CHAPTER 8

Epidemiology: Dental Caries

AT THE BEGINNING of the twentieth century, with Miller's chemico-parasitic theory newly propounded, there seemed little reason to look outside the human mouth for the causes of that almost universal disease, dental caries.[1] The oral environment, according to Miller, held the clue to the origin of this disease, and, if one could prevent the formation of bacterial plaques under which acid fermentation might occur, the whole problem of caries might be solved.

Dental research since that day has provided factor after factor which seemed to influence the occurrence of caries. Regardless of the validity of the Miller theory, the balance between dental health and dental caries was proving to be so delicate that a search for the predisposing factors of the disease became imperative. Detailed histological and chemical studies have shown the caries mechanism to be far more complicated than Miller dreamed it to be. Researchers for many years declared themselves to be in search of *"the*

cause" of caries, but less and less have they really expected that one cause would lie at the end of their trail. The concept of multifactorial disease is now seen to fit dental caries as well as it does any other mass disease with which the epidemiologist has to deal. The public health dentist needs the tools of epidemiology in his fight against caries not only to develop a fuller understanding of the predisposing causes of the disease, but also to aid in planning the public programs which will control it and prevent it. Let us apply the epidemiological methods outlined in the last chapter to the subject of caries, reversing the order, however, and considering secular variations first, then host, agent, and environmental factors.

SECULAR VARIATIONS

The steady increase in proportion of the population affected by dental caries during most of recorded history has been touched upon in the previous chapter. Fig. 12 shows graphically the pace of this increase in Greece, from 3000 B.C. to the present time. The steady increase in percentage of teeth affected by caries, reaching its climax in modern times is matched by an equally striking increase in defective calcification.[2,3] Even at age 6, 73.6 percent of a recent sample of Greek children showed some degree of caries in their primary teeth.[4] Dental surveys in many parts of the world give the impression that similar increases have been the rule, with dental caries now almost universal among adults in civilized countries.

These changes all make dental caries look like a disease of civilization: a general hypothesis strengthened by observations upon present-day primitive tribes which have suddenly been overtaken by rampant dental caries coincidentally with the advent of modern prepared foods and the other appurtenances of a "civilized" environment. This is clearly suggested by Waugh in his studies of adjacent Eskimo settlements with and without imported white-man's food, and by Mellanby, who cites 17 primitive groups, only one showing as many as 28 percent of individuals with caries, and 23 recent European groups, most with over 80 percent caries and only one with as few as 46 percent.[5,6]

The introduction of water fluoridation and other preventive

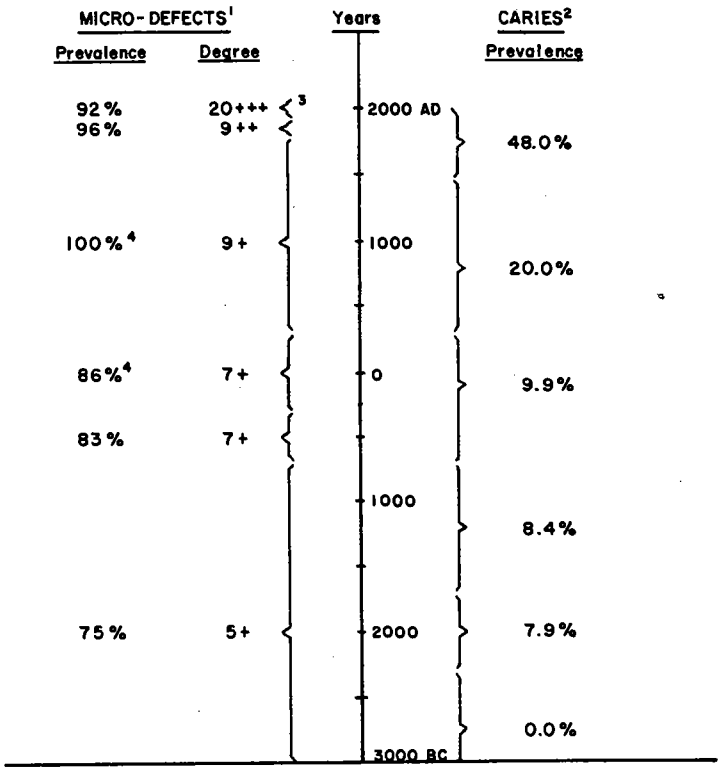

Figure 12. Prevalence of micro-defects (uncalcified interglobular spaces) and caries in teeth from Greek skulls, ancient and modern. From (1) Sognnaes, p. 547; (2) Krikos, p. 174. (3) The year 1948; (4) sample of fewer than 10 teeth.

measures in civilized areas were expected to make certain reversals in the upward trend of the disease, but suddenly during the late 1970s, reports of declining childhood caries began appearing from many developed countries, regardless of water fluoridation, and from nonfluoridated areas in the United States.[7] The developing countries, however, starting from severity levels below those of the developed countries, continued to show the familiar upward trend.[8]

Are these rapid changes in developed countries truly secular, as they are being called, or can specific causes be found for the de-

creases apart from elapsed time? A field for careful epidemiologic study lies ahead.

DENTAL CARIES AS A CHRONIC DISEASE

One of the outstanding features of dental caries is the long time it takes to develop. This is true not only of the individual lesion of caries, but the succession of lesions throughout the dentition as a whole, making caries a lifelong disease in most individuals who do not become edentulous at an early age.

What is a chronic disease? The time element of course must be present, but basically there are three types of chronic diseases and injuries. The first type is that which results from successful microbiological symbiosis. The most successful disease from the point of view of the bacteria is that which allows the host to live longest, and therefore a successful infectious disease is a chronic disease. Tuberculosis is a good example. The second type results from a defect in or breakdown of metabolism or structure. Diabetes fits this pattern. The third is nutritional-deficiency disease. Over a period of time these types often blend, or one type leads to another. All three processes appear to be involved to some extent in dental caries.

All chronic diseases by their very nature are influenced by many factors, and these factors are often duplicated from one disease to another. We can therefore list to advantage the host, agent, and environment factors for other well-known diseases in order to compare those diseases with caries. This has been done in Table 16, with tuberculosis and diabetes taken as examples of their respective types. To this table might be added a nutritional-deficiency disease such as rickets, but when one has listed age and vitamin D in this instance all other factors seem to assume secondary importance. For this reason rickets has not been included.

A glance at Table 16 will show the great similarity between host and environmental factors in tuberculosis, diabetes, and caries. To which of the three classes of chronic disease does caries chiefly belong? Caries resembles tuberculosis in that so far it has been impossible to produce it experimentally without the introduction of microorganisms and microorganisms may even be able to transmit it.[9,10]

Table 16. Factors influencing occurrence of three chronic diseases.

Locus	Tuberculosis	Diabetes	Dental caries
Host	Race	Race	Race
	Familial heredity	Familial heredity	Familial heredity
	Age	Age	Age
	Sex	Sex (menopause)	Sex
			Developmental defects
	Concomitant disease	Concomitant disease	Emotional disturbances
		Obesity	
Agent	Tubercle bacillus	None known	Plaque-forming streptococci and suitable carbohydrate residue
	a. Strain (human vs. bovine)		
	b. Origin (endogenous vs. exogenous)		
Environment	Nutrition	Nutrition	Nutrition
	Occupation	Occupation (sedentary)	Fluoride
			Climate (sunshine, temperature, relative humidity)
	War	Urban life	War
	Social level	(Climate?)	Oral hygiene
	Housing		

Dental caries resembles diabetes in predisposing factors, since defective tooth structure has been shown to be somewhat more susceptible to dental carries than healthy tooth structure.[6] This simile cannot be carried too far, however, since the defective structure of any tissue of the body would be likely to increase the susceptibility of that tissue to the invasion of those diseases associated with it.

Is dental caries a deficiency disease which we can prevent with the assurance now felt in the field of rickets? To a certain extent, of course, it is; for a very large measure of prevention is possible through the ingestion of proper nutrients, including small quantities of fluoride ion, and the exclusion of excess in the way of refined carbohydrates. There is some evidence that vitamin C de-

ficiency is associated with caries and more evidence that vitamin D deficiency is associated. Nevertheless, no nutrient or combination of nutrients seems the key to complete prevention of caries, and it falls far short of the facts to call caries a deficiency disease. All three major types of chronic disease, therefore, must be borne in mind, both in the collecting of data upon the epidemiology of dental caries and in planning public health programs for the control of caries.

HOST FACTORS

Race or Ethnic Group

Race or ethnic group has long been considered an important factor in the frequency of dental caries, yet little work has been done which would differentiate racial or ethnic heredity from environment. One of the best of the studies we do have is upon Army recruits during World War II.[11] A large study was made of draft rejection rates and a much smaller one of DMF tooth counts. Both gave similar results. The DMF material is reproduced in Fig. 13. The various racial and ethnic categories which are used are a little vague, yet considerable care was used in determining their limits. Environmental differences are probably at a minimum, since the recruits all resided in the same geographical area. The major contrasts which appear are not only in line with the clinical impression of most dentists, but are also corroborated at certain points by other studies. Specifically, both Chinese and black populations have been shown to have lower caries rates than corresponding white populations.[12] The U.S. Public Health Service National Dental Caries Prevalence Survey of 1979–80 confirms this as related to blacks.[13]

Age

It used to be generally believed that dental caries was "essentially a disease of childhood," and that its incidence among adults was very low compared with its pre- and postpubertal onslaught. This impression is borne out by the American Dental Association's 1965 survey of needs for dental care in the United States, where a very high peak in the number of teeth needing filling is seen to exist

between the ages of 15 and 24.[14] This peak, however, represents accumulated needs and not necessarily current needs or dental-caries incidence. The impression that caries is a disease of youth is further borne out by a study at the Metropolitan Life Insurance Company, where affected teeth per person and, more particularly, affected surfaces per person (the equivalent of DMF teeth and DMF surfaces) are shown to accumulate far more rapidly at ages 17 to 19 and 20 to 24 than at older ages.[15] Other studies in terms of DMF teeth show more uniform incidence of caries.[16,17] On grounds of probability alone it seems much more likely that the incidence of lesions of dental caries should be lower immediately after the teeth have erupted than it would be a few years later, since time is needed for lesions of caries to develop to an obvious

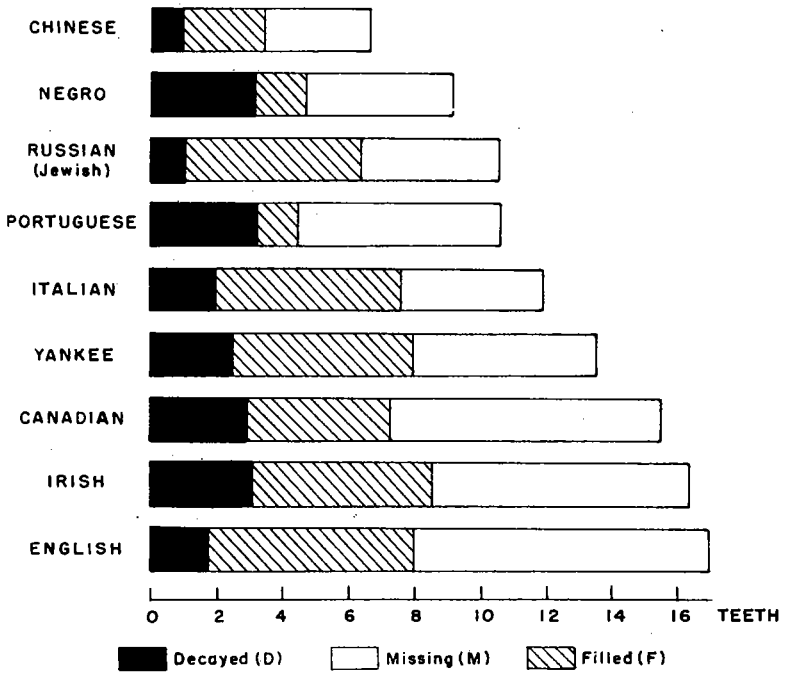

Figure 13. Relation of dental caries incidence to nationality. [Courtesy, *New England Journal of Medicine.*]

size. One would also expect caries incidence to be low in later life, when the more susceptible tooth surfaces in the average mouth have already decayed and when the number of remaining attackable surfaces is significantly reduced. Another factor operating toward lower caries in later life may be the gradual accumulation of fluoride in bones and teeth with advancing age everywhere, as noted in Chapter 16.

If this hypothesis were true, the best place to look for evidence would be among surveys in terms of surfaces, rather than DMF teeth. Large-scale surveys in terms of surfaces and covering a wide enough age span to be of interest are hard to find and are likely to be inaccurate if they do not include x-rays—at least a pair of bitewings. Evidence is accumulating that older teen-age children have more DMF surfaces per DMF tooth than do younger teenagers, a quite natural finding.[18] The work of Hollander and Dunning, among office workers over the age of 17, is expressed in surfaces and shows a gradual decrease in caries incidence with age, the most pronounced decrease occurring in the interval between 25 and 35 years of age. X-ray evidence is included in this study. Fig. 14 shows the data in question. These studies suggest that the greatest intensity of the caries process lies in the period from 15 to 30 years of age. The methods used to compile them are discussed in Chapter 14.

One exception to the trend just described occurs in ages over 60, with the development of *acute root caries,* a process named and described by Bodecker.[19] As root surfaces become denuded by gingival recession in advancing age, vulnerable dentine areas become well situated for the accumulation of bacterial plaque. Occlusal abrasion often contributes by permitting food impaction. If at the same time the patient's oral clearance ability deteriorates and his oral hygiene habits do too, an ideal condition exists for rapid development of caries. Lesions are broad and shallow at first, but if untreated can result in pulp involvement with surprising speed. Considerable tooth loss probably occurs among older people for this reason, but statistical studies to document the amount are lacking. There is evidence, however, that lifelong residence in fluoridated areas is accompanied by reduced root caries. Stamm and Banting have compared such adults with those living in nonfluoridated areas.[20]

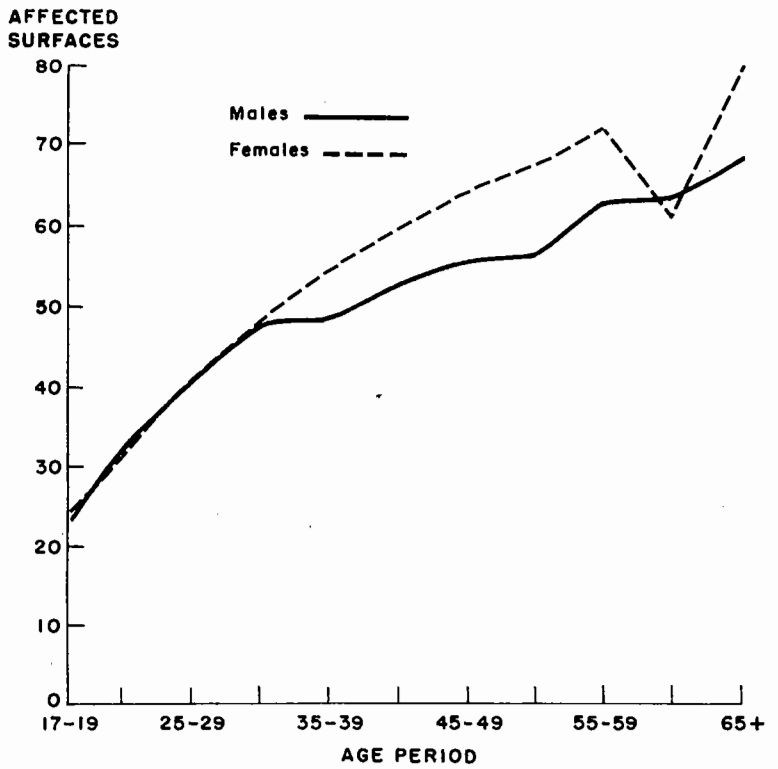

Figure 14. Affected surfaces per person by age and sex. Samples above age 60 are too small for reliable inferences.

Sex

Many statistical studies have been made to differentiate between the dental-caries experience of males and females. In young people, caries has been seen to be higher in the female to an inconsequential extent, but some studies show no significant difference between the sexes, and a few show slightly higher caries for males at certain ages but not at others. Above age 30, where statistical material is far scarcer, the study of the Metropolitan Life Insurance Company employees stands out in that it shows a very marked difference between the sexes (Fig. 14).[15] The difference appears first in the age group 35–39, and widens progressively after that to age 60. This study is impressive because most of the samples com-

posing it are of over 100 cases. The curve for caries experience in females here, if plotted on semilogarithmic paper as in Fig. 15, becomes a straight line, indicating a rate of increase proportional to some overall factor, perhaps the remaining attackable teeth in the mouth. The experience curve for males breaks sharply above the age of 30, as if some real change in caries susceptibility occurred about that time. Further work should be undertaken to substantiate this sex difference among older people, and, if it persists, an intensive study should be made of factors, perhaps physiological, which differentiate the male above 30 from the male below 30 and from the older female.

An impression has long been held that pregnancy accelerates dental caries in the female. No evidence has been found to substantiate this impression, in spite of several careful studies.

Familial Heredity

There is a widespread clinical impression that dental caries varies considerably from family to family, and that inheritance of a char-

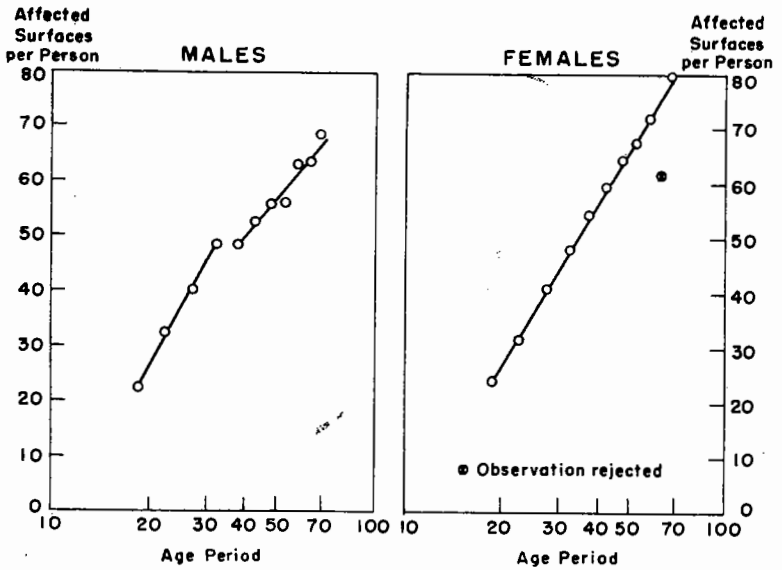

Figure 15. Affected surfaces per person in relation to logarithm of age by sex.

acterisic tooth structure, either good or poor, is common. Good genetic studies of caries incidence are few in number, and in such studies it is difficult to distinguish between true inheritance through the chromosomes and the transmission of dietary and other habits through family indoctrination. Twins offer perhaps the best opportunity for research upon this matter. Mansbridge finds greater resemblance in caries experience between identical twins than between fraternal twins, while unrelated pairs of children show less resemblance than either type of twin.[21] Summarizing the work of several authors on this subject, we may conclude that environmental factors have greater influence than genetic factors but that the latter also contribute to the causation of caries.

Emotional Disturbances

There is a widespread clinical impression that emotional disturbances, particularly transitory anxiety states, influence the incidence of dental caries. Most dentists have patients who have passed through periods of stress and associated high caries incidence, with a later return to a more normal mental health and caries rate. Such cases are extremely difficult to document because of the difficulty of defining stress and of relating it accurately to a chronic disease such as caries, but one such study is available, showing a sharp peak in caries incidence at times of acute stress, with return to previous caries incidence later.[22] Salivary changes have been shown to occur in connection with changes in mental health. Thus schizophrenics have been shown to have an increased rate of salivation, and salivary pH has been seen to rise under temporary emotional stress.[23] One study is available on dental disease itself, attempting to relate dental examinations in terms of DMF teeth to mental diagnosis among psychiatric patients.[24] Four major diagnostic groups were considered: primary behavior disorders and psychopathic personality (considered a continuum), schizophrenia, manic-depressive psychosis, and alcoholism without psychosis. Statistical analysis demonstrated a higher dental-caries experience at all ages among the manic-depressive group than in the baseline hospital population, significant *in toto* though not necessarily so at each age.

Concomitant disease other than emotional has often been considered to affect caries rate. The disease most often thought of in

this connection is diabetes, but statistical evidence of increased caries is completely lacking. Reduced dental caries has been demonstrated among controlled diabetics, probably due to the drastic dietary change needed.[25] Beyond this no particular disease seems to have been singled out. The emotional disturbance which so often accompanies systemic disease may turn out to be the common denominator which links these impressions.

Nutrition

Mention was made in the previous chapter of the difficulty of assigning a single logical place to nutrition in the list of host, agent, and environmental factors. Nutrition can be called a host factor to the extent that the individual instinctively selects specific foods from the array available to him, and after ingesting the foods metabolizes or excretes them according to a pattern dependent upon his normal physiology and his current state of health. Some people are natural protein feeders, others are natural carbohydrate feeders. In a civilized community with good transportation facilities, an extraordinary variety of food is actually available. Modern means of preservation and refrigeration in such a country as the United States permit an individual to construct almost any dietary regime he may wish, irrespective of the food-producing potential of the area in which he lives.

Insofar as a population is dependent upon locally grown food, and to the extent that climate and other factors influence the consumption of food in a given locality, nutrition is an environmental factor. Health education, directing a choice among foods, is an environmental factor. Nutrition is so much more often linked with environment than with host that it will be given detailed consideration later among environmental factors.

Variation in Caries within the Mouth

The epidemiologist in the dental field must deal, in effect, with populations of two different types: populations of people and populations of teeth within a mouth. While most of his work is likely to be with groups of people, the dental records which come to him are well suited to analysis of variations of caries within the mouth. Useful observations of this nature have so far grouped themselves under three main headings: (1) observations on types

of caries, according to tooth *surface* attacked, (2) observations upon the frequency with which the *different teeth* in the mouth are attacked, and (3) observations upon *bilateral symmetry.*

There are four main types of caries attack by surfaces. *Pit-and-fissure caries* is not only the easiest to detect, but also the first to appear and the easiest to explain in terms of tooth structure. To the extent that pits and fissures often represent actual structural defects in enamel, they constitute the most susceptible surfaces in the mouth. Attack commonly occurs fairly early in life.

Proximal caries is the next to appear. It is seen in the deciduous teeth toward the end of their life span, and in the permanent teeth predominantly between the ages of 15 and 35, after which it becomes less frequent.[26] It is easy to relate this timing of proximal caries to the fact that non-self-cleansing areas exist from the time of tooth eruption beneath the contact points of teeth. In view of the difficulty of restoring carious proximal surfaces, it is of interest that fluoridation is at its most effective in preventing proximal and other smooth-surface lesions.

Cervical caries is the third type of major importance. It occurs more or less uniformly through life, and can be related logically to the progressive changes in the free margin of the gingivae which increase susceptibility to plaque formation, and hence caries, with the advance of years. The fourth type of caries is *acute root caries,* described earlier in connection with the degenerative processes of old age.

Entirely aside from variation in the point of attack of caries, there are marked variations found between the different teeth in the mouth. Thus the lower incisors are far less frequently attacked than any other teeth, and are frequently the only teeth remaining in the mouth after all others are lost. Various articles document this situation. A good modern study is that of Backer-Dirks in Holland, giving details on intraoral variations in children 12 to 15 years.[27] The opening of major salivary ducts near the lower incisors has been advanced as a reason for this resistance to caries, but the opening of the parotid glands near the upper molar teeth has failed to give these teeth similar protection. We shall be much closer to an understanding of the etiology of caries when variations such as these have been explained.

Bilateral symmetry of caries in the human mouth may not help

our reasoning processes in tracing the causes of caries, but the knowledge that this symmetry exists is of great help to the public health worker in evaluating topical preventive measures for dental caries. Proof of bilateral symmetry can be found in various articles.[28] Knutson and Armstrong not only contribute materially to the evidence in their studies of the effect of topically applied sodium fluoride, but immediately put the knowledge to practical use by using one side of the mouth as a control against the other.[29]

AGENT FACTORS

Ever since the days of Miller, dental-caries research has been directed toward the identification of a microbial agent for the disease. Two generations ago a number of workers became impressed with the relation which appeared to exist between dental-caries rate and the number of lactobacilli in the mouth.[30] Whether or not these bacilli were causative of caries was not determined. Later, in working with germ-free rats, Orland et al. found that they were unable to produce dental caries in germ-free rats when feeding a diet which was highly cariogenic under normal circumstances.[9] This observation still stands, and has produced renewed attacks upon the microbiology of the oral cavity.

A large amount of information has now been obtained on experimental dental caries initiated by various bacteria in hamsters and rats. Human studies have progressed also, with attention centering on certain strains of streptococci, chiefly *S. mutans*. Gibbons and van Houte sum up current research:

> Collectively, the data indicate that *S. mutans* must be considered an important organism in the initiation of carious lesions on enamel surfaces, but unequivocal evidence concerning its direct involvement in human disease has yet to be obtained.
>
> As a consequence of the recent appreciation of the bacterial specificity involved in caries etiology, and particularly because of the apparent importance of *S. mutans*, efforts have been directed towards specifically controlling this organism . . . If enzymes are to be of practical use, difficult obstacles to overcome include: their mode of administration, their short contact time in the mouth, their slow diffusion into the plaque, their specificities, and the avoidance of continuous use.

> It is obvious that long term use of antibiotics and other chemotherapeutic agents which are of general medical importance is unadvisable for controlling dental caries. Nevertheless, the possibility exists that short term administration of these agents may be of practical value, particularly in severely infected or handicapped individuals.
>
> It has been possible to immunize experimental animals with vaccines of caries-inducing bacteria, and to obtain a partial reduction in dental caries development under certain experimental conditions . . . Immunization studies offer much promise, but they are still in their infancy, and the immunization of humans is only now getting under way.[10]

Not all agents need to be microbial. In the case of caries it has long been recognized that carbohydrate residues were essential. Workers now realize that these residues are not all equally conducive to plaque formation and multisurface caries. The rate of clearance from the mouth also affects the rate by which bacteria may act upon carbohydrate to produce acid. Foodstuffs with rapid oral clearance seem to be less dangerous than those which remain in the mouth for a long time.

With laboratory science now giving the epidemiologist definite objects for search, the study of agent factors have moved out into the field. Jordan et al. have searched for some of the best-known plaque-forming streptococci in various population groups and found them to be relatively common.[31] Their presence correlates with the extremes of caries activity on a group basis. Many other such studies have added to this picture, and others are to be expected in years to come, as agent factors are gradually linked to host and environmental factors.

ENVIRONMENTAL FACTORS

Major Geographic Variations

So many detailed environmental factors are dependent in one way or another upon geography that it seems of most interest to consider major geographic variations in dental caries first, and proceed from them toward more detailed factors. The writer has made an effort to analyze such major variations and from this analysis most of the material in the succeeding pages is drawn.[32]

In order to study geographic variations in dental disease apart

from racial or ethnic variations, it is necessary to select an area for study inhabited either by one racial or ethnic group predominantly or by such a mixture of ethnic groups evenly distributed over the area that variability from this source will be evened out in large samples. The United States is probably the best place to look for such material. A number of reports have been made upon the geographic variations in dental disease found in military populations within the United States. One study of children made by the U.S. Public Health Service was large enough to give regional and often local comparisons, but the fact that this study was based upon questionnaires makes one hesitate to use it for statistical analysis any more than necessary.[33] A few studies are available from other parts of the world showing variations in dental disease within widespread but racially homogeneous groups.

The term "dental disease" is used advisedly because, for lack of more accurate case histories, one cannot exclude teeth lost for reasons other than caries. Above the age of 40 or 50 years the tooth loss from periodontal disease seems to exceed loss from caries. Nevertheless, studies of decayed, missing, and filled teeth among children or young adults, and of military acceptance based upon these conditions, may be presumed to be predominantly studies of caries. The difficulty of obtaining accurate dental data from large geographic areas is tremendous, even when populations are racially and socioeconomically relatively homogeneous. Existing nationwide surveys are far from ideal. The larger the population groups to be studied, the more unknown variables are likely to be present, but also the greater is the chance that these variables will even out. We must do the best we can with the material we have. Any pattern seen consistently in large studies should be taken seriously.

The United States. The large studies of military populations available in the United States have been mentioned. These populations, while not completely homogeneous, represent predominantly the white race and have in common certain cultural and physical denominators that are relatively reliable. Since recruiting and draft standards are national matters, each study group has some uniformity in age composition and physical excellence within itself, state by state. The nationwide transportation of many common food items tends toward a more or less uniform diet. East,

Nizel and Bibby, and others have noted the similarity of the various existing studies as to relative amounts of dental disease found in certain major regions of the country at intervals since the Civil War.[34,35]

The three military studies best adapted to geographic analysis are those of Britten and Perrot, Ferguson, and Nizel and Bibby.[36,37,35] The first of these gives the prevalence of rejectable dental defects among men who were rejected or accepted for limited duty in World War I. The second study gives the average number of decayed, missing, and filled teeth per recruit among 4,602 white naval recruits seen at one induction center in peacetime. The third study gives average numbers of decayed and missing teeth per selectee among 22,117 men at a large Army camp in World War II. Each study has defects from a statistician's point of view. Internal comparisons, however, are justifiable on the basis of a rank list of states from 1, the state with the lowest prevalence of dental disease, to 48, the state with the highest.

Each of these three studies in itself shows a pronounced geographic pattern for the prevalence of dental disease similar to the other in major outline, but with occasional discrepancies. The act of combining the rank lists to give a mean rank seems to preserve the pattern but iron out many of the discrepancies. The mean ranks for states are plotted in Fig. 16. The list contains no number 1 and no number 48, since no state was at the beginning or end of the list in all three studies. Shading has been arranged to group the states of similar rank. It is of interest how clearly these groups cohere in a geographical pattern.

The pattern of prevalence of dental disease which emerges suggests two striking associations: one, latitude, the other, distance from the seacoast. It seems impossible to interpret the map without considering both of these factors. Statistical testing of the correlation between each of the factors and the prevalence of dental disease confirms the significance of the relation. A correlation among the states along the Atlantic coast from Florida to Maine, arranged in order of latitude, shows a coefficient of 0.844, with a probability of less than 0.001 that this coefficient could be the result of chance. A similar comparison among inshore states of different latitude gives a similar result. The states from Texas to

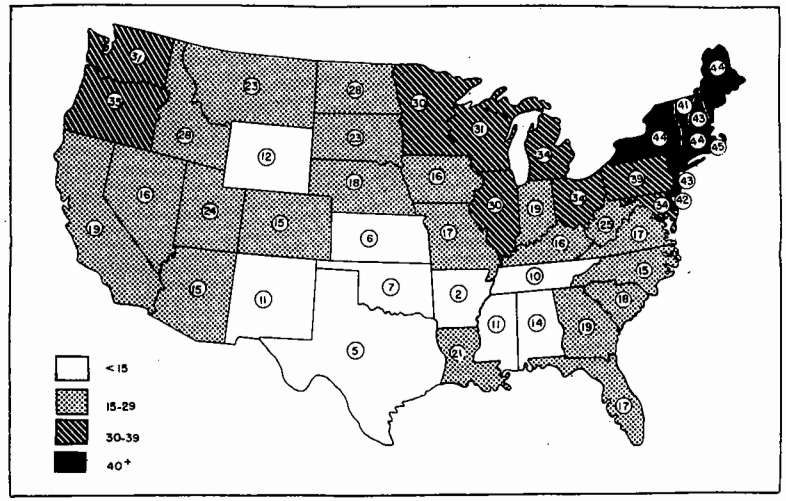

Figure 16. Mean rank of states in dental health from three nationwide military studies.

North Dakota show an almost perfect progression in prevalence of dental disease. The correlation coefficient between mean rank and latitude here is 0.923, with a probability of chance occurrence of just less than 0.01. Corroboration of this increase in dental disease with latitude is found in the U.S. Public Health Service National Dental Caries Prevalence Survey of 1979–1980.[38]

The relation of dental disease to distance from the seacoast in the military groups can be studied on almost any parallel of latitude. The 43rd parallel was chosen, since 11 states from New Hampshire to Oregon abut on or are crossed by this parallel. A correlation coefficient of -0.874 results, with a probability of chance occurrence of approximately 0.001.

South Africa. Ockerse has published a vast amount of data from the Southern Hemisphere which can be analyzed in a manner similar to that used in the United States.[39] His map of percentages of children with dental caries in the magisterial districts of the Union of South Africa gives a pattern which is almost the inverted image of that seen in the United States. Fig. 17 shows a plotting of these figures, with magisterial districts having more than 0.70 part per million of fluoride in the water supply excluded in order to

minimize the known influence of fluoride. The shape of the Union of South Africa makes it difficult to separate the influence of latitude from that of distance from the seacoast. For convenience, the latter has been used in a statistical analysis which demonstrates its significance.

The Eastern Hemisphere. Andrews studied 2,000 members of the Royal Australian Air Force, dividing his data by states.[40] He found that counts of DMF (decayed, missing, and filled) teeth vary considerably according to latitude. Analysis of the relation shows a certain amount of significance, particularly between Tasmania and the mainland.

The 1951 Yearbook for the Commonwealth of Australia gives interesting climatological data for the Australian capital cities which help elucidate the Andrews figures for dental caries.[41] Table 17 gives the data and the corresponding rank lists. It is interesting to note that mean annual hours of sunshine, temperature, and relative humidity all vary more or less together, the first two

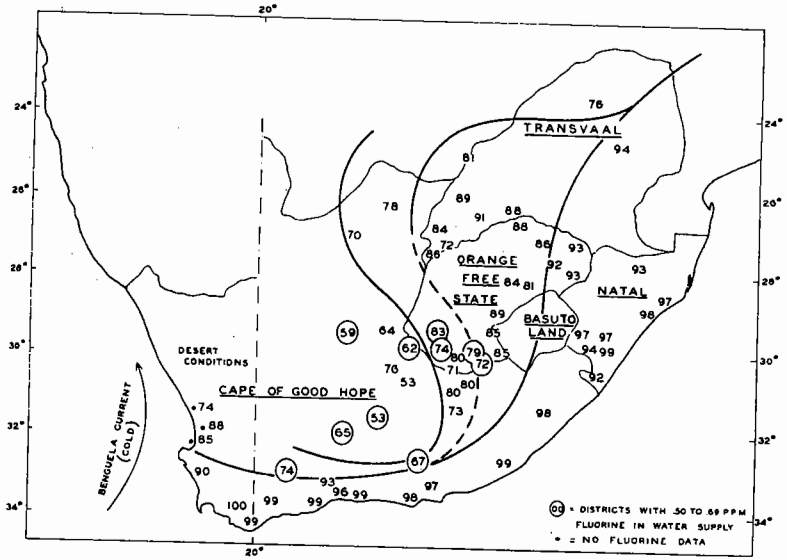

Figure 17. Prevalence of dental caries in districts with less than 0.70 ppm fluoride in the water supply, Union of South Africa. [Adapted from Ockerse.]

Table 17. Ranks of Australian states in dental disease compared with ranks of their capital cities in certain climatologic features.

State and city	Dental disease: DMF teeth	Rank	Mean daily sunshine: Hours	Rank	Mean temperature: Degrees F.	Rank	Mean relative humidity: %	Rank
West Australia Perth	17.50	1	7.8	6	64.5	5	62	2
South Australia Adelaide	18.57	2	7.0	4	63.1	3	52	1
New South Wales Sydney	18.66	3	6.8	3	63.7	4	68	4
Queensland Brisbane	19.72	4	7.5	5	69.0	6	67	3
Victoria Melbourne	19.74	5	5.6	1	58.8	2	69	6
Tasmania Hobart	21.98	6	5.8	2	54.4	1	65	5

Rank correlations (Spearman): dental disease-sunshine $r' = -0.771$; dental disease-temperature: $r' = -0.600$; dental disease-humidity: $r' = 0.829$.

rising as the third declines, and that all three are related fairly well to the variations in dental disease. Dental disease correlates best with relative humidity ($r' = 0.829$) and also fairly well with sunshine ($r' = 0.771$). Because of the small number of observations, only the coefficient for relative humidity approaches the 5 percent level of significance.

In other parts of the Eastern Hemisphere, the pattern of low caries near the equator and higher caries away from it seems carried out by studies of the Interdepartmental Committee on Nutrition for National Defense (ICNND), which coordinates nutrition studied in the armed forces and in civilian areas friendly to the United States, and by other related studies reported by Russell.[42] He lists New Zealand and Australia (the latter with greater population centers in the south) as relatively high caries areas; India, China, and Ethiopia are listed as relatively low.

Interpretation of Geographic Variations

The identification of variations in dental disease with latitude and distance from the seacoast will prove most useful if it helps us in a study of individual environmental factors influencing caries or periodontal disease. The largest group of such factors is climatological and includes sunshine, rainfall, humidity, and temperature. These factors are often related to one another, directly or inversely, but they deserve separate study. Chemical composition of water supply and urbanization are other important factors. Any effort to divide all these factors into those associated either with latitude or with distance from the seacoast is arbitrary, for a factor such as sunshine may often be related to both. Such an effort, however, is a necessary part of the analysis of the obvious geographic trends in dental disease and will help us to an understanding of the factors themselves.

Sunshine. One of the factors most commonly thought to vary with latitude is sunshine. The measurement of sunshine is not a simple matter. Total possible hours of sunshine per year actually increase a little as one leaves the equator, since long days in summer compensate for long nights in winter.[43] Actual hours of sunshine in the absence of cloud cover give a better measure and one which generally decreases as one leaves the equator. Even this does not tell as much as it might about sunshine available to human beings, for a given ground area intercepts less sunlight when the sun is low in the sky, temperature falls, and human beings clothe themselves or are driven indoors. The atmosphere, too, intercepts more ultraviolet light when the sun is low.

Mean annual hours of actual sunshine are shown on U.S. Weather Bureau Map No. 13, from which Fig. 18 has been adapted. This map shows a pattern very similar to that for dental disease in the United States (Fig. 16), at least in terms of latitude variation. Corroboration comes from East, who compares dental caries among the rural children reported in Public Health Bulletin No. 226 with the mean annual sunshine where they live and finds an inverse relation which is highly significant statistically.[44] Ockerse gives sunshine figures for certain areas in South Africa.[39] When these figures are compared with those for dental disease in the same areas a high rank correlation of -0.879 results.

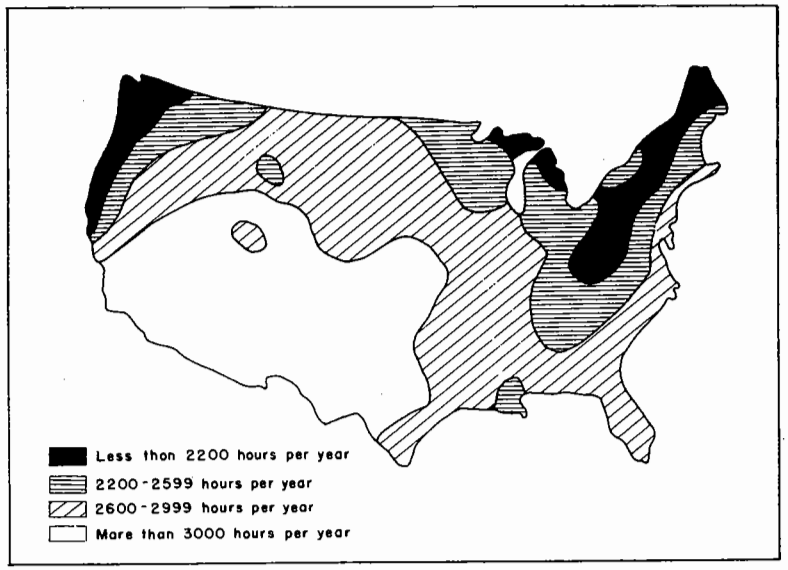

Figure 18. Mean annual sunshine, U.S. Weather Bureau Map No. 13.

This high correlation leads to consideration of the mechanism relating sunshine to caries. Ultraviolet light from the sun is known for its ability to promote synthesis of vitamin D in skin tissues, and thus to reduce caries incidence (Chapter 3). Ultraviolet light is blocked by the thickness of the atmosphere and by the water vapor it may contain. This phenomenon helps to explain several geographical parameters: sunshine (the absence of cloud cover), a high angle of incidence of the sun's rays upon the earth (greatest in low latitudes), nearness to sea coast (relative humidity), and even altitude above sea coast. Barmes in Papua, New Guinea, has observed a correlation of caries with altitude which needs further study.

Temperature. As Fig. 19 shows, temperature varies almost entirely with latitude. The only other factor to vary temperature seems to be high altitude, as is seen in the Rocky Mountain area. Temperature, in turn, acts to vary the caloric requirements and water intake of human beings. Carbohydrate food is not only a quick, but a relatively cheap, source of caloric energy. Our knowledge of the etiology of caries, therefore, indicates a way in which

this disease may be related to temperature. Data on variations of civilized diet with latitude or temperature are scarce. One study by the U.S. Department of Agriculture showed the consumption of baked goods (breads, cakes, and pastries not baked at home) to be higher in the North.[45] Consumption of sugar was also found to be higher among northern farm families than among farm families elsewhere in the country, though this contrast was not seen in a later study.[46] Further studies of carbohydrate consumption are needed, as well as further basic studies on carbohydrate nutrition and on methods of refinement.

Relative Humidity. This is the ratio of the amount of moisture in the atmosphere to the maximum amount that can occur without precipitation at a given temperature and barometric pressure. It is a better indication of the dampness of a climate than is actual precipitation. A mapping of the mean annual relative humidities over a period of years in the United States (Fig. 20) shows these humidities to be greatest along the immediate seacoasts, both Atlantic and

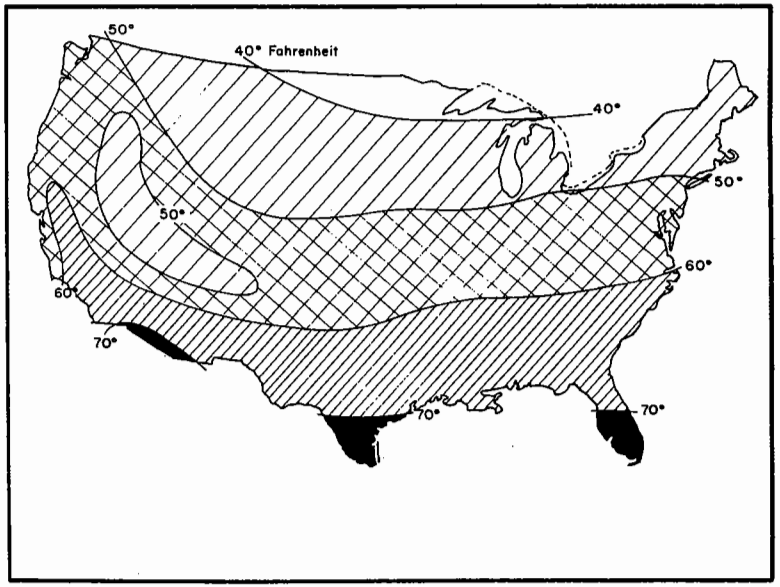

Figure 19. Mean annual temperature; U.S. Weather Bureau Bulletin S.

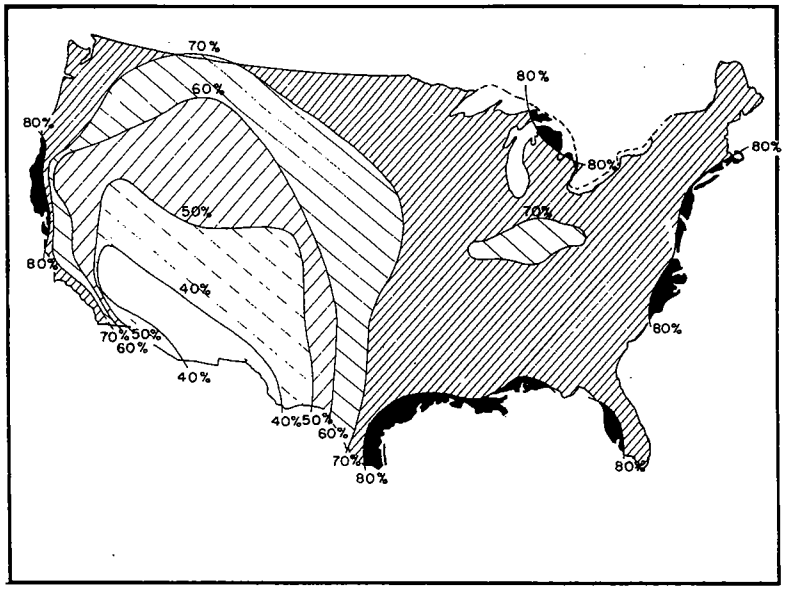

Figure 20. Mean annual relative humidity, U.S. Weather Bureau Bulletin O.

Pacific. From a short distance inshore, east of the Mississippi, most of the area shows little variation from percentages in the middle 70's (the "humid East"). The western states show sharp and regular variations with both distance from the seacoast and latitude, giving an appearance to the map much like that for dental caries. The data for individual weather stations in U.S. Weather Bureau Bulletin O are difficult to compare with state averages for dental caries, since many sharp changes in relative humidity cut across states, especially those on the seacoast. A better study can be done.

The data from Australian states (Table 17) show a higher correlation (0.829) between caries and relative humidity than between caries and any other climatic factor. Relative humidity, for these reasons, needs careful consideration as a factor in the causation of caries.

Rainfall. Another factor is rainfall, which leaches minerals from the soil and blocks sunlight. Fig. 21 shows mean annual precipitation for the United States. Though no latitude relation is evident, there is a regular decrease in rainfall as one proceeds inshore.

Only on the Atlantic coast is this pattern at variance with the one for prevalence of dental disease. Rainfall, though decreasing inland, is greater in the South than in the North. The mechanisms by which relative humidity and rainfall might be linked to dental caries, either together or separately, need further study.

Fluoride. Among nonclimatic factors, fluoride is the one that one would first attempt to relate to geographical variations in dental caries. Van Burkalow's mapping of maximum fluoride concentrations in communal and noncommunal water supplies on a county basis gives the most comprehensive material available for the United States.[47] No latitude variation is evident. Area for area, there are as many counties with fluoride maxima above 1.5 parts per million in the Dakotas as there are in Texas, and a similar situation holds to the east and to the west. Van Burkalow discounts climatic variations and feels that differences in geologic formation are probably more important. There is, however, some relation between fluoride maxima and distance from the seacoast. The coastal states seem to have somewhat fewer high fluoride maxima,

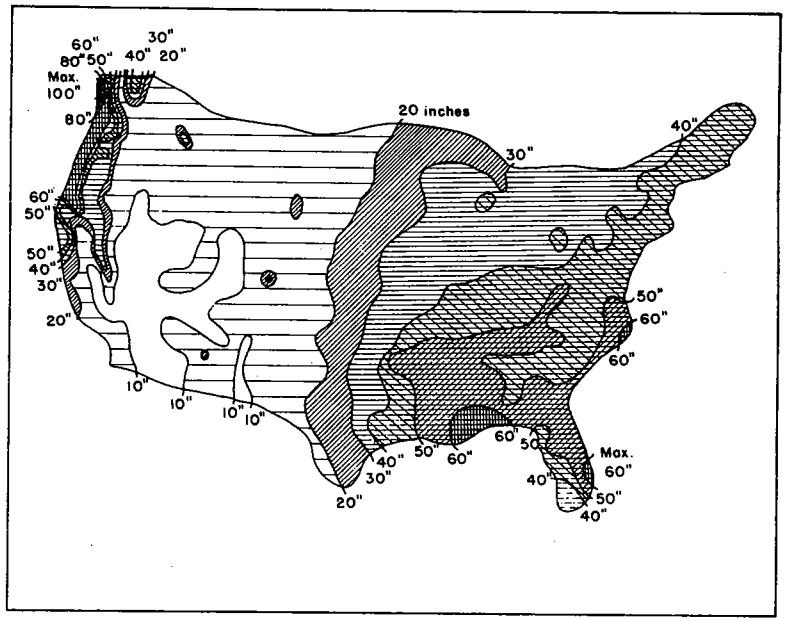

Figure 21. Mean annual precipitation, U.S. Weather Bureau Bulletin D.

though unmapped areas in these states contribute to the impression. Correlation between fluoride and distance from the seacoast is probably much better than is shown on Van Burkalow's map, for one deep-well water supply in a county may provide a high maximum figure, while the bulk of the county uses surface supplies (river, lake, or shallow well) which are almost always fluoride-free. Rivers are larger near the seacoast, the surface water is more commonly used there than it is inshore. Other aspects of the epidemiology of fluoride in relation to caries are taken up in Chapter 16.

Total Water Hardness. Usually measured in terms of calcium carbonate, total water hardness is an etiological factor in caries that has been known for many years. More recently it has faded from sight as attention was focused more sharply on fluorides. Röse[48] and Ockerse[39] are among the authors who have reported an inverse relation. Ockerse's correlation coefficient between calcium carbonate and percentage of caries is -0.811, even higher than his coefficient of -0.662 between caries and fluorides. If his data for calcium carbonate are plotted on a map zoned in hundreds of miles from the seacoast, a significant correlation coefficient of 0.462 is obtained for the increase seen as one proceeds inland. All of this returns our attention sharply to total hardness. Known components of hardness deserve further study. Trace elements must be studied and factors now unknown must be sought.

Trace Elements. A number of trace elements deserve attention, some found in water supplies but most found in greater concentration in common foodstuffs. Epidemiological studies must rely, therefore, not only on studies of local water supplies and perhaps soils, but upon concentration of the trace elements in the urine, hair, or other ultimate repositories. Hadjimarkos has found marked increases in dental caries in areas where selenium was high both in water and foodstuffs and the only environmental factor which could not be ruled out was sunshine. He summarizes this and other work on trace elements:

> Selenium is the first micronutrient element shown to be capable of increasing caries, particularly when consumed during the developmental period of the teeth and incorporated into their structure. Epidemiological studies on selenium and caries should be undertaken not only in high seleniferous areas but also in low-seleniferous ones. In the latter areas the element may be present in sufficient amounts to increase caries without producing other symptoms of selenosis which are clearly apparent, as was the case among Oregon children.
>
> The proposed caries-inhibiting influence of molybdenum and vanadium remains unresolved at present. Contrary to common belief, water supplies in general do not contribute significant amounts of micronutrient elements to the daily diet.[49]

Soils. Where populations depend largely on locally grown food products it is logical to look to differences in soil composition to help explain differences in caries experience. A number of studies upon soil have yielded negative or confusing results. The only really suggestive study to date relates to molybdenum content and acidity. Ludwig, Healy, and Malthus noted marked differences in caries between the towns of Napier and Hastings, New Zealand, without any environmental factor other than soil to account for it.[50] Differences in diet, fluoride, climate, and so forth were negligible. The soil of Napier, however, had higher pH, higher molybdenum, and the children there had lower caries. There is enough collateral evidence to justify further study of both these factors. In Hadjimarkos's work on selenium, the most logical source of this trace element was the local soil.[49]

Soil is not likely to prove an important element in programs for the prevention of dental disease, even if further study confirms relationships such as the above. Modern methods of preserving and transporting food products give our markets of today a variety that is bound to neutralize the effect of the local soil under all but extreme conditions.

Urbanization. Ferguson attributed the regional variations he found in caries to the greater frequency of large industrial cities in the North.[37] This claim deserves examination, and Public Health Bulletin No. 226 affords dental data on a number of cities, together with similar data on nearby rural county balances. The defects of Bulletin No. 226 have been mentioned. Nevertheless, errors should be at a minimum when comparing closely adjoining samples within it. Care was taken to select areas of similar distance from seacoast, lake, or major river. Urbanization was found to be accompanied by only an 11 percent increase in caries, which did not prove statistically significant.[32] The National Dental Caries survey of 1979–80 differentiates between regional levels and those of standard metropolitan statistical areas within them, with oppo-

site but probably insignificant results.[38] A careful study by the World Health Organization of metropolitan and nonmetropolitan areas in five cities in different countries shows a consistent but small trend in the opposite direction, with the high caries scores in the nonmetropolitan areas.[51]

A small, but probably accurate, study of urbanization is found in Fulton's report on the New Zealand national dental service.[52] A sample of 4,072 children was selected to include 2,048 rural and 2,024 urban children, all ages from 7 to 14 being equally represented. Small differences are found in DMF teeth, but Fulton states that "neither these differences in average DMF nor any other differences between corresponding figures . . . seem to be significant."

Nutrition

To the extent that broad geographic, cultural, or educational factors influence the food available to a population, nutrition is an environmental factor. Fluoride, available in a water supply, is similarly environmental; this matter is taken up in Chapter 16.

We have already discussed an instance in which temperature may affect food intake within a population able for the most part to make its own selection among a broad array of foods. A more striking example, where the question is not one of choice but of the actual availability of certain nutrients, is that of the Eskimos in their original surroundings. The lack of plant food in the Eskimos' usual habitat makes it necessary to seek animal food and in turn receive a diet consisting largely of protein and fat. The results have been excellent for the Eskimo dentition, as the work of Waugh[5] and others testifies. Only when civilized transport facilities permit the introduction of foods other than native do the Eskimo teeth break down.

By far the most interesting study of caries rates in native population groups throughout the world have come from the ICNND. Russell, working with this committee, reports widely different DMF counts from eight countries, as shown in Fig. 22. In commenting on these very diverse figures, Russell states:

> The lowest prevalence of caries was noted in Ethiopia and the Far East; an intermediate experience in Lebanon, representing the Near East; and the highest experience, paralleling that in the United States, in Central and South America. Sugar consumption as determined by ICNND dietary teams followed the same pattern.

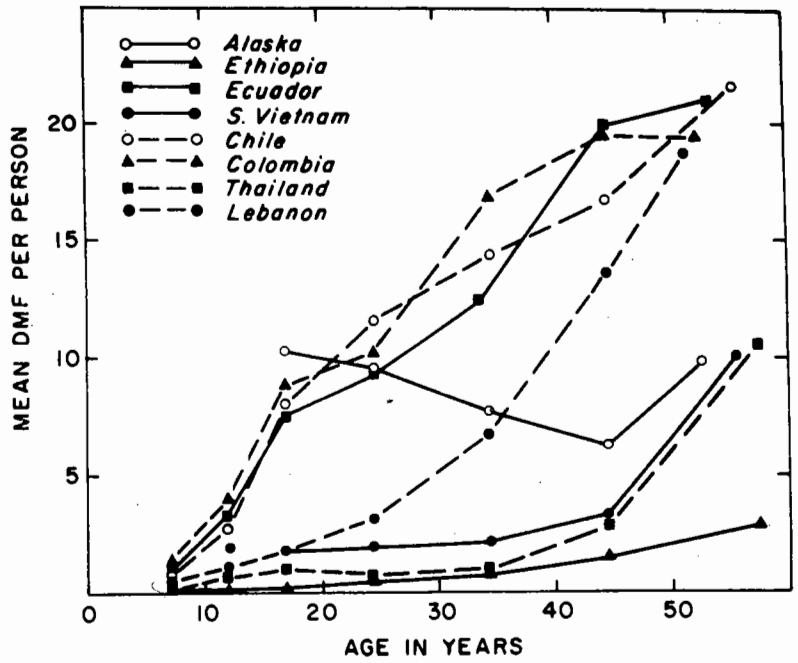

Figure 22. DMF teeth in eight countries, ICNND survey. [Reprinted by permission of Dr. A. L. Russell and *Journal of Dental Research.*]

High carbohydrate diets were not necessarily associated with high caries prevalence, unless sugar was a prominent factor. The principal food in Ethiopia is a cereal called teff, and rice is the staple in South Vietnam, Thailand, and Burma.

There is no suggestion anywhere in the ICNND data that any particular nutritional factor (except fluorine) is caries-inhibitory, or that caries is lessened by adequate nutrition.

It does not follow that starvation or deprivation is necessarily caries-inhibitory; if so, one would expect that within malnourished populations people with caries would be better fed than people without caries; but it is not so.[53]

Social Factors

Good *economic status* and *social pressure* in the direction of good mouth appearance are both strong factors in creating demand for

dental treatment. Evidence of these facts can be found in the American Dental Association's findings of differing needs for fillings and other dental operations among different economic groups.[54] The effect of social pressure can be seen on an international basis by comparing dentist-population ratios among civilized countries all well able to afford as many dentists as may be considered important. Neither of these relations is solely a measure of caries incidence, yet evidence does exist here and there that economic or social factors can affect caries incidence too, as may be the case in the major decline in developed countries. A good economic status carries with it a lower caries rate, most pronounced and best documented among preschool and young primary children but extending, in one instance at least, up through high school age.[55] In adults, there is a slight trend in the opposite direction. The National Center for Health Statistics finds low-income whites (but not blacks) to have fewer DMF teeth than their higher income counterparts.[56]

Industrial hazards to the teeth probably belong in the economic category, and will be discussed in Chapter 21. As examples, carbohydrate dust and acid fumes are both known to be deleterious to the teeth, the one promoting caries and the other chemical erosion.

On a broader scale than any peacetime variation in the structure of society is the influence of *war*. The physical surroundings of whole populations are altered by drastic dietary change, and there are many other environmental changes less easy to measure. Impressive evidence has been assembled by Toverud, Mellanby and Mellanby, and others showing reductions in dental caries after the third or fourth year of war in several European countries and continuing for several years into peacetime thereafter.[57,58] Analyzing reports from some ten different European countries, Sognnaes concludes that children's teeth there "show 1) a definite tendency to decrease in caries in the latter part of and following the recent wars. This is 2) most significant in young children, and 3) in those teeth of older ones which have developed or matured during the war years. Finally, and perhaps the most important, the general principle evolved from our analysis is 4) that there seems to be several years' delay in the initial effect of wartime conditions on the teeth, and, following the First World War even a greater delay in the return to prewar dental status."[59]

The predominant environmental modification to which this change in caries might be laid is a reduction in refined carbohydrates. Thus Norway before the war consumed 36.3 kilograms of sugar per person per year, and during the war only 10 kilograms per person. Other countries had comparable caries reductions, as in Fig. 23. In many of the countries where caries reductions occurred, the children were known to have remained in generally good physical condition, and as active as ever. From this and other evidence it is inferred that the children must have ingested more than the usual amounts of available natural foods, including a considerable bulk of less refined carbohydrates—potatoes, kohlrabi, wartime bread of high-extraction flour, and so forth. Sognnaes does not attempt to decide whether the reduction of refined carbohydrates or the introduction of favorable nutrients in the natural foods is the true cause of the caries reduction, but from a

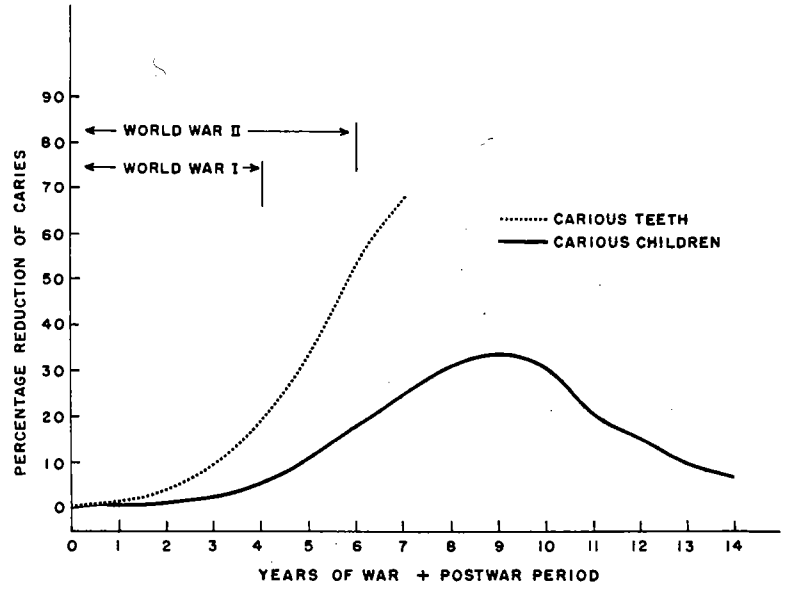

Figure 23. Year-to-year reduction of caries-susceptible teeth and carious children during the two world wars. Data on carious teeth represent averaged findings from Norway, Denmark, Finland, Sweden, and Britain; data on carious children are from Norway, Denmark, Britain, France, and Germany. [Adapted from Sognnaes.]

practical public health point of view the first change made the second change necessary and may therefore to a certain extent be considered causative. He does reason that the favorable influence, whatever it is, acts upon teeth before eruption and produces tooth structure which will withstand the renewal of high carbohydrate intake at the close of the war.

Sognnaes has reinforced this hypothesis by work upon rats and other animals changed at various stages in development from a stock diet to a purified diet high in sucrose and complete in known nutritional essentials. The changes were made at the prenatal period, before weaning, and after weaning.[60] Animals brought up on the sucrose diet from the beginning of the prenatal period had caries scores almost four times as high as those changed from a stock to a sucrose diet after the eruption of the teeth, as seen in Fig. 24. All this does not constitute a denial of the intraoral effect of carbohydrate upon caries. It may show instead a reduction in the reservoir of cariogenic streptococci which we now know are available for transmission from a mother to her offspring, either before or after birth.

Sognnaes's study and those of others in "undernourished" areas make it clear that a diet adequate in known nutrients, and balanced according to ratios currently thought desirable, cannot be claimed to be the only route to a low caries rate. Bacteria need to be present in the oral environment. The importance of the location of bacteria *in the mouth* is borne out by the study of Kite et al. on the tube feeding of rats.[61] Rats fed a cariogenic diet by stomach tube developed no caries at all, whereas their litter mates, fed the same diet by mouth, developed considerable caries.

Current Changes in Caries

During the 1970s a series of reports began to be received from various countries of the developed "Western World" to the effect that children everywhere were getting far less tooth decay than they were a couple of decades earlier. These changes were occurring in both fluoridated and nonfluoridated communities, though previously large decreases in the fluoridated areas left less opportunity for further change. A fairly large number of factors could be involved in these changes, in addition to fluoridation.

Is this a secular change, of the long-term, noncyclical nature seen in the Greek skulls? How can it be reconciled with an opposite

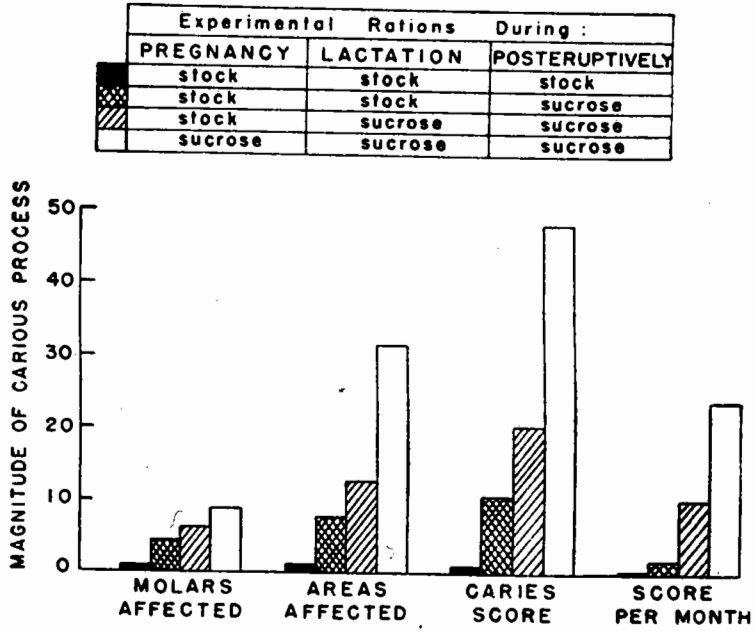

Figure 24. Caries incidence in groups of hamsters whose experimental feeding on the purified ration was commenced before, during, or after tooth development. "Sucrose" refers to the purified ration containing 67 percent sucrose; "stock" refers to the Purina laboratory chow. [Reproduced from *Science* by permission.]

trend in developing countries? Whether or not the term *secular* can properly be applied to it, the change needs careful description and further epidemiologic study. An appraisal of the effect of this decrease in childhood caries upon dental practice is attempted in Chapter 13.

The best description of the downward trend is found in the report of the First International Conference on the Declining Prevalence of Dental Caries, held in Boston in 1982.[7] Published as a special issue of the Journal of Dental Research, the report includes papers from Denmark, England, Ireland, the Netherlands, New Zealand, Norway, Scotland, Sweden, and the United States.[62] All tell the same story, whether in terms of DMF teeth, DMF surfaces, percentage of children caries-free (this one, up), restorations placed, or extractions performed. Drops of 30 to 40 per-

cent are common. Typical is that from the United States.[63] A large-scale comparison in DMF surfaces is provided by the National Center for Health Statistics Survey figures for 1971–1973 and the NIDR Dental Caries Prevalence Survey figures for 1979–1980. In this interval of approximately eight years, regional decreases of 28 to 39 percent are reported, with a national average of 32 percent. During this period, approximately one-half of the United States population, chiefly in the urban areas, were receiving fluoridated water.

The extent to which these decreases may be the result of individual fluoride preventive procedures in nonfluoridated areas can be inferred from Horowitz's eight-year study in a nonfluoridated area.[64] A combination of weekly rinses, daily supplement tablets, and a fluoride dentifrice produced reductions in DMF surfaces of 40 percent in 12-year-old children and 62 percent in 14-year-old children.

What factors can be postulated for the causation of this great change? Water fluoridation tops the list, but fluoridated dentifrices may be a close second. The list can also include children migrating from fluoridated to nonfluoridated communities, fluoride rinse programs, topical fluoride applications, better oral hygiene, and better nutrition. A final factor, as yet unmeasured, is the influence of antibiotics. A generation ago Helmut Zander studied a penicillin-containing dentifrice and recorded 48 to 60 percent caries reductions over a two-year period.[65] This dentifrice could never be generally marketed, but according to Gibbons, the increased use of antibiotics in treating childhood infections "has likely contributed to some degree to the decline in caries."[66] The magnitude of this contribution has yet to be determined.

On the other side of the coin is the story from developing countries. The World Health Organization Data Bank records upward changes of from 1.6 to 10.4 in DMF teeth over periods of 9 to 50 years, averaging 20 years.[8] Table 18 gives these figures.

The reasons for these increases can be inferred to some extent from earlier experience in the developed countries. The use of refined carbohydrate foods had increased more rapidly in these countries than the knowledge of oral hygiene and the increase in dental manpower. Native diets with associated low caries had given way to cariogenic diets, and only recently have preventive measures overtaken and reversed the upward trend in caries. Dental manpower increases have usually correlated with an upswing in dental health education as well as in a demand for dental care. Møller reported that seven developed countries showed ratios ranging between 1090 and 2890 people per dentist, whereas six developing countries showed ratios of 5520 to over one million persons per dentist. The rural areas in developing countries must indeed get no dental care at all, owing to the habit of 80 to 90 percent of all dentists to locate in urban areas.

These conflicting trends in caries incidence tend to make one wonder whether the downward trend in developed countries can really be called secular. Barmes observes that among 12-year-old children in developed countries the downward trend was toward 3 DMF teeth, and among children of similar age in developing countries the upward trend was also toward 3 DMF teeth.[67] Is it possible that these figures indicate an approximation to an endemic caries level which will actually be slightly greater than that of previous centuries?

The term *secular* may or may not apply to this situation. According to Webster, the term means "belonging to an age" or involving long periods of time, probably centuries. In practical public health work the term seems to be applied mostly where the factors causing change are unknown. To call the current drop in caries in developed countries secular may be allowable, but it is counterproductive if it discourages further search for specific causative factors.

Table 18. Increase in prevalence of dental caries in children aged 10–14 from selected countries.

Country	Increase in DMFT		Within no. of years
	From	To	
Ethiopia	0.2	1.6	17
Kenya	0.1	1.7	21
Iraq	0.7	3.5	9
Thailand	0.7	4.5	15
Vietnam	2.0	6.3	11
French Polynesia	negligible	7.5	50
Greenland	1.5	10.4	20

The foregoing mass of material, which has been primarily descriptive, is indicative of a variety of causative factors for caries. Fluorides and streptococci have made it possible to advance beyond the descriptive phase of epidemiological strategy (see Chapter 7) into hypothecation, analysis, and finally experiment. It is now important to do similar work in connection with the numerous other factors that will produce certain levels of caries attack in certain people at certain times and at certain places.