroblasts and collagen fibrils. There is a lack of periodontal support because of a disturbance of periodontal collagen and destruction of periodontal fibers. A disturbance in alveolar bone formation also occurs (32).

Diet and Periodontal Disease

An adequate, nutritious diet for all ages that helps maintain the entire body's integrity and resistance to infection is necessary in order to diminish the destructive processes of periodontal disease (76). Attention must be given to detergent and fibrous foods at meals and between meals. In addition, the consumption of carbohydrates between meals should be discouraged. Above all, proper oral hygiene is necessary for the removal of impacted foods and the accumulation of debris, calculus, and microorganisms (8). Nutritionists must be concerned about proper health of the periodontium for patients with periodontal disease often make a poor selection of foods. They find that many coarse but nutritious foods cause pain when chewed; and therefore, deliberately omit such foods from their diet. Hence the entire body's nutritional health is affected (2).

SUMMARY

Improper tooth and periodontal development, dental caries, and periodontal diseases affect a large segment of the United States and world populations. Both food and nutrition assume a role in these prevalent diseases of mankind. The purpose of this paper was to review and summarize studies reported in the literature regarding the effects of foods and nutrition on dental health. The diets and dietary habits of various age groups and how they might be changed for improved dental health were also discussed.

Pertinent literature reported that nutrition is important for proper development of the tooth and dental tissues especially during the period of time when teeth are undergoing matrix formation and calcification. The most influential nutrients reported were proteins, calcium, phosphorus, and vitamins A, D, and C. Special attention must be given to the maternal, prenatal, infant, preschool, and pre-teen diets which were frequently reported to be deficient in one or more of these nutrients.

Dental caries is one of the most common diseases affecting the human race. Microorganisms and the local presence of fermentable carbohydrates (their physical form, composition, frequency of usage, level in the diet, and type) were reported from both animal experimentations and human clinical investigations to affect the dental caries process. Literature indicated that proper tooth development and composition, the use of fluoride and foods high in phosphates, the chemical composition and rate of flow of saliva, and fats and proteins help to inhibit or retard dental caries. Consistent findings revealed that foods differ in their cariogenicity; fibrous, detergent, non-retentive, liquid, and less refined form of foods are less cariogenic than solid or semiliquid sugars, sticky, retentive, and soft foods. Emphasis must be placed on the reduction of the intake of sucrose and the increased use of the less cariogenic foods at meals and between meals in the diets of all ages, particularly children and adolescents.

Periodontal diseases occur in all age groups. Such local factors as malocclusion, impacted food, other debris, and calculus accumulated about the teeth, the physical nature of the diet, carbohydrates, and the oral microflora were reported by the literature to be the primary causes of periodontal diseases. Although studies indicated that the systemic factor is not the main cause of periodontal disease, deficiencies of protein, calcium, phosphorus, iron, and vitamins D, A, C, and the B-complex were reported to adversely affect the periodontal structures predisposing the periodontium to inflammation, causing sufficient cellular damage to contribute to ease of infection of the tissues by the local factors. An adequate, nutritious diet that helps maintain the entire body's integrity and resistance to infection, and detergent fibrous food which exercise the periodontal tissues are necessary for the prevention of periodontal diseases.

The science of nutrition is truly an indispensable discipline in oral biology and a practical parameter in preventive dentistry.

LITERATURE CITED

1. Roth, H. 1965 Dietotherapy in family dental practice. N.Y.J.D. 35:321.

2. Anonymous 1964 Nutrition and dental health. Dairy Council Dig. 35:105.

3. Anonymous 1969 Nutrient survey reveals numerous dental problems. J. Amer. Dent. Ass. 78:723.

4. Ritchie, J. A. S. 1969 Why education in food and nutrition? In: Learning better nutrition. ed. 2. Food and Agriculture Organization of the United Nations. p. 14.

5. National Institute of Dental Research 1970 Research explores nutrition and dental health. National Institutes of Health. Bethesda, Md.

6. Nizel, A. E. 1968 The science of nutrition, a discipline in oral biology and a parameter in preventive dentistry. In: The Science of Nutrition and its Application in Clinical Dentistry. ed. 2. W. B. Saunders Company. p. 4.

7. Fisher, W. T. 1955 Prosthectics and geriatric nutrition. Oreg. State Dent. J. 24:25.

8. Sandell, P. 1967 What teachers should know about dental health. In: Teaching dental health. ed. 2. American Association for Health, Physical Education, and Recreation. Washington, D.C. p. 5.

9. Brauer, J. C., L. B. Higley, R. L. Lindahl, M. Massler, and I. Schour 1964 Development of the teeth. In: Dentistry for children. McGraw-Hill Company. p. 69.

10. Wilson, E. D., K. H. Fisher, and M. E. Fuqua 1965 Diet and dental health. In: Principles of Nutrition. ed. 2. John Wiley and Sons. p. 394.

11. Finn, S. 1957 Morphology of the primary teeth. In: Clinical Pedodontics. ed. 3. W. B. Saunders Company. p. 56.

12. Massler, M. and I. Schour 1958 Oral structures. In: Atlas of the Mouth. ed. 2. Amer. Dent. Ass. Chicago, Ill. p. 5.

13. Burnett, G. W. and H. W. Scherp 1966 Structures of the oral cavity. In: Oral microbiology and infectious disease. ed. 2. Williams and Wilkins Company. p. 237.

14. Zipkin, I. 1968 The chemical composition of bones, teeth, calculus, saliva, and the periodontium of the human. In: The science of nutrition and its application in clinical dentistry. ed. 2. W. B. Saunders Company. p. 281.

15. Brudevold, F. 1962 Chemical composition of the teeth in relation to caries. In: Chemistry and prevention of dental caries. Charles C. Thomas. p. 32.

16. Hanke, M. T. 1931 The role of diet in the cause, prevention, and cure of dental diseases. J. Nutr. 3:433.

17. Yoon, S. H., F. Brudevold, F. A. Smith, and D. E. Gardner 1965 Fluoride, calcium, phosphate, ash, and water content of human dental pulp. J. Dent. Res. 44:696.

18. Volker, J. F. and R. C. Caldwell 1967 Food and dental caries. In: Clinical pedodontics. ed. 3. W. B. Saunders Company. p. 681.

19. Lossee, F. L. 1965 Enamel caries research: 1962-1964. J. Amer. Dent. Ass. 70:1428.

20. Shaw, J. H. and D. Griffiths 1963 Dental abnormalities in rats attributable to protein deficiency during reproduction. J. Nutr. 80:123.

21. Paynter, K. J. and R. M. Grainger 1956 The relation of nutrition to the morphology and size of rat molar teeth. J. Cand. Dent. Ass. 22:519.

22. Cheraskin, E. 1967 Nutritional factors in disease. In: Clinical pedodontics. ed. 3. W. B. Saunders Company. p. 561.

23. Nizel, A. E. and A. M. Sanford 1968 Proteins in nutrition. In: The science of nutrition and its application in clinical dentistry. ed. 2. W. B. Saunders Company. p. 45.

24. Bawden, J. W. 1963 Calcium metabolism during pregnancy. J. Dent. Child. 30:93.

25. Mitchell, H. S., H. J. Rynbergen, L. Anderson, and M. V. Dibble 1968 Malnutrition--a world problem. In: Cooper's nutrition in health and disease. ed. 15. J. B. Lippincott Company. p. 232.

26. Haldi, J., W. Wynn, K. D. Bentley, and M. L. Law 1959 Dental caries in the albino rat in relation to the chemical composition of the teeth and of the diet. IV. Variations in the Ca/P ratio of the diet induced by changing the calcium content. J. Nutr. 67:645.

27. Harris, R. S. 1968 Macro minerals: calcium, phosphorus, magnesium and calcification. In: The science of nutrition and its application in clinical dentistry. ed. 2. W. B. Saunders Company. p. 67.

28. Gershoff, S. N. 1968 Fat soluble vitamins. In: The science of nutrition and its application in clinical dentistry. ed. 2. W. B. Saunders Company. p. 125.

29. Schour, I., M. M. Hoffman, and M. C. Smith 1941 Change in incisor teeth of albino rats with vitamin A deficiency and effects of replacement therapy. Amer. J. Path. 17:529.

30. Dinnerman, M. 1951 Vitamin A deficiency in unerupted teeth of infants. Oral Surg. 4:1024.

31. Mitchell, H. S., H. J. Rynbergen, L. Anderson, and M. V. Dibble 1968 Nutrition in pregnancy and lactation. In: Cooper's nutrition in health and disease. ed. 15. J. B. Lippincott Company. p. 155.

32. Gould, B. S. 1968 Ascorbic Acid (Vitamin C) and its role in wound healing and collagen formation. In: The science of nutrition and its application in clinical dentistry. ed. 2. W. B. Saunders Company. p. 169.

33. Navia, J. M., L. P. Di Orio, L. Manaker, and S. Miller 1970 Effect of undernutrition during the prenatal period on caries development in the rat. J. Dent. Res. 49:1091.

34. Hampton, M. C., R. L. Huenemann, L. R. Shapiro, and B. W. Mitchell 1967 Caloric and nutrient intake of teenagers. J. Amer. Diet. Ass. 50:385.

35. Everson, G. J. 1960 Bases for concern about teenagers' diets. J. Amer. Diet. Ass. 36:17.

36. Food intake and nutritive value of diets of men, women, and children in the United States, Spring, 1965. A preliminary report 1969 Consumer and Food Economics Res. Div. A.R.S. U.S.D.A.

37. Hinton, M. A., E. S. Eppright, H. Chadderdon, and L. Wolins 1963 Eating behavior and dietary intake of girls 12-14 years old. J. Amer. Diet. Ass. 43:223.

38. Beal, V. A. 1969 Breast- and formula-feeding of infants. J. Amer. Diet. Ass. 55:31.

39. Guthrie, H. A. 1963 Nutritional intake of infants. J. Amer. Diet. Ass. 43:120.

40. Beal, V. A. 1953 Nutritional intake of children. I. Calories, carbohydrates, fat, and protein. J. Nutr. 50:223.

41. Beal, V. A. 1954 Nutritional intake of children. II. Calcium, phosphorus and iron. J. Nutr. 53:499.

42. Beal, V. A. 1955 Nutritional intake of children. III. Thiamine, riboflavin, and niacin. J. Nutr. 57:183.

43. Beal, V. A. 1956 Nutritional intake of children. IV. Vitamins A and D and ascorbic acid. J. Nutr. 60:335.

44. Fryer, B. A., G. H. Lamkin, V. M. Vivian, E. S. Eppright, and H. M. Fox Adequacy of diets of preschool children in the North Central region: calories, protein, carbohydrates, and fat. (Submitted to J. Amer. Diet. Ass.)

45. Fox, H. M., B. A. Fryer, G. H. Lamkin, V. M. Vivian, and E. S. Eppright Adequacy of diets of preschool children in the North Central region: calcium, phosphorus, and iron. (Submitted to J. Amer. Diet. Ass.)

46. Eppright, E. S., H. M. Fox, B. A. Fryer, G. H. Lamkin, and V. M. Vivian 1969 Eating behavior of preschool children. J. Nutr. 1:16.

47. Dierks, E. C. and L. M. Morse 1965 Food habits and nutrient intakes of preschool children. J. Amer. Diet. Ass. 47:292.

48. Egan, M. C. 1969 Combating malnutrition through maternal and child health programs. Children. 16:68.

49. Volker, J. F. and R. C. Caldwell 1967 The epidemiology of dental caries. In: Clinical pedodontics. ed. 3. W. B. Saunders Company. p. 610.

50. Nizel, A. E. and J. S. Shulman 1968 Interaction of dietetics and nutrition with dentistry. J. Amer. Diet. Ass. 54:470.

51. Volker, J. F. and R. C. Caldwell 1967 The etiology of dental caries. In: Clinical pedodontics. ed. 3. W. B. Saunders Company. p. 634.

52. Miller, S. A. 1968 Carbohydrates in nutrition. In: The science of nutrition and its application in clinical dentistry. ed. 2. W. B. Saunders Company. p. 25.

53. Shaw, J. H. 1954 Effect of carbohydrate-free and carbohydrate-low diets on incidence of dental caries in white rats. J. Nutr. 53:151.

54. Haldi, J., W. Wynn, and J. H. Shaw 1953 The relative cariogenicity of sucrose when ingested in the solid form and in solution. J. Nutr. 49:295.

55. Kite, O. W., J. H. Shaw, and R. F. Sognnaes 1950 Prevention of experimental tooth decay by tube feeding. J. Nutr. 42:89.

56. Volker, J. F. 1951 Some observations concerning dental caries production in the Syrian hamster. J. Dent. Res. 30:484.

57. Steinman, R. R. and M. J. Haley 1957 The biological effect of various carbohydrates ingested during the calcification of the teeth. J. Dent. Child. 24:211.

58. Schweigert, B. S., J. A. Shaw, P. H. Phillips, and C. A. Elvehyem 1945 Dental caries in the cotton rat. 3. Effect of different dietary carbohydrates on the incidence and extent of dental caries. J. Nutr. 29:405.

59. Mitchell, D. F., D. S. Chernausek, and E. Z. Helman 1951 Hamster caries. The effects of three different dietary sugar levels and an evaluation of scoring procedures. J. Dent. Res. 30:778.

60. Keyes, P. H. and R. C. Likens 1946 Plaque formation, periodontal disease and dental caries in Syrian hamsters. J. Dent. Res. 25:116.

61. Volker, J. F. and R. C. Cladwell 1967 Food and dental caries. In: Clinical pedodontics. ed. 3. W. B. Saunders Company. p. 681.

62. Anonymous 1970 Accepted dental therapeutics. Council on Dental Therapeutics. p. 188.

63. Blayney, J. R. and I. N. Hill 1967 Fluorine and dental caries. J. Amer. Dent. Assn. 74:233.

64. Zipkin, I. 1968 The metabolism and safety of fluorides. In: The science of nutrition and its application in clinical dentistry. ed. 2. W. B. Saunders Company. p. 11.

65. Harris, R. S. and A. E. Nizel 1964 Metabolic significance of the ratio of Ca to P in foods and diets. J. Dent. Res. 43:1090.

66. Nizel, A. E. and R. S. Harris 1964 The effect of phosphates on experimental dental caries: A literature review. J. Dent. Res. 43:1123.

67. Miller, S. A. 1968 Fats in nutrition. In: The science of nutrition and its application in clinical dentistry. ed. 2. W. B. Saunders Company. p. 39.

68. Bavetta, L. A. and F. J. McClure 1957 Protein factors in experimental rat caries. J. Nutr. 63:107.

69. Dodds, M. L. 1964 Protein and lysine factors in the cariogenicity of a cereal diet. J. Nutr. 82:217.

70. Bibby, B. G. 1968 The cariogenicity of different foodstuffs. In: The science of nutrition and its application in clinical dentistry. ed. 2. W. B. Saunders Company. p. 318.

71. Andlaw, R. J. 1960 The relationship between acid production and enamel decalcification in salivary fermentations and carbohydrate foodstuffs. J. Dent. Res. 39:1200.

72. Weirs, R. L. and A. H. Trithart 1960 Between-meal eating habits and dental caries experience in preschool children. Amer. J. Public Health. 50:1097.

73. Ohlson, M. A. and B. P. Hart 1965 Influence of breakfast on total day's food intake. J. Amer. Diet. Ass. 47:282.

74. Koehne, M., R. W. Bunting, and F. P. Hadley 1934 Review of recent studies of cause of dental caries. J. Amer. Diet. Ass. 9:445.

75. Burnett, G. W. and H. W. Scherp 1966 Periodontal disease. In: Oral microbiology and infectious disease. ed. 2. Williams and Wilkins Company. p. 423.

76. Parfitt, G. J. 1967 Periodontal diseases in children. In: Clinical pedodontics. ed. 3. W. B. Saunders Company. p. 507.

77. Richard, J. F. 1966 Physical character of the diet. In: Advanced periodontal disease: Surgical and prosthetic management. W. B. Saunders Company. p. 401.

78. Auskaps, A. M., O. P. Gupta, and J. H. Shaw 1957 Periodontal disease in the rice rat. 3. Survey of dietary influences. J. Nutr. 63:325.

79. Harris, R. S. 1968 Iron and Micro Elements. In: The science of nutrition and its application in clinical dentistry. ed. 2. W. B. Saunders Company. p. 87.

80. Russell, A. L. 1963 International nutrition surveys: A summary of preliminary dental findings. J. Dent. Res. 42:223.

81. Dreizen, S. 1968 The B-vitamins. In: The science of nutrition and its application in clinical dentistry. ed. 2. W. B. Saunders Company. p. 146.

THE ROLE OF FOODS AND NUTRITION IN DENTAL HEALTH

by

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Dental diseases and problems are chronic and prevalent among populations of the world. Good dental health may be achieved through the use of proper foods and nutrition by all age groups. The purpose of this paper was to review and summarize studies reported in the literature regarding the effects of foods and nutrition on dental health. The diets and dietary habits of various age groups and improvement of these diets for better dental health were also discussed. An overview of the structure, function, development, and composition of the tooth and periodontium was presented.

Pertinent literature reported that nutrition is important for proper development of the tooth and dental tissues. The most influential nutrients reported were proteins, calcium, phosphorus, and vitamins A, D, and C. Studies revealed that maternal, prenatal, infant, preschool, and preteen diets are frequently deficient in one or more of these nutrients.

Microorganisms and the local presence of fermentable carbohydrates (their type, physical form, composition, level in the diet, and frequency of usage) were reported to affect the dental caries process. Literature indicated that proper tooth development and composition, the use of fluoride and foods high in phosphates, the saliva, and fats and proteins help to inhibit dental caries. Consistent findings revealed that foods differ in their cariogenicity; fibrous, detergent, liquid, non-retentive, and less refined forms of food are less cariogenic than solid or semiliquid sugars, sticky, retentive, and soft foods. The less cariogenic foods should be emphasized at meals and between meals for all age groups.

Local factors were reported by the literature to be the primary cause of periodontal diseases. Although studies indicated that the systemic factor is not the main cause of periodontal disease, deficiencies of protein, calcium, phosphorus, iron, and vitamins A, C, D, and the B-complex were reported to adversely affect the periodontal structures contributing to ease of infection by the local factors. A nutritious diet that helps maintain the body's integrity, resistance to infection, and exercises the periodontal tissues is necessary for prevention of periodontal disease.