atoms of phosphorus for every three atoms of calcium. All plant and animal foods are rich in phosphorus, and except for the prematurely born infant who is fed human milk, nutritional deficiencies of phosphorus are rare. Human breast milk contains enough phosphorus to nourish a full-term infant but not, apparently, to support the growth requirements of preterm infants.

Phosphate is important in mineralization both in animal models and in clinical disease. In rats, rickets occurs only with a combined deficiency of vitamin D and phosphate relative to calcium, but in people, rickets can be produced by phosphate deficiency even in the presence of a high concentration of 1,25-dihydroxyvitamin D (Rowe et al. 1979). Phosphate's role in mineralization is obvious in persons with phosphaturic osteomalacia or vitamin D-resistant rickets. In these persons, a combination of frequent administration of phosphate plus large doses of vitamin D restores bone growth and mineralization to an extent that cannot be achieved by either agent alone. Even with the use of calcitriol, phosphate supplementation is needed to mineralize bone in this condition (Chan, Alon, and Hirschman 1985).

Some forms of phosphate, such as plant phytates, are poorly absorbed and can also bind calcium, increasing calcium excretion in the feces. Increased phytate in the diet may contribute to osteomalacia but does not necessarily influence bone mass. When large amounts of aluminum hydroxide antacid gels are ingested, phosphate depletion can also occur because of the formation of aluminum phosphate, which is insoluble and unabsorbable. The clinical significance of these interactions has not been established.

Many studies indicate that phosphate regulates bone formation and resorption (Raisz and Kream 1983). *In vitro* studies have shown that increasing phosphate concentration beyond the physiologic range can inhibit bone resorption (Lorenzo, Holtrop, and Raisz 1984). Although osteoclast activity may decrease, morphologic studies suggest that the osteoclasts are less effective rather than inactivated.

Increasing the phosphate concentration in cultures of bone-forming cells increases both collagen synthesis and deposition of mineral. *In vitro* mineralization may require the addition of an organic phosphate compound such as beta-glycerol phosphate. Organic phosphate may provide a constant delivery of phosphate that does not precipitate, or it may provide more effective organic forms because they are hydrolyzed locally in the matrix.

The enzyme alkaline phosphatase can cleave organic phosphate compounds in bone, thus raising the local concentration of inorganic phosphate and promoting mineralization. Although the precise role of alkaline phosphatase is uncertain, mineralization is impaired when this enzyme is deficient, as in the disease familial hypophosphatasia.

Some *in vivo* studies also indicate that phosphate promotes bone growth. The rate of bone formation across animal species is generally proportional to their serum phosphate concentration. For example, rats with high serum phosphate levels also have high rates of skeletal growth. Within species, serum phosphate levels are also higher at times of rapid bone formation, as in early infancy and puberty in humans. Hormones may be more likely than nutrition to control these changes in serum phosphate concentration. An inadequate intake of phosphorus could, in theory, impair skeletal growth as well as soft tissue growth, but phosphates are so abundant in foods that a seriously deficient intake would probably indicate decreased intake of other nutrients.

While phosphate deficiency can lead to decreased bone mass, excessive phosphate intake can also harm the skeleton. The adverse effects of high phosphate intake have been studied extensively in animals (LSRO 1981; Jowsey, Reiss, and Canterbury 1974). Excessive dietary intake of phosphate produced bone disease in animals, particularly if the diet was also low in calcium. The current American diet is quite rich in phosphorus; this imbalance might affect bone health adversely, but that hypothesis has not been proved.

Recent studies suggest that the adverse effects of excessive phosphate are largely mediated through secondary hormonal responses or through toxic effects of phosphate deposition in soft tissues. Excessive phosphate intake reduces serum calcium concentration, particularly when the calcium intake is low, because some of the phosphate carries calcium with it into soft tissue. The resulting hypocalcemia stimulates PTH secretion, which leads to increased bone resorption and increased phosphate excretion in the urine. Because the effect of PTH on the kidney continues after the phosphates have been absorbed, fasting hypophosphatemia is common in persons consuming large amounts of phosphate, presumably due to secondary hyperparathyroidism (Herbert et al. 1966; Reiss et al. 1970; Sherwood et al. 1968). High phosphate intakes can also decrease calcitriol production in the kidneys, impairing calcium absorption and producing further secondary hyperparathyroidism (Portale, Halloran, and Morris 1987).

The aforementioned sequence of events occurs in experiments and may also occur in persons with renal failure who cannot excrete phosphate efficiently; whether nutritional phosphate excesses in American diets produce similar changes in the healthy population is in question. Bone mass may be lower in people who consume diets that contain a relatively high ratio of phosphorus to calcium as compared with vegetarians, for whom this ratio is substantially lower (Marsh et al. 1980). The difference is small, however, and many other uncontrolled variables may be involved. Some young adults have elevated PTH activity with a high phosphorus intake (Bell et al. 1977). More recently, direct measurement of increased PTH level and action with high phosphorus intake was reported in young adults consuming ordinary foods (Calvo, Kumar, and Heath 1988).

Role of Calories and Protein in Skeletal Disease

An adequate intake of calories and protein is essential for the growth and maintenance of a healthy skeleton. Calories support the synthesis of bone tissue and also "spare" the body from using for energy the protein that is needed to form bone matrix. In children, insufficient calorie or protein consumption inhibits skeletal growth. Adults who consume insufficient calories or protein may also lose bone mass and become susceptible to bone fractures.

Calories

Changes in calorie consumption may affect hormone production. In children, the decreased skeletal growth associated with malnutrition is probably mediated, at least in part, by decreased production of somatomedin or insulin-like growth factors. In adolescents and adults, calories may also affect the production of sex hormones (Cuttler et al. 1985), as demonstrated in adolescent women (whose growth spurt is associated with menarche). The onset of menses may depend on a certain body weight or level of fat stores, that, in turn, depends on adequate nutrition, including calories. Healthy women who lose large amounts of weight may stop menstruating, although factors other than calories may also be involved. In persons with anorexia nervosa, menstrual disorders may occur earlier than can be accounted for simply by weight loss (Drossman, Ontjes, and Heizer 1985).

The combination of decreased intake of calories, calcium, and other nutrients and decreased production of estrogen may be responsible for reduced density of bone mass in persons with anorexia nervosa (Rigotti et al. 1984). Female athletes, particularly runners and ballet dancers, who have

very low body fat content, a low intake of calories and calcium, and amenorrhea also have decreased bone mass (Drinkwater et al. 1984; Marcus et al. 1985).

Low body fat, which presumably reflects a low calorie intake, may also cause reduced bone mass in postmenopausal women (Dequeker, Goris, and Uyterhoeven et al. 1983). One hypothesis suggests that after menopause, body fat provides a continued source of estrogen that retards bone loss in obese women. Another possibility is that women who weigh more put more stress on the skeleton and stimulate bone mass to increase, perhaps through an effect on the vitamin D-endocrine system (Bell, Epstein, et al. 1985). Cigarette smoking seems to affect bone mass adversely, but this finding may be related to the lower caloric intake and decreased estrogen production observed in women who smoke (Rundgren and Mellstrom 1984).

Protein

Adequate dietary protein is essential for bone growth; the skeleton is second only to muscle in terms of total body protein content. Abnormal protein nutrition may cause osteoporosis, and a decreased protein intake could contribute to the decline in bone mass observed in alcoholics, although nutritional factors other than the direct effects of alcohol may also be involved.

High protein intake may also cause bone loss. In young individuals, increasing dietary protein intake increases calcium excretion in urine and produces a negative calcium balance (Heaney et al. 1982; Hegsted 1986). Under ordinary circumstances, increased phosphate intake accompanies increased protein intake; high-protein foods are often high in phosphate as well. If dairy products are the source of protein, calcium intake may also rise. Thus, whether moderately high-protein diets have an adverse effect on bone mineralization has not yet been established (Heaney and Recker 1982).

Why calcium loss results from high protein intakes is also uncertain. The acid content of the diet may be a major factor because much of the calcium loss from a high-protein diet can be reproduced by administering the sulfur-containing amino acids that were in that diet (Tschope and Ritz 1985). Increased acidity induces calcium loss by increasing renal excretion directly as well as by increasing the dissolution of mineral from the skeleton and impairing mineral deposition.

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Role of Alcohol in Skeletal Disease

Excessive alcohol intake is a risk factor for osteoporosis (see Table 7-1), but the basis for this relationship has not been established (Consensus Development Panel 1984). In women, the incidence of hip fractures has been observed to increase with increasing alcohol consumption (Paganini-Hill et al. 1981). The association between alcohol and bone loss was first described more than 20 years ago in a population of young male alcoholics (Saville 1965). More recent radiographic evidence has confirmed extensive bone loss among alcoholic patients ranging in age from 24 to 62 (Spencer et al. 1986). Other studies have reported significant bone loss, decreased bone density, increased bone resorption, and increased fracture incidence among alcoholics (Nordin 1984).

Why alcohol induces bone loss is uncertain. The main hypotheses include poor nutrition, alcohol-induced calcium diuresis, secondary effects of liver disease, and induction of excessive PTH secretion. Malabsorption of calcium has been observed in some studies (Nordin 1984) but not in others (Spencer et al. 1986). The current lack of information on mechanism of action, the effects of excessive alcohol intake on bone loss in women of different ages, and the effects of moderate alcohol intake on bone metabolism suggest a need for further research.

Role of Other Minerals in Skeletal Disease

The skeleton is a storehouse for relatively large amounts of sodium, magnesium, copper, silicon, and other minerals, whose intake may affect mineralization of the skeleton.

Fluoride

The effects of fluoride on bones and teeth have been observed and studied for nearly 100 years. Fluoride is incorporated into tooth enamel, and its beneficial effect in reducing the incidence of caries is well established (see chapter on dental diseases).

Fluoride could be a possible treatment for osteoporosis because it is rapidly and extensively accumulated into bone mineral. It stimulates osteoblast activity and new bone growth, particularly that of trabecular bone, the type most susceptible to fracture. Animal studies have not produced consistent data about the effect of fluoride on experimentally induced osteoporosis (Bikle 1983). Human epidemiologic studies show that subjects living in areas where water is fluoridated have greater bone densities than those

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living in low-fluoride areas (Bernstein et al. 1966; Simonen and Laitinen 1985); however, other epidemiologic studies suggest that small amounts of fluoride have no consistent effect on fracture incidence (Kanis and Meunier 1984). Nevertheless, human clinical studies have demonstrated an improved calcium balance with short-term administration of large doses of fluoride, increased bone mass when fluoride was administered for at least a year, and an increased volume of trabecular bone and a decreased incidence of fractures in groups treated with fluoride for at least 2 years (Bikle 1983).

Consequently, fluoride in relatively high doses of 50 to 100 mg of sodium fluoride daily has been used to treat osteoporosis. These doses increase bone mass in a substantial proportion of persons and may decrease fractures (Riggs, Seeman, et al. 1982). Such high intakes of fluoride, however, produce skeletal abnormalities such as osteophytes (bony overgrowths) and stimulate bone matrix formation at inappropriate sites. Fluoride therapy without supplemental calcium and vitamin D may produce osteomalacia, and gastrointestinal and rheumatic side effects have also been reported (Bikle 1983). Controlled prospective trials of fluoride in the treatment of osteoporosis are currently under way in the United States.

Aluminum

The effect of consuming large amounts of aluminum hydroxide in antacids was mentioned earlier in the phosphates section. Aluminum hydroxide's role in impairing bone growth and mineralization is complex because it affects phosphate absorption, osteoblast function, and mineral deposition itself. Whether aluminum causes impaired mineralization in renal osteodystrophy or whether it accumulates as a result of this condition is uncertain (Quarles et al. 1985).

Magnesium

Bone recycling may be impaired by the direct effects of magnesium on bone cells (Johannesson and Raisz 1983) as well as by the decreased production of PTH and hypocalcemia that develop in severe magnesium deficiency (Rude et al. 1978). The high levels of circulating magnesium observed in renal failure are associated with impaired mineralization *in vitro* and can slow the formation of hydroxyapatite. Changes in dietary magnesium, however, do not significantly affect the regulation of bone metabolism.

Sodium

High sodium intakes can cause increased loss of calcium in the urine and could affect age-related bone loss (Breslau et al. 1982; Goulding 1983). The

issue of sodium in the diet is discussed in the chapter on high blood pressure.

Trace Elements

Other ions may affect mineral metabolism. Zinc and silicon, which are deposited in bone, are important factors in bone mineralization (Calhoun, Smith, and Becker 1974; Carlisle 1981). Circadian variations in serum zinc concentration parallel serum calcium changes (Markowitz, Rosen, and Mizruchi 1985). Copper has been shown to inhibit bone resorption (Wilson, Katz, and Gray 1981). Low boron intake may also be a risk factor for osteoporosis (Nielsen et al. 1987). The clinical importance of these substances in bone metabolism is unknown.

Role of Other Vitamins in Skeletal Disease

Vitamin A deficiency can alter bone remodeling in animals but is not a major cause of bone disease in humans. Excessive levels of vitamin A, obtained from animal foods or supplements (but not as beta-carotene from plants), can produce hypercalcemia and skeletal abnormalities, with thinning of the cortex and fractures in some areas and overproduction of bone in other areas.

Vitamin C deficiency is associated with osteoporosis but is not a major factor in the disease in the United States (Lynch et al. 1967). There is no evidence that megadoses of vitamin C adversely affect the skeleton.

Vitamin K-dependent proteins, particularly osteocalcin, have recently been identified in bone. At present, their metabolic roles are not clearly defined. Their current importance as clinical indicators for bone turnover is difficult to assess (Delmas et al. 1983; Slovik et al. 1984; Cole and Gundberg 1985; Price, Parthemore, and Deftos 1980). Depressed circulating levels of vitamin K have been reported recently in patients with osteoporosis (Hart et al. 1985), and vitamin K deficiency produces hypercalcemia in rats (Robert et al. 1985). Warfarin, an anti-coagulant that blocks the action of vitamin K, can reduce bone mineral in vitamin D-treated rats (Price and Sloper 1983). Further investigations are needed to explain this interaction between vitamin K and bone mineralization.

Role of Exercise in Skeletal Disease

Although not strictly a nutritional issue, the role of exercise in skeletal disease is important. That physical activity affects maintenance of skeletal

mass was first noted in spinal injury patients (Freedman 1949) and has since been confirmed in patients who require bed rest, in normal volunteers who stay in bed for long periods of time, and in astronauts who work in gravity-free environments (Anonymous 1983).

Whether exercise prevents osteoporosis has not yet been established. Although the evidence from cross-sectional and prospective studies suggests that physical exercise may increase the peak bone mass achieved at maturity and may also reduce losses of bone mass after maturity, many of these studies have methodological flaws (Block et al. 1987). Nevertheless, until better information becomes available, 3 to 4 hours of weight-bearing exercise per week is potentially beneficial to the skeleton and could represent a safe, low-cost method for maintaining bone mass.

Implications for Public Health Policy

Dietary Guidance

General Public

The prevalence, health consequences, and expense of osteoporosis among Americans make it a compelling public health priority. Dietary factors of particular concern are calcium, phosphate, vitamin D (and its hormonally active form calcitriol), protein, sodium, calories, and alcohol. How these factors affect peak bone mass development is important and requires further investigation. Other lifestyle factors that may decrease the risk for osteoporosis include increased exercise and decreased cigarette smoking. In postmenopausal women, estrogen-replacement therapy has been the best documented method of preventing osteoporosis.

The dietary factors associated with bone mass, the universality of bone loss with age, the interaction of diet and lifestyle with genetic factors, and the difficulties in measuring bone loss in populations make defining the relationship between diet and osteoporosis difficult. However, evidence suggests that, particularly during the first three to four decades of life, ingesting adequate calcium, maintaining appropriate body weight, exercising, restricting alcohol, and avoiding cigarette smoking are appropriate public health strategies for prevention of osteoporosis.

Most interest in the dietary control of osteoporosis focuses on calcium. Although current epidemiologic and clinical evidence is uncertain, chronic low calcium intake may decrease peak bone mass, especially during adolescence. Surveys indicate that dietary calcium intake of adolescent girls is

one-third or more below the 1,200 mg/day recommended for this population and that adult women of reproductive ages also consume less than the recommended 800 mg/day. Although the ideal level of calcium intake for development of peak bone mass is unknown, and although it has not yet been established whether increased calcium intake will prevent osteoporosis, females, particularly adolescents and young adults, in the United States should increase food sources of calcium. The public should also be educated about the calcium content of various foods, particularly low-fat dairy products, and should maintain adequate calcium intake at all ages.

Additional study of the epidemiologic association between diets high in protein and increased prevalence of osteoporosis is required to make further conclusions.

Special Populations

Children, pregnant and lactating women, and older people have special needs for calcium based on, respectively, the extra skeletal demands of growth, milk production, or the age-related decrease in absorption of calcium. Older Americans consume amounts of calcium that average as much as 40 percent below current recommendations of 800 mg/day. Postmenopausal women should receive counseling on supplemental use of estrogen, and all groups should receive information about calcium-rich foods. People who take calcium supplements also need education on appropriate use, side effects, the forms in which they are best absorbed, and interactions with other medications.

Nutrition Programs and Services

Food Labels

Present evidence on the role of dietary factors in skeletal disease has no special implications for change in policy related to food labeling. However, nutrition labeling, which lists calcium and other nutrient content, should be encouraged on most food products.

Food Services

Aside from the special populations noted below, evidence related to the role of dietary factors in skeletal diseases currently holds no special implications for change in policy related to food service programs.

Food Products

Foods abundant in calcium are widely available in the United States. However, the diversity of U.S. dietary patterns suggests the possibility of calcium fortification of a limited number of foods. These additions should be carefully selected to avoid excessive calcium in the food supply. Fortification should be chosen based on the frequency of consumption of a food by the targeted populations, and the calcium should be in a physiologically available form. It is important to continue fortification of suitable foods with vitamin D because this has been instrumental in reducing the prevalence of rickets and osteomalacia in the United States.

Special Populations

Food services offered to children, adolescents, and young adults should provide diets with sufficient calcium to enhance achievement of peak bone mass. Persons who are unable to convert vitamin D to its active form may require supplementation with calcitriol. Those with chronic malabsorption syndromes may require supplementation with calcium or calcitriol.

Whether calcium, vitamin D, or calcitriol should be provided to older women to prevent or delay postmenopausal bone loss is as yet uncertain. Although evidence for the precise role of physical activity in prevention of osteoporosis is still emerging, it seems reasonable to include exercise as a component of any program to enhance the skeletal integrity of older Americans. Older persons should be encouraged to maintain regular activities such as walking and other weight-bearing exercise.

Research and Surveillance

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

- Changes in calcium and phosphate requirements throughout life.
- The effects of altering proportions of phosphate and protein on calcium requirements and bone mineralization.
- The effects of increased calcium intake on peak bone mass and on prevention of postmenopausal bone loss.
- Potential toxicities of high-dose supplements of calcium.
- The development of calcium sources with improved bioavailability.
- Safe and adequate levels of vitamin D added to the food supply.

- The relationship of vitamin D and its metabolites to calcium in the development of peak bone mass and prevention of bone loss.
- The levels of vitamin D and its metabolites, fluoride, and calcium that are safe and adequate for the treatment of osteoporosis.
- The effects of moderate and excessive alcohol intake on bone mineral metabolism.
- The effects of various levels of physical activity on loss of bone mass.
- The relationship of other vitamins and minerals to peak bone mass and to prevention of bone loss.

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Chapter 8

Dental Diseases

Sweet things are bad for the teeth.
Jonathan Swift
Polite Conversation, Dialogue II (1738)

Introduction

Historical Perspective

Descriptions of the oral manifestations of scurvy and various single and multiple B-complex deficiencies in humans were among the earliest contributions to nutrition knowledge (Jolliffe 1962). Recognition of the oral signs of nutrient deficiency was facilitated by the pain associated with pathology in the mouth and the easy access to the mouth for examination.

Early research on oral pathology was controversial because of diverse results from laboratory research and the lack of basic knowledge about the complexity of nutrition and the interrelationships among nutrients. Experimental diets were crude by today's standards and often resulted in multiple nutrient deficiencies. Moreover, pure nutrients were not available for supplementation of deficient diets, and dietary supplements rarely contained the full complement of essential nutrients. Early experimental design also differed significantly from naturally occurring conditions that might predispose to nutritional disorders.

Controversy especially centered on dental caries (tooth decay) observed in rats in early studies. Two schools of thought developed, with one emphasizing that decay was a result of environmental influences on the tooth surface and the other maintaining that lesions were evidence of systemic nutritional deficiencies. Neither perspective considered the role of microorganisms in decay processes until much later (Shaw 1952).

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Role of Sugars

The relationship between sweet foods and tooth decay was observed by the ancient Greeks. Aristotle, for example, associated rotted teeth with consumption of soft figs. In the 15th century, Arculanus recommended avoidance of sweet and sticky foods to prevent tooth loss. The experimental demonstration of tooth demineralization in the presence of carbohydrates (but not meat) mixed with saliva occurred in 1890 (Newbrun 1982). These studies foreshadowed the many animal and human studies of sugar and caries incidence that have led to our current understanding of the etiology of this condition.

Role of Fluoride

One of the first descriptions of mottled enamel appeared near the turn of this century when the tooth enamel of immigrants to America from Naples was observed to be speckled with white areas or to be mottled, pitted, or discolored (Eager 1901). This condition, called *denti di Chiaie*, was thought to result from local geologic conditions, and a change in the source of water was suggested as the reason for the lower incidence of this abnormality among the children of these immigrants.

A similar phenomenon was observed in children living in Colorado Springs (Black and McKay 1916; McKay and Black 1916) and other locations by investigators who noted that the mottled teeth of these children, although apparently inadequately mineralized, were more resistant to dental caries than normally calcified teeth (McKay 1929). Studies in the 1930's demonstrated the relationship of water-borne fluoride to the prevention of tooth decay (Smith, Lantz, and Smith 1931; Dean 1938) and led to early suggestions that dental caries might be controlled by increasing the fluoride concentration of drinking water. More conclusive evidence of the inverse relationship between fluoride in the water supply and dental caries was provided by early surveys (Dean, Arnold, and Elvove 1942) that were later confirmed (Russell and Evolve 1951; Englander and Wallace 1962; Adler 1970).

Results of the epidemiologic surveys of the 1930's established the fluoride concentrations in natural water that provided near maximal caries protection without producing objectionable mottling of teeth (fluorosis). The first clinical trial to add fluoride to communal water supplies began in Grand Rapids, Michigan, in 1945, where fluoride concentration was adjusted to 1 part per million (ppm). After 6.5 years of fluoridation, caries prevalence in 4- to 6-year-old children in Grand Rapids was about 50 percent of that in the nonfluoridated (less than 0.2 ppm) neighboring community of Muskegon.

After 15 years of fluoridation, children 12 to 14 years of age, born and raised in Grand Rapids, had about 55 percent lower caries scores than children of the same age prior to fluoridation (Arnold et al. 1962). Many other studies demonstrated that controlled fluoride supplementation of community water supplies could reduce dental caries without causing undesirable side effects (Galagan and Vermillion 1957; Dean et al. 1950; Ast and Chase 1953; Knutson 1970; Hutton, Linscott, and Williams 1956; Connor 1970).

Significance for Public Health

Dental caries and periodontal disease are important and widespread public health problems in the United States. They are rarely life threatening but can cause substantial expense, pain, restriction of activity, and work loss (Corbin, Kleinman, and Lane 1985). Although dental caries among children, as well as some forms of adult periodontal disease, appear to be declining, the overall prevalence of these conditions imposes a substantial burden on Americans. Of the 13 leading health problems in the United States, dental disorders rank second in direct costs (Carter Center Health Policy Task Force 1984). Dental care cost $21.3 billion in 1985 (U.S. Department of Commerce 1986).

Dental Caries

Dental caries, or tooth decay, is caused by a progressive dissolution of mineral from tooth surfaces by acid produced by oral bacteria. Advanced disease can result in tooth loss. The National Health and Nutrition Examination Survey (NHANES) of 1971–74 reported that Americans ages 1 to 74 years had an average of 1.3 decayed, 5.3 missing, and 6.4 filled teeth. Because people normally have a total of 28 to 32 permanent teeth (depending on the presence of the four wisdom teeth), nearly half of the teeth in the average American mouth were filled, missing, or decayed at that time (NCHS 1979). A more recent survey (NIDR 1987) summarized the prevalence of coronal caries by the mean number of decayed and filled permanent surfaces (DFS). As shown in Figure 8-1, the mean DFS (which does not include missing teeth) for age 18–19 is 12, and for employed adults over age 40, the mean DFS is 29. The average for all ages was 23 DFS out of 128 possible surfaces. Comparing mean decayed and filled teeth (DFT) in the recent National Institute of Dental Research survey (NIDR 1987) with the National Center for Health Statistics (NCHS) household survey (NHANES I) conducted in 1971–74, the employed persons in the recent NIDR survey had a lower mean DFT through age 34. Beyond that age, tooth loss prevents any meaningful comparisons with earlier data.
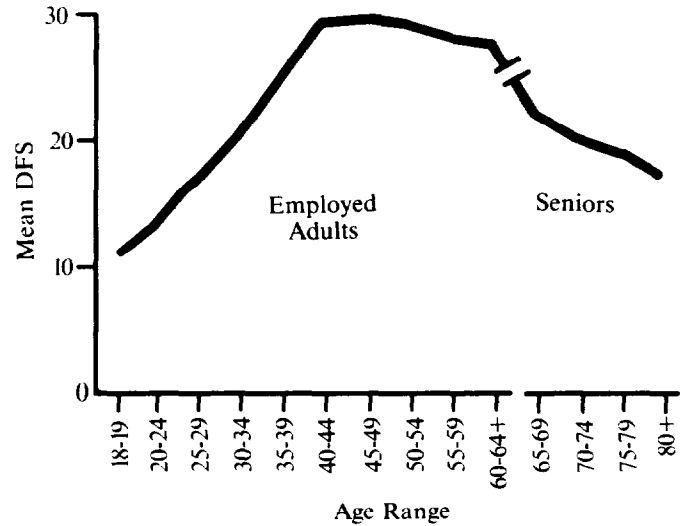
Figure 8-1. The distribution of mean decayed and filled coronal surfaces (DFS) by age as determined from the NIDR survey of employed adults and seniors.

Source: National Institute of Dental Research 1987.

The mean number of root surface lesions by age is shown in Figure 8-2. Decay of tooth roots (root caries) was three times higher in persons 65 years and older than in employed adults, 63 percent as opposed to 21 percent, and only half of such lesions had been filled. The higher prevalence in older persons results from increased exposure of root surfaces due to age-related gum recession or periodontal disease.

The number of decayed, missing, and filled permanent teeth increases steadily with age. In 1980, the average child had at least 1 carious lesion in a permanent tooth by age 8, 4 by age 12, and 11 by age 17 (NIH 1981). In the 1971–74 NHANES, the number increased from an average of 1.7 in children 6 to 11 years to 22.2 in adults 65 to 74 years (NCHS 1979). The number does not differ by sex, but blacks overall tend to have fewer decayed, missing, and filled teeth during adulthood.

Within the past two decades, the incidence of dental caries in children has been declining, perhaps by as much as 30 to 50 percent. This decline has been attributed largely to increased intake of fluoride from drinking water, food, toothpastes, mouth rinses, and topical application, although decreased intake of cariogenic foods and improved dental hygiene and care have also been suggested as contributory factors (Leveille and Coccodrilli 1982; Navia 1985; Corbin et al. 1987).
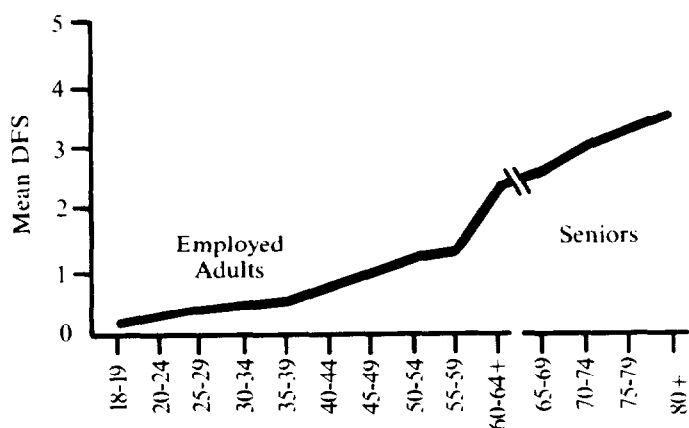
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Figure 8-2. **The distribution of mean decayed and filled root surfaces (DFS) by age as determined from the NIDR survey of employed adults and seniors.**

Source: National Institute of Dental Research 1987.

Both the benefits and the safety of water fluoridation have been carefully examined and endorsed by many professional and scientific organizations and by U.S. Surgeons General since the mid-1940's. More than 120 million people in 8,000 U.S. communities currently are provided with optimally adjusted fluoridated water. An additional 10.7 million people in 3,000 communities drink water with naturally occurring fluoride (Center for Prevention Services 1985; Ismail et al. 1987). Nevertheless, despite the proven benefits of fluoride, only 61 percent of the U.S. population on public water supplies now receive fluoridated water (Löe 1986).

Periodontal Disease

Periodontal disease includes a spectrum of pathologic conditions ranging from minor gum inflammation (gingivitis) to severe loss of the bone structure supporting the teeth (periodontitis). Advanced disease results in loosening of and eventual loss of teeth. In national surveys of the early 1970's, 23 percent of children ages 6 to 17 years had gingivitis (NCHS 1979). Subsequent statewide surveys have reported rates varying from 19 to 44 percent in this group (Corbin, Kleinman, and Lane 1985).

National surveys of adult periodontal disease found little change in prevalence of periodontitis between 1960–62 and 1971–74. The National Adult Dental Health Survey of 1985–86 reported gingival bleeding in 43 percent of employed adults and 47 percent of persons over age 65. Seventy-seven

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percent of all adults, and 95 percent of older persons, had at least one site in the mouth with periodontal attachment loss of 2 mm or more. More severe periodontal destruction, 4 mm or more attachment loss, was observed in 24 percent of adults and 68 percent of older persons. The percent of persons by severity of loss of attachment and age group is shown in Figure 8-3. For both working adults and older persons, the severity of periodontal disease increases with age, and it is more prevalent among males than among females (NIDR 1987). Periodontal disease is a major cause of tooth loss, and efforts to prevent this condition are desirable (Rank et al. 1983).

Loss of Teeth

The two major causes of tooth loss are dental caries and periodontal disease. Toothlessness limits individual selection of foods to those that require little biting or chewing, therefore influencing the nutritional intake of affected individuals.

In the 1971–74 NHANES, about 15 percent of the adult population ages 18 to 74 years had lost all permanent teeth. Another 9 percent had lost all of their upper or their lower permanent teeth, and by the ages of 65 to 74, 46 percent of Americans were edentulous (NCHS 1979). Nevertheless, these figures represent a significant decline from surveys taken in 1960–62 (Ismail et al. 1987).

This favorable trend for decreased tooth loss has continued. The 1985–86 survey of employed adults showed that toothlessness has almost been eliminated in this group; only 4 percent were missing all their teeth, and half had lost none or at the most one tooth. However, as shown in Figure 8-4, toothlessness increases with age and remains a major problem among older Americans. The survey indicated that 42 percent of Americans over age 65 were edentulous and that only 2 percent have retained all 28 permanent teeth. Comparing the prevalence of tooth loss in the recent survey to that reported by the NCHS survey in 1960–62, the current sample had less tooth loss at every age interval.

Scientific Background

Normal Tooth Development

The tooth is a very specialized structure important for the proper initial processing of solid foods. A schematic cross-section of a tooth is presented in Figure 8-5. People normally develop two sets of teeth, 20 deciduous, or primary, teeth and 32 permanent, or secondary, teeth. Each tooth develops from a tooth bud that forms in the area of the jaw. Each tooth bud contains a