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Research Design in Oral Epidemiology

Definition of epidemiology is the study of health and disease in populations, and how of these states are influences by heredity,biology,physical environment, social environment and ways of living.

Epidemiology
- Desciptive : data only describe the distribution of a condition in population and no specific hypothesis is tested
- observational :observe outcome without intervening to affect them
- analytic : data and analysis are designed to answer a particular question

- The effect or outcome : in analytic studies in epidemiology is look at people with and without the disease in question and the exposure is look at people with and without exposure that may increase the risk of disease.
- Every study needs a protocol which is a wrriten plan encompassing the purpose and detailed operation of the study.
- Causality is a certain exposure results in particular outcome, can only be demonstrated with the experimental study design of clinical trial.A conclusion of causality criteria from nonexperimental epidemiologic analyses,known as the Bradford Hill Criteria.
- Risk factor is an environmental,behavioral or biologic factor confirmed by temporal sequence, ussualy longitudinal studies, which if present directly increases the probability of a disease and if absent or removes reduces the probability. Risk factor are part of the causal chain, or expose the host to the casual chain. Once disease occurs, removal of the risk factor may not result in cure. For example of risk factor is an occupation that requires handling toxic material, the occupation itself is not a risk factor, but because it brings person into toxic material then it does increase the chance of disease.
- If risk factor can not be confirmed by longitudinally, it will classified as risk indicator which is factor shown to be associated with the disease in cross sectional data and assumed to be the risk
- Another risk indicator is a risk marker is an attribute or exposure that is associated with the increased probability of disease although it is not considered part of causal chain. A risk marker can also be called a risk predictor when included predictive statistical models.

Nonexperimental study designs

- Survey is a collection data to be used for descriptive purpose
- Prevalence is the number or proportion of person in the population who exhibit a condition at any given time

- Cross-sectional is group of people who are or assumed to be sample of particular population are assessed at the same time.
- Longitudinal study is the same group of people is studied on two or more occasion so that incidence the change condition over time can be assessed.
- Sometimes an analytic study can be cross sectional, such as when several cross sectional studies are performed over a period of time for analytic purpose. An examination of mortality trends (obviously it cannot the same people who died on different occasion)
- Prospective studies is collect information on an exposure of interest and compare eventual outcomes, whereas retrospective begin with the outcome of interest and probe back for exposure information.
- A cohort study is prospective design, cohort is a group of people from whom data are collected longitudinally.
- An ecologic study is an analytic study in which data for both exposure and outcomes come from the population rather than from individuals.Such studies are relative quick and inexpensive because they avoid sampling, interviewing and clinical examination. Their weakness however is that they cannot certain people with the outcome condition are the same ones who had exposure.

Experimental study design

- A clinical trial is a controlled experimental study or group comparison, based on epidemiological principles and designed to test the hypotesis that a particular agent or procedure alters the natural history of disease.
- The group receiving the agent or regimen under study is the test group.or experimental or studygroup whereas the comparable group not subject to the agent or regimen is the control group.
- Choice of population to be conducted should be created to show that test is work.this mean that a population of a specific age range is usually chosen deliberately. For example sex,race,socioeconomic status and geographic location.
- Adequacy of the numbers of subject is important to anticipate loss of subjects during a prospective clinical trial ⟶ few subject ⟶ real difference cannot be detected ⟶ related to data analysis and the conclusion from the trial.
- Random allocation means that each participants has an equal chance of being assigned to either the study or the control group.
- The principle is simple, although is carefully planned and controlled procedure. It is not haphazard assignment. It needs stratification which is before allocation the base population is separated by those factors known (usually age, sex, race,socioeconomic status and previous disease experience.), subject from each stratum then ramdomly allocated to study and control groups.
- Placebo is a material or formulation like the test product but without the active ingredient. The purpose is to keep subject unware of whether they are in the test group or control group (blind study), so that their health behavior will not be consciously or unconsciously affected by group allocation.
- Researchers need to be sure that the agent is used as intended, the protocol must specified precisely how the agent will be used, for how long,how often, at what concentration and by whom. A placebo or positive control would be applied the same way. The purpose of the study must be kept in mind.
- The validity of clinical trial results depends on the reliability of the examiner

(s). Intraexaminer reliability is the ability of an examiner to record the same conditions and the same way over time. Interexaminer reliability is consistency beetwen different examiners. Standarization or calibration can be applied.

- Clinical trial must be continued long enough to permit detecton of new disease of extension of lesions already present. Example for carries trials, the minimum duration is usually 2-3 years.For plaque-inhibiting agents.8-21 days
- Power of the test dependent on : 1.The magnitude of difference observed beetwen the two treatments 2.The number of subjects in each group 3.The population varience 4.The alpha choosen
- When comparing the observed results in a clinical trial. It has been traditional to accept 5 % as outer limit of acceptable statistical significance.The statement p=0.05. To offer greater assurance that an observed difference is real, investigators may set alpha at 1 % (p = 0.01)。
- Type I errors usually arise from the fact that sometimes the random allocation process results in bias in the composition of the groups, it can come from absence of random allocation and from lack of blindness in either examiners or participans.
- Type II errors come from group sizes that are too small to permit demonstrating statistical significance.Imprecise diagnosis by examiners can also lead to type II error.
- For ethical considerations, human taking part must give informed consent,which means providing a wrriten acceptance that the participant understands the conduct of the trial an of any risk involved. Researchers must certify in the report of the trial that study protocol has been accepted by their institution`s review board.

The Measurement of Oral Disease

- This chapter describes the philosophies that underlie disease measurement and some of the procedures used.
- Using the characteristics listed in the definition of epidemiology, one which may related patterns among people who can be grouped by particular characteristics is :
  - Heredity : a person`s genetic endowment
  - Biology : age, gender, race
  - Physical environment : sanitation levels, food and water supply, air quality, occupational hazards, housing quality, neighborhood characteristics.
  - Social environment : educational attainment, cultural beliefs and practices, neighborhood quality
  - Lifestyle : smooking, exercise, dietary patterns, dental attendance,toothbrushing habits.

- Uses of the science of epidemiology
  1. Describing normal biologic processes example time and order of tooth eruption
  2. Understanding the natural history of diseases
  3. Revealing the distribution of disease ⟶ Indicating how disease occurs in the population by age, gender, race, geographic region and socioeconomic status.
  4. Identifying the determinants of disease by identifying the risk factors and risk indicators associated with a disease.
  5. Testing hypothesis for disease prevention and control ⟶ agents, regimens or procedures for the prevention and control of disease can be experimentally tested in clinical trial.
  6. Planning and evaluating health care service ⟶ data describe the distribution of disease, the population utilization, the avaibility and productivity of health care service can be employed to assist planning decisions on service and type of personal required.

Sampling from Human Population

- The sample should closely represent the population. Sampling error is the discrepancy between the sample and the base population in one or more important characteristics and with modern statistical methods it can be remarkably small.
- Probability sample is the chance of each person`s being selected in the sample is known,though not necessarily equal so the degree of sampling error can be calculated.

Methods of Measuring Oral Disease

- Counts = counting the number of cases of its occurance. Simple counts become less useful as prevalence increases.
- Proportions = a count becomes a proportion when a denominator is added, and prevalence is thus determined. Proportion do not include a time dimension.
- Rates = proportion that uses a standardized denominator and includes a time

dimension.

- Indexes = a numerical scale with upper and lower limits, with scores on the scale corresponding to specific criteria.
- Types of scale used in disease measurement : Nominal, Ordinal, Interval, Ratio.
- Other terms used is reversible and irreversible. An irreversible index is one that measures cumulative conditions that cannot be reversed : dental caries. And reversible can be reversed : gingivitis.
- Properties of an ideal index : Validity(measure what it is intended to measure), reliability (measure consistently at different times and under a variety of conditions), clarity, simplicity,and objectivity (the criteria should be clear and unambiguous), quantifiability (the index must be able to statistical analysis), sensitivity (the index should be able to detect reasonably small shift),acceptability (the index should not be painful or demeaning subject).

Examiner Reliability

- The measure most frequently used for expressing interexaminer reliability is the kappa statistic, a value between 0 and 1.0 that expresses the degree of agreement beyond that expected by chance alone.
- A reversal (a negative reversal) is a change of diagnosis in an illogical direction over a period of time, long enough for real change to have taken place.For example, when a surface scored as caries into dentin at the first examination is scored as sound 1 year later.

Measuring the Value of a diagnostic test

- The criteria for an ideal test are simple, inexpensive, acceptable in the patient, valid and reliable. A test should be sensitive (a positive result in those with the disease) and specific (a negative result in those who do not have the disease). Since there only a few tests that rate highly in both sensitivity and specificity, so the choice may be whether to use a tests that is highly sensitive but not very specific (which would capture a lot of false positive : people who test positive but really don`t have the disease) specific but not sensitives (Which would lead to a lot of false negatives : people who test negative but really do have the disease)

Test Result	Disease	No Disease	Total
Positive	TP	FP	TP + FP
Negative	FN	TN	TN + FN
Total	TP + FN	FP + TN	all

Sensitivity : Proportion of people with disease who test positive: TP / (TP + FN)
Specificity : Proportion of people without disease who test negative: TN / (FP + TN)
Positive predictive value : Probability that person who tests positive will have disease : TP / (TP + FP)
Negative predictive value : Probability that person who tests negative will not have disease: TN / (TN + FN)
False-positive rate : Proportion of people with positive tests who do not have disease : FP / (FP + TN)
False negative rate : proportion of people with negative tests who have disease : FN / (TP + FN)

Measuring Dental Fluorosis

Dental fluorosis is a hypomineralization of the dental enamel caused by excessive ingestion of fluoride during tooth development. Depending on the quantity and timing of fluoride ingestion during this period, the clinical appearance of fluorosis can range from barely noticeable changes to an ugly brown stain with pitting and flaking of friable enamel.

Dean`s Flourosis Index

An index of flourosis was seeded when the initial investigations of flourosis began in the 1930s.Dean`s first fluorosis index set criteria for categorizing dental fluorosis on a seven-point ordinal scale:normal, questionable, very mild, mild, moderate, moderately severe, and severe, but by 1939 his experience led him to combine the moderately severe and severe categories into single severe category. By 1942, Dean had revised his fluorosis index into a six point scale, including normal or unaffected enamel, that still finds some use today.

Criteria for Dean`s Fluorosis Index

1. Normal : The enamel represent the usual translucent semivitriform type of structure. The surface is smooth, glossy, and usually of a pale creamy white color.
2. Questionable : the enamel discloses slight aberrations from the translucency of normal enamel, ranging from a few white flecks to occasional white spots
3. Very mild : Small, opaque, paper-white areas scattered irregularly over the tooth but not involving as much as approximately 25 % of the tooth surface. Frequently included in this classification are teeth showing no more than about 1-2 mm of white opacity at the tip of the summit of the cusps of the bicuspids or second molars.
4. Mild : The white opaque areas in the enamel of the teeth are more extensive but do not involve as much as 50 % of the tooth.
5. Moderate : all enamel surfaces of the teeth are affected, and surfaces subject to attrition show marked wear. Brown stain is frequently a disfiguring feature.
6. Severe : Includes teeth formerly classified as moderately severe and severe. All enamel surfaces are affected and hypoplasia is so marked that the general form of the tooth may be altered.The major diagnostic sign of this classification is the discrete or confluent pitting. Brown stains are widespread and teeth often present a corroded appearance.

Tooth Surfaces Index of Fluorosis

By 1980s the tooth surface index of fluorosis (TSIF) was developed and used by researchers at the National Institute of Dental Research. The TSIF ascribes a score on a scale of 0-7 to each tooth surfaces in the mouth, whereas Dean`s index applies only to the two most affected teeth in the mouth.

The World Health Organization, however, still recommends use of dean`s fluorosis index in its basic survey manual. The TSIF is viewed as a public helath index rather than as a research tool. It does not call for dying of the teeth prior to scoring, on the grounds that when the appearance of teeth is judged in everyday life it is done so with the teeth wet. The very mildest forms of fluorosis are there likely to be missed

with the TSIF.

Thysltrup-fejerskov index (TF Index)

The resulting TF index has a stronger biologic basis than dean`s more or less arbitrary index, because the index scores were developed by relating them to histologic features of affected enamel. Since its use necessitates drying of the teeth, the TF index is the most sensitive of existing indexes. At the sme time, it requires assessment of only one surfaces per tooth because fluorosis affects all tooth surfaces equally. It can be used on selected teeth or the whole dentition and results again are expressed as distributions rather than as mean scores.

Fluorosis risk index

The fluorosis risk index (FRI) is designed for use in analytic studies that seek to identify risk factors for fluorosis, it explicity recognizes that the risk of fluorosis is related to fluoride exposure at particular stages of dentition development. It divides the buccal and occlusal surfaces of each permanent tooth into four zones based on the age at which calcification begins and selectively classifies each zone into one of two categories.

Measuring Dental Caries

DMF Index

- The DMF Index, an irreversible index, is applied only to permanent teeth. D for decayed teeth, M for Missing teeth due to caries, and F for teeth that had been previously filled. The DMF score for any one individual can range from 0 to 32, in whole numbers.
- A mean DMF score for a group, which is the total of individual values divided by the number of subject examined, it can have decimal value.
- The DMF index can be applied to whole teeth (designed as DMFT) or to surfaces (DMFS)
- To save time in a large survey, the DMF index can be used a half mouth, that is, it can be applied to opposite diagonal quadrants and the score doubled, an approach which assumes that the carious attack is bilateral.
- The DMF index for permanent teeth is always signified by uppercase letters, the equivalent index for the primary dentition is the *def* index. *d* for decayed teeth, *e* for teeth indicated for extraction, and *f* for filled teeth.
- Limitation of the DMF index :
  - DMF values are not related to the number of teeth at risk. A DMF score for an individual is a simple count of those teeth that in the examiner have been affected by caries. It has no denominator. A DMF score thus does not directly give an indication of the intensity of the attack in one any individual.
  - The DMF index gives equal weight to missing, untreated decayed, and well-restored teeth. Comon sense suggests that this way is faulty for many purposes.
  - The DMF index is invalid when teeth have been lost for reasons other than caries.
  - The DMF index can overestimate caries experience in teeth with preventive restoration. In an epidemiologic survey, such teeth must be included in the F component of DMF, although they had not been filled in the first place they might have been diagnosed as sound teeth. DMF score thus be inflated.
  - The DMF index cannot account for sealed teeth. Sealent did not exist in 1938 and thus are obviously not included in the description of the index. Here is where the DMF index shows it age, Sealent and for other composite restorations for cosmetic purposes have to be dealt with separately.

- Modification of this index are :
  1. the dmf index for use in the children before the age of exfoliation
  2. the dmf index applied only to the primary molar teeth
  3. the *df* index.
- Because of the present day skewed distribution of caries prevalence in the population, the Significant Caries Index (SiC index) was developed. It is based on the distribution of DMF values in a population, but is a way of expressing caries distribution that goes beyond mean DMF.
- Criteria for diagnosing **coronal caries**
  Diagnosis through the full range of caries :
  - 0. Surface sound : no evidence of treated or untreated clinical caries

- D1 Initial caries. No clinically detectable loss of substance. For pits and fissures, there may be significant staining, discoloration, or rough spots in the enamel.
- D2 Enamel caries : Demonstrable loss of tooth substance in pits, fissures, or on smooth surfaces, but no softened floor or the wall or undermined enamel.The cavity may be chalky or crumbly but there is no evidence that cavitation has penetrated the dentin.
- D3 : Caries of dentin : Detectably softened floor, undermined enamel, or a softened wall, or the tooth has temporary filling.
- D4 : Pulpal involvement. Deep cavity with probable pulpal involvement.

- Investigators in North America, Britain and the other English-speaking countries have traditionally recorded caries is diagnosed only as present or absent. (We refer to this as the dichotomous scale). In dichotomous recording, caries is only noted when it has reached the level of dentinal involvement, that is the D3 level.
- Caries diagnosis is also complicated by hidden caries, the name given to dentinal caries found radiographically beneath an apparently sound occlusal surface. Some see it by-product of the fluoride age, in which the original break in the enamel remineralizes before the dentinal lesion has reached the pulp, but natural history is really unknown. The possibility of hidden caries has led to a further look at the use of radiographs for caries diagnosis.
- **Root caries**, the RCI first described in 1980,was intended to make the simple prevalence measures more specific by including the concept of teeth at risk. The RCI is computed by scoring root lesions and restorations and nothing teeth with gingival recession, according to the following formula :

$$\frac{\text{(Root surfaces : decayed + filled) x 100}}{\text{(Root surfaces with loss of periodontal attachment : decayed + filled + sound)}}$$

- The index can be computed for an individual, for particular tooth types or for a population at large. An RCi of 7 % means that, of all teeth with gingival recession, 7% were decayed or filled on the root surfaces.
- The criteria for diagnosing root surface caries.
  - A discrete, well-defined, and discolored soft area is present
  - The explorer enters easily and displays some resistence to withdrawal
  - The lesion is located either at the cemento enamel junction or wholly on the root surface.
  - Restored root lesions are counted only if it was obvious that the lesions originated at the cementoenamel junction or is confined to the root surface completely.
- Early childhood caries (ECC) is the name given to extensive caries attack in infants and young children that seems to be associated with regular exposure to sugar, often from fluid in the bottle. Other names for the condition is baby bottle tooth decay, nursing caries, labial caries, and others.
- Assessment of the caries treatment needs of a group, at first glance, appears to be nothing more than the D segment of mean DMF score assessed from the survey. This approach, however, has been show not to work for the following reasons:

- Criteria used to diagnose caries in survey usually not the same as those used by practitioners in forming a patient`s treatment plan.
- Patien`s own perceived needs, level of interest in their dental conditions, and ability or willingness to pay all influence the level of treatment carried out.
- A practitioner has to judge whether a minor lesion will develop into major lesion over time, and whether a lesion in primary tooth can safely remain untraited for the life of the tooth. A survey scores a tooth by how it appears at the time of the surveys.
- Treatment philosophies change with expanding knowledge and technologic developments, a treatment that is standard today may not be so tomorrow.

Measuring Periodontal Disease

Measuring Gingivitis

- Gingivitis is inflammation of the gingivae without involvement of the deeper supporting tissues. The oldest reversible index is the P-M-A (standing for papillary-Marginal-Attached), which dates from immediately after World War II. As the inflammatory process became better understood, it gave way to the gingival Index (GI) of Loe and Silness. The GI grades the gingival on the mesial, distal, buccal and lingual surfaces of the teeth. Each area is scored on an ordinal scale of 0-3 according to the criteria

  0 : Normal gingival

  1 : Mild inflammation – slight change in color, slight edema ; no bleeding on probing

  2 : Moderate inflammation – redness, edema, and glazing ; bleeding on probing

  3 : Severe inflammation – marked redness and edema ; ulceration ; tendency to spontaneous bleeding.
- Gingival bleeding after gentle probing has become standard measure of gingivitis in clinical trials. Although visual assessment of inflammation (color, swelling) are subjective, the appearance of spots of blood after the probe more objective in those sites that are difficult to view directly.

Measuring Periodontitis

- Periodontitis is a bacterially included inflammation of the gingival tissues together with some loss of both the attachment of the periodontal ligament and bony support. The clinical manifestations of periodontitis come from the interaction between bacterial infection and the host response, basically it measurements by Clinical Attachment Loss (CAL) and probing depth.
- The most widely used periodontal index for many years was the periodontal index, first described by Russell in 1956. The PI was composite index, meaning that it scored both gingivitis and periodontitis on the same scale. Modern understanding has shown the PI to be invalid because it does not include evaluation of CAL, grades all pockets of 3 mm or more equally, and scores gingivitis and periodontitis on the same weighted scale. However, in the 1960s, the PI was viewed as an ideal field index and was used in a series of epidemiologic studies that correlated disease scores with clinical and social determinants.
- The same fundamental problem of a composite index was evident in the Periodontal Disease Index (PDI), intended as more sensitive version of the PI for use in clinical trials.Although the PDI is also no longer used, the indirect method of measuring CAL that Ramfjord described then is still employed today. The PDI also gave us the “Ramfjord teeth”, a set of six teeth taken to represent the whole mouth during examination. The ramfjord teeth are **the maxillary** right first molar, left central incisor and left first bicuspid and **the mandibular** left first molar, right central incisor and right first bicuspid. Ramfjord chose this group to save time in clinical examinations.
- In field studies today periodontitis is still measured by Ramfjord`s technique for the indirect measurement of CAL.
  1. The examiner measures probing depth from the gingival crest to the base of

the pocket.

2. the cemento-enamel junction is located and the depth from this junction to the gingival crest is recorded. The differences between these values gives an indirect measure of CAL. These measurements are usually carried out at between two and six sites per tooth, depending on the purpose of the study, either for selected teeth or for the whole dentition.

- Measurement of any disease is based on a case definition and case definition for periodontitis need are what depth of CAL at any one site constitutes evidence of disease processes, how many such sites need to be present in a mouth to establish disease presence, and how probing depth and BOP are to be included in the case definition.

Periodontal Treatment Needs

- The CPITN was first described in 1982 and with some promotion by the World Health Organization (WHO) it received worldwide use. The index is now referred to as the Community Periodontal Index or CPI. This change followed a workshop on the index in Manila in 1994. The workshop recommended that the CPI remain the global standard for data on health planning but that the treatment need codes be eliminated because they had become obsolete in view of current treatment methods.
- Codes and criteria for the Community Periodontal Index described by the World Health Organization

0 : Healthy gingival
1 : Bleeding observed, directly or by using the mouth mirror, after sensing (gentle probing)
2 : Calculus felt during probing but all the black area of the probe visible (3.5 – 5.5 mm from ball tip)
3 : Pocket 4 or 5 mm (gingival margin situated on black area of probe, 3.5 – 5.5 mm from probe tip)
4 : Pocket >6mm (black area of probe not visible).
X : excluded segment (less than two teeth present)
9 : Not recorded.

Plaque and Calculus

- The Simplified Oral Hygiene Index (OHI-S) had wide use in surveys. It is quick and practical to apply, although it lacks sensitivity. The OHI-S scores calculus and plaque together, both supragingivally and subgingivally.
- Silnes and Loe developed the Plaque Index (PII) to be used along with their GI. The same surfaces of the same teeth are scored as in the GI and a 0-3 ordinal scale is again used.The principal difference in approach between the PII and the OHI-S is that the PII scores plaque according to its thickness at the gingival margin rather than its coronal extent, a measure claimed to be more valid.
- Scores and Criteria for the plaque index

0 : No plaque in the gingival area
1 : A film of plaque adhering to the free gingival margin and adjacent area of the tooth. the plaque may only be recognized by running a probe across the tooth surface.
2 : Moderate accumulation of soft deposits within the gingival pocket, on the gingival margin and/or adjacent tooth surface, which can be seen by the naked eye.

3 : Abundance of soft matter within the gingival pocket and/or on the gingival margin and adjacent tooth surface.

Partial-Mouth Periodontal Measurements

- Because full mouth examinations for gingival bleeding, CAL, plaque and calculus can be time consuming, investigators have tried using various indexes on a subset of teeth to save time. The expectation is that the subset of teeth will act as a representative sample of all teeth in the mouth, yielding information that can be applied to the whole mouth but taking much less time to examine. Partial-mouth recording was pioneered by Ramfjord with the Ramfjord teeth subset in 1959, and the CPI uses it today.
- Partial mouth recording is adequate for surveys in which a degree of underestimation is an acceptable trade-off for lower costs, but it is not recommended for use in clinical trials or in any other situation that demands a high degree of precision data. It is a pragmatic measure, not recommended for analytic research but adequate for surveillance.

Tooth Loss

This chapter reviews the issues and trends in tooth loss and the reasons why people lose teeth.

The Historical Picture

- During the nineteenth century, much of the work of dentists was still devoted to tooth extraction. Caries was rampant at this time, restorative techniques crude and painful, prevention unknown. As result people expected to lose teeth and dentists expected to extract them.
- During world war I (1914-18) was generally dreadful. The response of authorities was to extract more teeth, so that troops preparing to bayonet would not be bothered by toothache..
- After the world war I, surveys in New York found that school girls ages 13-17 years had lost 13.5% of first molars and 2.5 % of second molars, whereas adults of different ages had lost 22 % - 47 % of first molars.
- During world war II (1939-45), extensive tooth loss was still common in the United States.
- By the beginning of the twenty-first century tooth retention was much improved in all the high-income nations. Change came about with improvements in restorative dentistry (especially the development of the air turbine dental engine in the late 1950s), increasing affluence and accompanying positive attitudes toward tooth retention.

Edentulism

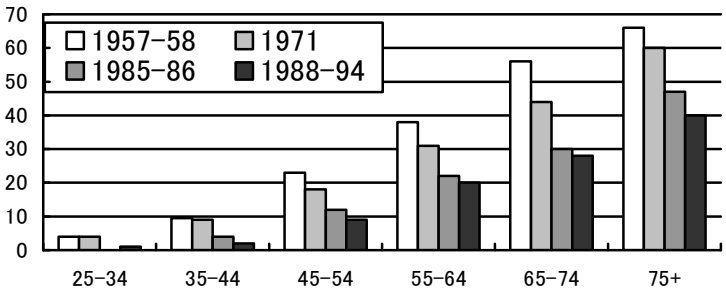

Fig.1 Proportion of the US population edentulous in 1957-58, 1971, 1985-86 and 1988-94 by age.

- Many people are puzzled by the apparently slow rate of progress in decreasing edentulism when we constantly hear of rapid improvements in oral health status. When the data in fig.1 are considered, it can be noted that the youngest individuals in the 75-and-older cohort for 1988-94 were born in 1919, and most were born earlier. The early adult years for this group were during the great depression of the 1930s and many of them then served in world war II. Indeed, it is probable that a good proportion of this cohort first became edentulous during that war, and they have been influencing the statistics ever since. In time,these aging cohort will be replaced by the baby boomers, who grew up in a

totally different world of affluence and modern disease prevention, and with fundamentally more positive attitudes toward tooth retention.

- In the United states, there has historically been a greater degree of edentulism among whites than among African-Americans, perhaps because whites have traditionally had better access to dental care and thus were at greater risk of having teeth extracted.
- Edentulism is tightly related to socioeconomic status (SES) but these SES-related differences consistently are found in many societies and probably reflect expectations and health attitudes at least as much as occurrence of oral disease.
- Edentulous people have also been found to have more risk factors for cardiovascular disease than dentate people, and it should not be surprising that older people in good health enjoy greater tooth retention than do people of the same age in poor health.

Partial Tooth Loss

- Like edentulism, the extend of partial tooth has been diminishing in the united states as caries comes under control, more and better treatment becomes available, and attitudes toward tooth retention improve with increasing affluence. In contrast to edentulism, in which attitudes are major factor in a person`s decision to have all the teeth removed, partial tooth loss appears to be more closely related to oral disease.
- Longitudinal studies to identify risk factors that lead to tooth loss, either total or partial, have not been very successful. Smoking, not surprisingly, has been identified as a risk indicator, and early tooth loss was found to be strong predictor of subsequent edentulism. SES in early life is also a demonstrated predictor of tooth loss on early adulthood.

Reasons For Tooth Loss

- Conventional wisdom for many years was that caries was the main reason for tooth loss before age 35 and periodontal disease the main reason after age 35, this belief was based on some ancient and rather dubious data.
- From around the mid-1980s, studies in a number of countries and among different types of populations have been consistent in finding that caries is the principal cause of tooth loss at most ages, with the possible exception of the oldest. Data on which these conclusion were based came from surveys of practitioners, reviews of dental records, and examinee questioning or diagnosis during survey examinations

Dental Care and Tooth Loss

There are two major reasons for continuing improvement in tooth retention :

1. The modern preventive dental treatment, so that most present day dentists extract teeth only when there is no practical alternative.
2. Positive attitudes among today`s adults, both younger and older. Tooth loss for many of them are simply unacceptable.

In summary, tooth retention is improving because of better prevention and control of the oral disease, more positive attitudes toward tooth retention and more conservative dental treatment philosophies.

Dental Caries

This chapter examines the distribution of dental caries in populations and the factors that influence that distribution

Global distribution of Caries

- For most of the twentieth century, caries was seen as a disease of the high income countries, but with low prevalence in poorer countries. The most obvious reason for this pattern is diet. The high level of consumption of refined carbohydrates in the wealthier countries led to selective proliferation of cariogenic bacteria. Poorer societies, on the other hand, survived by hunting and farming, both of which provided diets low on fermentable carbohydrates.
- When caries was more prevalent and severe than at the present, affected teeth were attacked within 2-4 years after eruption. By the early 1980s, there were reports from local surveys to suggest that average prevalence and severity of caries among children in the united states was declining from its previously high levels. Similar information from other high-income countries around the same time indicated that this reduction in caries experience was widespread.
- As caries prevalence falls, the least susceptible sites (proximal and smooth surfaces) reduce by the greatest proportion, while the most susceptible sites (occlusal) reduces by the smallest proportion. Although the total number of new carious lesions has been declining, an increasing proportion of them is made up of pit and fissure lesions. This trend has enhanced the attractiveness of fissure sealants as a preventive measure.
- No clear reasons for the caries decline have been identified, although most researchers view the various uses of fluoride as the main cause.

What does caries free mean?

The term caries free has traditionally been used to describe people with DMF score of 0, usually when the presence of a dentinal, or D3, lesion is the stated or implied criterion for caries. As the understanding of caries has increased, it has become evident that very few people are literally caries free. Just about everyone, at any given time, has some level of carious activity taking place. Most of this activity consists of early demineralization-remineralization cycles or a white spot or stained fissure that does not progress. In a healthy mouth, the bulk of this activity does not reach the stage where restorative dental treatment is needed, although preventive intervention may be called for. But this still means that term caries free is not correct. Perhaps more importantly, use of this term can tend to promote a mindset that caries does not matter, or perhaps does not even exist, until it involves the dentin. That is clearly incorrect, for preventive treatment at this stage can forestall the need for later restorative treatment. A most accurate term would be free of caries requiring restorative treatment, but that is much too clumsy for everyday use.

Caries Distribution:Demographic Risk Factors

Age

- Mean DMF scores increase with age, for school children and for adults. It can be seen that increase with age for the children's cohorts comes largely from an increase in numbers of restored teeth, whereas for adults most of the increase with age comes from missing teeth. Both figures are from cross-sectional data,

so as younger cohorts replace today's older people, the M component will decrease.

Gender

- Females have usually demonstrated higher DMF scores than do males of the same age, although this finding is not universal. In national survey data, males usually have more untreated decayed surfaces than females, and females have more restored teeth. Females visit the dentist more frequently so this observation is perhaps to be expected. In NHANES III, females ages 12-17 years had the same mean number of decayed and missing surfaces as their male but 25 % more filled surfaces. We cannot conclude from these figures that females are more susceptible to caries than are males, a combination of earlier tooth eruption plus treatment factor is a more likely explanation for the observed differences.

Race and Ethnicity

- Long-held contentions that certain races enjoy a high degree of resistence to dental caries probably came with early observations that some non-european races, such as those in Africa and India, enjoyed a greater freedom from caries than the Europeans. Today, however, we accept that global variations in caries experience result more from environment than from inherent racial attributes. No reason to believe that inherent racial differences exist in caries susceptibility among African and Americans; people of Hispanic origin, and whites. Socioeconomic self-care practices, attitudes, values, available income, and access to health care appear to be far more important.

Socioeconomic Status

- Socioeconomic Status as measured by such factors as education, income, occupation and place of residence, however, obtaining a valid measure of SES is always a problem because of its complexity. For example In the United States SES is usually measured by annual income or years of education, despite acknowledged shortcomings in these measures
- Relationships between caries status and broad range of SES measures (residence in private versus public housing, car ownership, quality of neighborhoods) have also been reported in Britain. The British studies noted that, although fluoridation of water supplies reduces the differences between the social classes, it does not entirely remove it.
- These studies collectively demonstrated that dental caries today can be looked upon as a disease of poverty or deprivation. The greatest reductions in caries experience have been enjoyed by the upper social groups, whereas reductions in the lower social groups have been more modest. When treatment programs are planned, caries experience can be expected to be more extensive and severe among lower SES populations.

Familial and genetic Patterns

- Studies of identical twins have concluded that, although genetic factors could have affected caries experience to some extent, the influence of environmental variables was stronger.
- With the explosion of research discoveries of genetic influences in many disease, dental caries is being viewed in a different light. It is likely that host attributes which could affect an individual's caries experience, such as salivary flow and composition, tooth morphology and arch width, are genetically determined and the genetics of the cariogenic bacteria themselves must have an effect. The

rapid growth of research technology and interest in genetics holds promise that a new view of caries will emerge in the future.

Caries Distribution:Risk Factors and Risk Indicators

Bacterial Infection

- Caries is a bacterial disease. The bacteria principally involved, the mutans streptococci and lactobacilli, are normal constituents of the flora in most mouths, so in that sense caries can be seen as an ecologic imbalance rather than an exogenous infection.
- Cariogenic bacteria are transmissible, usually passed along from mother to child. The complexity of bacterial interactions in caries development is illustrated by the finding that establishment of streptococcus sanguinis in an infant appears to be antagonistic to the colonization of streptococcus mutans.
- The evidence is not yet clear enough to permit quantification of the risk attributable to specific bacteria, which in any case may vary in different populations. However, because infectionus with cariogenic bacteria is a necessary condition for caries to occur, it is obviously a risk factor for caries.

Nutrition and Caries

- The term diet refers to the total oral intake of substances that provide nourishment and energy, whereas nutrition refers to the absorption of nutrients.
- There is evidence from studies of children in Peru that chronic and severe malnutrition during the first year of life is associated with increased caries years later, although this association is difficult to demonstrated because malnutrition delays eruption and exfoliation of the primary teeth. Chronic malnutrition among children in India has been shown to reduce salivary flow, which could be one reason for causative link.
- In the United states, no relation between nutritional adequacy and DMF scores could be found in NHANES I in 1971-74.
- The limited epidemiologic evidence resulted the conclusion that severe chronic malnutrition during infancy can predispose people to later dental caries. This situation is found in countries where malnutrition during early childhood is common but where there is later exposure to cariogenic foods, the malnutrition itself does not produce caries without the later cariogenic challenge. In the high-income nations, this degree of severe malnutrition is rare and is seen only in highly unusual circumstances.

Diet and Caries

- In contrast to nutrition, diet has a clear influence on caries development. In particular, the relation between the intake of refined carbohydrates, especially sugars and the prevalence and severity of caries is so strong that sugars are clearly a major etiologic factor in the causation of caries.
- Sugars are not the only food sources involved in the carious process. Cooked or milled starches can be broken down to low molecular weight carbohydrate by the salivary enzyme amylase and thus act as a substrate for cariogenic bacteria. By contrast, the high molecular weight carbohydrates in lightly cooked vegetables are considered noncariogenic because so little breakdown of these foods occurs in the mouth.

Caries and Soft Drinks

- There is more recent evidence to show that soft drink consumption is related to

caries, the more soft drinks consumed, the greater the extent and severity of caries.

Root Caries

- Root caries is defined as caries that begins on cemental root surfaces below the cervical margin. It thus is found only where loss of periodontal attachment has led to exposure of the roots to the oral environment and hence to the accumulation of bacterial plaque around these exposed roots. Root caries appear to be poly microbial, with the baterial composition of dental plaque in root lesions apparently little different from that of plaque found in coronal lesions. As with coronal caries, sugars are part of the etiology.
- Root caries by definition is strongly associated with tooth loss of periodontal attachment. Other factors found to be associated with root caries are primarily socioeconomic, such as years of education, number of remaining teeth, use of dental services, oral hygiene levels, and preventive behavior. An important risk factor is also the use of multiple medications among the elderly, a common practice in nursing homes and one that can promote xerostomia (salivary diminution).
- People who suffer from coronal caries also seem likely to be at risk of root caries when gingival recession occurs, and root caries is less prevalent in high-fluoride areas than it is in low fluoride communities. Smokers exhibit more root caries than nonsmokers and severity tends to be inversely related to the number of teeth remaining.
- Root caries seems to be a particular problem among older people of lower SES, those who have lost some teeth, those who do not maintain good oral hygiene, and those who do not visit the dentist regularly. Because of the aging of the population and increasing retention of teeth, the dimensions of the root caries problem are likely to continue to grow in the future, even if the number of lesions per person shows little change. The attention of dental practitioners should be increasingly devoted to treating and preventing root caries in adults, as less time is needed to deal with coronal caries in children.

Periodontal Disease

This chapter describes the epidemiology of gingivitis and adult periodontitis, their distribution and the risk factors and background characteristics associated with them.

Periodontal Infections and Host Response.

- Gingivitis is an inflammatory process of the gingival in which the junctional epithelium, although altered by the disease, remains attached to the tooth at its original level. There are initial, early, and established gingivitis lesions and a sequential microbial colonization leads to bacteriologically more complex plaque as the lessons progress.
- Periodontitis is also an inflammatory condition of the gingival tissues, characterized by clinical attachment loss (CAL) of the periodontal ligament and loss of bony support of the tooth. Periodontitis develops as an extension of gingivitis, although only a small proportion of gingivitis sites makes this transition.
- What happen in this transition is that supragingival plaque serves as a reservoir for periodontopathogenic organism, and when this infection is strong enough to overwhelm the host defense, bacteria in supragingival plaque migrate subgingivally to form a subgingival biofilm. Inflammatory mediators play an important role in the progression of periodontitis.
- There are two distinct types of periodontitis. One is the plaque and local factors type, the most common form, in which specific patogens dominate the host response in controlling disease expression. The second is the compromised host type in which severity and rate of progression are often rapid and are not well correlated with local factors like plaque deposits. The compromised host type is less common, responds much less favorably to standard treatments and is thought to be the type of disease found in aggressive and diabetes-associated periodontitis.

Current Models of Periodontal Disease.

Basic, clinical and epidemiologic research from around the late 1970s onward has led to a perception of the periodontal disease that can be summarized as shown in the buttom

- Only a small proportion of persons (5%-15%) exhibit severe periodontitis, where severe means that tooth loss occurs or is threatened.
- Gingivitis and periodontitis are associated with bacterial flora that have some similarities but also some differences between the two conditions. Gingivitis precedes periodontitis, but only a fraction of sites with gingivitis later develop periodontitis.
- Although periodontitis is usually related to age in cross sectional surveys, it is not a natural consequence of aging
- Periodontitis is not the major cause of tooth loss on adults, except perhaps in the oldest age groups in some populations.
- Periodontitis is usually site-specific condition and is only occasionally seen in generalized forms. Generalized periodontitis is usually severe and of the early-onset type.
- Periodontitis is usually thought to proceed in bursts of destructive activity with

quiescent periods between the bursts.

Distribution of Periodontal Disease

- Data collected since 1980 in WHO's global Oral Health Data Bank suggest that although gingivitis and calculus deposits are more prevalent and severe in low income nations, there are fewer global differences in the prevalence of severe periodontitis. Gingivitis and calculus deposits can be controlled by personal oral hygiene and professional dental care, so it is to be expected that they are less severe in high – income nations. This geographic profile, in which severe periodontitis is not clearly dependent on the presence of plaque and calculus, is consistent with the compromised host model of periodontitis described earlier.

Prevalence of Gingivitis

- Gingivitis is found in early childhood, is more prevalent and severe in adolescence. The prevalence of gingivitis among scholl children in the United States has been around 40%-60% in various national surveys. In the first national survey of adults that measured gingivitis, conducted in 1960-62 some 85% of men and 79% of women were affected. In a national survey of employed adults in 1985-86, 47% of males and 39% of females ages 18-64 had at least one site that bled on probing. Even with allowance for the differences between the two surveys in measurement techniques and the populations studied, it seems fairly clear that there has been an improvement in gingival health over that period. Gingivitis is likely to have declined over recent years in the United States because of greater attention to oral hygiene as a part of personal grooming.
- In Norwegian professionals and students among whom oral hygiene was excellent, and in Sri lanka tea workers, among whom gingival conditions and oral hygiene were poorer, there was no age related increased in gingivitis. Surveys in other low income countries show that gingivitis, associated with extensive plaque and calculus deposits, is the norm among adults.

Prevalence of Periodontitis

- Data from many parts of the world have shown that the prevalence of generalized, severe periodontitis in range of 5%-15% in almost all populations, regardless of their state of economic development, conditions of oral hygiene or availability of dental care.
- A longitudinal project of major importance was the 15-year study of periodontitis among 480 tea workers in Sri Lanka. A subsequent finding on disease incidence in this group was that gingival recession progressed over time on virtually all surfaces, whereas in comparison group of high income Norwegians it was largely confined to the buccal surfaces. The buccal only recession was thought to come from toothbrush abrasion, whereas the all surfaces recession among the Srilanka was seen as plaque related.

Demographic Risk factors in Periodontitis

Gender and Race or Ethnicity.

- Surveys of periodontal conditions usually show that men have poorer periodontal health than women. Women usually exhibit better oral hygiene than do men, which would explain the differences seen in gingivitis. The fact

that women show less subgingival calculus is likely to contribute toward their better periodontal conditions as measured by CAL and pocket depth. Current knowledge of the pathogenesis of periodontitis, when added to the epidemiologic evidence, indicates that there are no inherent differences between men and women in susceptibility to periodontitis.

- Early epidemiologic studies showed considerable differences between nations.but no consistent associations with race or ethnicity when persons of the same age and oral hygiene status were compared.
- The WHO Global Oral Health Data Bank, which maintains data from many nations collected using the community periodontal Index suggests that race and ethnicity in themselves cannot be considered as demographic risk factors for periodontitis.

Age

- The relationship between age and periodontitis is not always an easy one to understand. Much of the problem dates back to older perception of the disease,in which the interpretation of cross-sectional survey data was generally that severity of the disease increase with advancing age. However, today we do not view periodontitis as a disease of aging. The greater prevalence and severity of CAL in older people in cross-sectional surveys come not from a greater susceptibility in older people but from the cumulative progression of lesions over time.
- The cross-sectional data by NHANES III in United States during 1988-94 show there is a linear relationship between age and the proportion of people with at least one site with 4-mm of 6-mm CAL.
- To summarize the data on age and periodontitis: cross-sectional surveys data invariably show, on average, a greater extent of CAL among older than among younger persons. The apparent increase of CAL with age is more a life time accumulation of effects than a greater susceptibility in the older years. Limited longitudinal data suggest that CAL increases rapidly with age among the 5%-15% of any population that is susceptible to serious disease and to a lesser extent among the majority that exhibits moderate disease. Those susceptible to serious disease exhibit CAL and bone loss when young, often in the teenage years.

Socioeconomic Status

- Generally, those who are better educated, wealthier, and live in better circumstances enjoy better health status than the less educated and poorer segments of society. Many disease conditions are associated with SES, a complex variable that can subsume a lot of cultural factors. Periodontal disease are among this group and have historically been related to lower SES. The periodontal ill effects of living in deprived circumstances can start early in life.
- Gingivitis and poorer oral hygiene are clearly related to lower SES, but the relationship between periodontitis and SES is less direct.

Genetics

- The original 1997 report, based on data from patients in private practices, found that a specific genotype of the polymorphic interleukin 1 (IL- 1) gene cluster was associated with more severe periodontitis. This relationship could only be demonstrated in nonsmokers, which indicated immediately that the genetic factor was not as strong a risk factor as was smoking. The IL-1 gene

cluster has received a lot of research attention since then. This is appropriate, given that the proinflammatory cytokine IL-1 is a key regulator of the host response to microbial infection, although IL-1 is unlikely to be the only genetic factor involved. IL-1 has been identified as a contributory cause of periodontitis among some patient groups and in an epidemiologic study.

- Although there is little doubt that periodontitis has a genetic component, the strength of that component is still to be determined.
- IL-1 has been described as playing a clear, but not essential role in regulating host response to infection. Further research, including epidemiologic studies of people with and without disease, are necessary before the genetic contribution to the initiation and progression of periodontitis can be specified. Current knowledge tells us that inducing periodontal patients to stop smoking should be a higher priority than genetic testing.

Risk Factors for Periodontitis

Oral Hygiene, Plaque and Microbiota

- The presence of plaque and calculus deposits is found to correlate poorly with severe periodontitis in population studies and the same is true for other measures of plaque quantity. What these studies of populations with poor oral hygiene and little dental treatment suggest is that, although gingivitis and calculus deposition are more severe, the prevalence and severity of periodontitis is not all that different from conditions in develop nations. Even among health professionals in the United States, oral hygiene practices seem unrelated to periodontitis.
- More recently it has become clearer that within the broad spectrum of gram-negative organism found at diseased sites, several putative pathogens are consistenly found. The predominant group includes Actinomyces actinomycetemcomitans, Bacteroides forsythus, Porphyromonas gingivalis, Prevotella intermedia, Fusobacterium nucleatum, Campylobacter rectus, and Treponema denticola.
- Maintaining excellent oral hygiene affects only the plaque and not the host response, one reason why oral hygiene is not always effective in controlling periodontitis.

Local Factor

Nutrition

- Subsequent studies suggest that there may be an association between more extensive gingival bleeding and ascorbic acid deficiency, but whether such a mechanism relates to bone loss or CAL is not known. Nutritional adequacy is of course a precondition for successful treatment of virtually any disease, but there is no evidence to support the use of ascorbic acid, or any other nutrient in the treatment of periodontitis.

Tobacco use

- Smoking was first identified as a risk factor for periodontal disease in an analysis of data from the first National Health and Nutrition Examination Survey (NHANES I) in 1971-74 in the United States, and the evidence has continued to mount since then. Assessments of randomly chosen patient groupings invariably show a higher prevalence of periodontitis among smokers, and healing following treatment is slower in smokers. Slower healing could be

due to the inhibition of growth and attachment of fibroblasts in the periodontal ligament of smokers and in their slower postheraphy reduction of white blood cells and neutrophils.

- In summary, the evidence is clear that smoking is a major risk factor for periodontitis. The first line of treatment for periodontitis should always be to induce patients who smoke to quit smoking.

Psychosocial Stress

- Psychosocial stress seems to be associated with progressive periodontitis, whether assessed in a case-control study, cross-sectionally, or in a longitudinal design.

Periodontitis and Systemic Conditions

Diabetes

- Both type 1 diabetes mellitus (type 1 DM, formerly called insulin-dependent diabetes mellitus) and type 2 DM (formerly called non-insulin dependent diabetes mellitus) are risk factors for periodontitis.
- Younger adult patients with type 1 DM, especially those in whom the disease is of long duration, have more gingivitis and more deep pockets than nondiabetic patients.
- Periodontitis also progress more rapidly in individuals with poorly controlled diabetes, and early age of onset of diabetes also is considered a risk factor for more severe disease.
- Studies of quantitative and qualitative aspects of microflora in individuals with diabetes (both type 1 DM and type 2 DM) reveal no notable differences between diabetics and non diabetics. Other suggested mechanisms by which diabetes may contribute to periodontitis include vascular changes, polymorphonuclear leukocyte dysfunction, abnormal collagen synthesis, cytokine production, and genetic predisposition. Individuals with poorly controlled diabetes have also been shown to have impaired salivary flow. Periodontal treatment should always be a standard feature of health care for diabetic patients.

HIV Infection

- HIV-positive patients showed a more sensitive reaction to plaque than did the HIV-negative patients. This study concluded that immunosuppression, especially in combination with older age, was a risk factor for progression of CAL and that seropositivity, independent of immune status, was a risk factor for gingivitis.
- If HIV infection is really a risk factor for periodontitis, we would expect to see an inverse relation between severity of periodontitis and CD+4 counts. The evidence is mixed, however, except for those in the most severe stages of AIDS.
- Even in an African population with no access to modern antiretroviral drugs, periodontitis among HIV-positive individuals was less prevalent and severe than had been expected. The usual oral manifestations of AIDS (candidiasis, hairy leukoplakia, Kaposi's sarcoma) were less common than expected in a patient group receiving antiretroviral therapy, and HIV-infected children whose disease was under good medical control had no more periodontal disease than did HIV-negative controls.
- However, there are contrary findings as well. HIV-positive patients in North

Carolina hospitals were found to have more severe periodontitis than uninfected persons, and these lesions were related to the degree of immunosupression. A british study found more periodontopathogenic bacteria in HIV-positive patients than ini HIV- negative controls. And greatly increased numbers of mast cells and neutrophils were found in the gingival tissue of HIV-positive patients.

- Our understanding of the relationship between periodontitis and HIV/AIDS demands further research, especially since the success of highly active antiretroviral theraphy has changed the outlook on HIV infection.

Cardiovascular Disorders

- Circumstantial evidence supports an association between periodontitis and cardiovascular conditions. The presence of C-reactive protein is a risk factor for cardiovascular disease, and the protein has been found at higher levels in persons with periodontitis both in clinical studies and in NHANES III. The NHANES III data, interestingly enough, showed C-reactive protein levels also to be high in edentulous people, for reasons unknown.

Osteoporosis

- Osteoporosis is a condition of bone fragility characterized by low bone mass and structural deterioration. It is common old age, especially in postmenopausal women. Osteopenia is a less severe form of bone mineral loss. Because alveolar bone loss is often seen with generalized osteoporosis, the question naturally arises as to how much alveolar bone loss is actually due to osteoporosis rather than to periodontitis.
- Cross-sectional studies, not surprisingly, give conflicting results. A number of studies show an association between periodontitis and low bone mineral density at various locations in the body, however, other cross-sectional studies have shown no association between periodontitis and systemic bone mineral density measured at eight points in the body and no difference in periodontitis levels in postmenopausal women with osteoporosis. A literature review concluded that CAL was greater in osteoporotic women than in non osteoporotic women, although the body of literature available for review was rather sparse.
- More research is clearly needed in this area.

Adverse Pregnancy Outcomes

- A 1996 report concluded that mothers of premature, low-birth-weight infants were about 7.7 times more likely to have periodontitis during pregnancy than mothers of normal weight infants.
- The evidence favors an association between maternal periodontitis and the risk of delivering a preterm low-birth-weight infant. It is too strong enough to indicate that periodontal monitoring and treatment when necessary is a good idea during pregnancy.

Dental fluorosis

- Dental fluorosis is a permanent hypomineralization of enamel that is characterized by greater surface and subsurface porosity than in normal enamel and that results form exposure of the immature tooth to excess fluoride (F) during developmental stages.
- Although sufficiently high F concentrations might affect enamel at all developmental stages, early preeruptive maturation appears to be the time when enamel is most sensitive to the effects of F both in animals and in humans. Elegantly designed human studies have suggested that this critical period for the development of fluorosis in the human maxillary permanent central incisor begins around the age of 22 months and extends for periods of up to several years after for later developing teeth.
- Teeth that mineralized later in life generally show more severe fluorotic disturbances than do those that mineralize earlier. Fluorosis is less common in the primary than in the permanent dentition, although fluorosis of the primary teeth does occur. It is common in the primary dentition in high F areas of the world such as east Africa.
- Because dental fluorosis is a dose response condition, the higher the F intake during the critical period of tooth development, the more severe the fluorosis. The threshold, if indeed there is one, is low : 0.03 – 0.1 mg F/kg body weight has been suggested as the border line zone, at least European children.

Risk factor for dental fluorosis

- Because fluorosis is a disturbance of enamel due to excessive F intake during the developmental period, risk factor are related to the ingestion and absorption of F at the critical period of preeruptive tooth development. Age is a demographic risk factor in that fluorosis can only occur with preeruptive F exposure. There is no evidence for racial or ethnic differences, and socioeconomic status (SES) is a demographic risk factor only to the extent that F exposure from toothpaste. The presence of professionally applied gels and varnishes are risk factors for fluorosis although obviously the protocols for application of these product must be designed to minimize ingestion.
- Drinking fluoridated water is a minor risk factor for fluorosis. It was documented long ago that in the United states, even at around 1.0 parts per million (ppm) F, 7%-16% of children born and reared in areas with fluoridated water exhibit mild of very mild dental fluorosis in the permanent dentition. This degree if prevalence was recorded at a time when drinking water was virtually the only sources of exposure to F, and prevalence has risen relatively more in the nonfluoridated areas since then. Even small changes in F concentrations in drinking water can lead to considerable change in fluorosis prevalence.
- Regardless of the role of supplements in preventing caries there is strong evidence that supplements are risk factor for mild to moderated fluorosis. Case control studies in nonfluoridated areas of new England found that exposure to F supplements during the first 6 years of life, together with higher SES, significantly increased the risk to be extremely high when supplements are used (inappropriately) in fluoridated areas. Other studies have demonstrated

the link between use of supplementation was later confirmed as a risk factor for fluorosis in a comprehensive systematic review. It was this evidence that led the American Dental Association in 1994 to reduce the recommended F supplement dosage for caries prevention in children.

- One study of a Toronto area with fluoridated water found that early use of fluoride toothpaste (before 2 years old) and prolonged use of infant formula produced with fluoridated water were strong risk factors for the later development of fluorosis. Although most of the fluorosis seen in that Toronto study was very mild, later study were able to confirm a clear risk of fluorosis with early use of F toothpaste. The risk from early use of F toothpaste, however, was usually not as high as that seen with F supplements.
- Use of infant formula has been recognized as a risk factor for fluorosis, both because of its own F content and especially because it may mixed with fluoridated water. Soy-based formulas contain higher F concentrations than do milk-based formula.

Dental caries and fluorosis

- Caries could increase with higher F levels in drinking water either because restorative treatment is sought for fluorosed enamel or because pitted and friable enamel is diagnosed as caries. Although friable enamel can certainly lead to loss of function and require dental restoration, it is not caries. However, it is possible that broken enamel makes a tooth more vulnerable to caries. Whatever the link, severe fluorosis is obviously a condition to be avoided.

Dental fluorosis as a public health problem

- Dental fluorosis can not be classed as a public health problem in the US and other countries where controlled water fluoridation is extensive. It would be mistake, however, to assume that it cannot become so. There is evidence from several parts of the world that people are quite aware of even the milder forms of fluorosis in their teeth. If high F toothpaste become widely marketed, and if the esthetics standards of the public regarding fluorosis become more stringent, then dental and public health authorities could be faced with demands to do something about it. This could include restricting exposure to F, so that possibility is best avoided by prudent use of F now.

Fissure Sealant

This chapter disccusses the use of sealants in caries prevention, examines the issue of their cost effectiveness, and makes recommendations for their use

Historical Development

- In the late 1960s the 'Bis-GMA' formulation was developed and proved successful in a feasibility trial. The American Dental Association (ADA) issued provisional acceptance of the first bis-GMA material, Nuva-seal, in 1972 and full acceptance in 1976. Currently the most widely used sealant materials are either Bis-GMA resin or urethane based. There is also considerable interest in the potential use of glass ionomer-based materials and fluoride-containing varnishes as sealant materials, but the research literature to date shows their retention to be inferior to that of the conventional sealant materials.

Rationale for Sealants

- It has been recognized for years that fissured occlusal surfaces are the most vulnerable to caries. With the continuing caries decline among children, caries is more a disease of the fissured surfaces as the rate of interproximal caries development continues to decline faster than the overall rate of caries experience. Occlusal surfaces are also those least protected by fluorides, so the case for sealant application as a complementary procedure to fluoride use is even stronger. As of the early 1990s, at least 83% of all decayed or filled olds were in pit and fissure surfaces.

Sealant Efficacy

- The first clinical sealant studies in the 1960s yielded spectacular results, with caries reductions of 99% reported. These initial studies, however carefully selected both the patients and the teeth to be sealed. By the end of the 1970s, there was clear evidence from numerous clinical trials in different populations that sealant were highly efficacious when applied correctly.
- Well-controlled clinical trials have shown good results after 5 years, 7 years, and 10 years and 10-year and 15-year retrospective reports also showed encouraging results. The favorable evidence has led the ADA to strongly support the appropriate use of sealants in general practice.
- As has been pointed out in a study of the use of sealants in a Medicaid program, the children who actually received sealants tended to be at lower risk, that is , they were more likely to have been classified by the study examiners as not needing sealants.
- Findings from the earlier clinical studies of sealants that have been supported by later research include the following :
    - Sealant is generally retained better on mandibular than on maxillary molars. This is attributed to better accessibility and more favorable tooth morphology.
    - Sealants are better retained when placed in older children. This is thought to be due to the ability to achieve better isolation in more completely erupted teeth and the ability of the older child to cooperated in maintaining a dry field.

- Retention seems better on bicuspid than on molars. This too is likely to come from better accesibility, plus the fact that in studies in which children have had bicuspids sealed they were obviously older than children who had only their first molars available for sealing.
- Retention of sealant is synonymous with freedom from caries. An early concern was how the caries status of a tooth could be judge beneath intact sealant, but subsequent clinical research have shown that caries does not progress beneath intact sealant.
- Loss of sealant is greatest in the first 6 months after application. The sealant is probably lost very early in that period, however, because the data suggest that the rapidly lost sealants are those that never properly adhered in the first place. The most likely reason for this kind of failure is moisture contamination. A properly placed sealant will gradually wear down after a period of years, but protection from caries seems to remain, perhaps because of the sealant tags. The quickly lost sealant almost certainly has no tags, so the tooth concerned becomes vulnerable again.

Sealant Can Be Safely Placed Over Incipient Caries

- A conclusion of the national institutes of health consensus panel in 1983 was that evidence supported the use of sealants to arrest the progress of incipient lesions. Given that sealants occluded the fissures, it was logical to question whether caries could progress beneath a sealant. The answer, after a number of studies, is now clear. Given that sealants occluded the fissures, it was logical to question whether caries could progress beneath a sealant. The answer after a number of studies, is now clear. When a sealant is placed over an incipient caries lesion, meaning a stained fissure in which softness at the base can be detected but in which cavitation has not yet occurred, caries does not progress provided the sealant remains intact. Sealant is retained on the carious teeth just as well as on sound teeth, and neither lesion depth nor microbiologic counts progresses under intact sealant. Reviews of these and other studies have concluded that the evidence is strong that caries active lesions become caries inactive beneath intact sealant. As restorative philosophy continous to evolve toward increasingly conservative cavity preparations, more recent reports confirm that even carious dentin, when isolated under a minimal restoration and sealant, does not progress.

Sealants are of Uncertain Value on Primary Teeth

- Some early research showed poorer retention of sealant on primary tooth enamel, although results were better in some later studies. The different enamel structure of primary teeth was thought to be a possible reason, although moisture contamination may also have been greater with younger children. Further, when caries is a problem in primary molars, the first lesion is often interproximal. Sealants are not effective in these circumstances.

Sealants in Public Programs

- Research has shown that trained auxiliaries can apply sealant just as successfully as can dentists. This is an important finding in public health, for the cost effectiveness of sealant programs virtually depends on deployment of

auxiliaries. It is unfortunate that regulations in some states do not permit auxiliaries to apply sealant.

- One is in New Mexico, which required revision of its dental practice act to permit auxiliaries to apply sealant in the state-administered program. The new-mexico program, which uses mobile teams with portable equipment, found 67% retention of one time sealant applications after 6 years. In sixth graders who had received sealants on their first molars in grades 1,2, or 3 only 5.6 % of those surfaces subsequently become carious, compared with 26.9% of the same surfaces in children who were not in the sealant program.
- In the United States, where children begin school at age 6, grades 1-2 are the best times for sealing first molars and grades 6-7 for sealing second molars.
- Sealant placement is an obvious adjunct to water fluoridation, a comprehensive 1989 review found that sealants were more effective in fluoridated areas than in nonfluoridated areas, although the difference was to slight.
- In new york study, after 2 years the 84 children in grades 2-3 with sealants had an increment only 0.03 DMFS compared with an increment of 0.47 DMFS in the control group. In the 84 children in the sealant-rinse group, there were only 3 new decayed or filled surfaces over the 2 years, 2 of them occlusal, whereas in the 51 controls, there were 24 new decayed or filled surfaces, 15 of them occlusal.

Cost Effectiveness of Sealants

- These observations are also in agreement with the findings of a simulation and sensitivity analysis. In this analysis, if caries increments were high and sealant cost were low, a strategy of sealing all would be most cost effective. On the other hand, if caries levels were low and sealant cost were high, a targeted strategy would be preferable.
- These results suggest strongly that applying sealant only to those teeth with early lesions or to the teeth of children with a history of caries is likely to be much more efficient than a blanket sealing of all potentially at risk teeth.
- The more pertinent issue facing a practitioner is whether to seal or restore a deeply fissured molar with a suspicious fissure. Is it valid to compare these two options on a cost-effectiveness basis ? some commentators have said no, because one is a preventive procedure and the other is restorative. One study directly compared the costs of sealing a caries free molar with the costs of restoring the carious contralateral molar with amalgam. When all maintenance care was taken into consideration, including necessary replacements of both sealant and amalgam, the average cumulative time to place and maintain a sealant over 7 years was 10 minutes 45 seconds. For an amalgam it was 14 minutes 26 seconds. Another small-scale follow up of 12 pairs of children concluded that treatment costs in children who did not receive sealant was 1.64 times greater than the costs in a group who had sealant maintained over the period.

Public and Professional Attitudes Toward Sealants

- Data on the prevalence of sealants in national surveys also show that their use is increasing. Data from the 1986-87 national survey of U.S. schoolchildren ages 5-17 years indicated that 7.6% had one or more dental sealants on permanent teeth. By 1991, however, the results from the first part of the Third National

Health and Nutrition Examination Survey (NHANES III) showed that this proportion had risen to 18.5%. Although this increase is encouraging and is likely to continue, a note of caution is in order because this higher level of sealant application will be most helpful if the sealant are being placed in children who are most likely to develop carious lesions.

- A key criterion is that the fissured surface must be at significant risk for disease. This also has led to the view that sealant placement is appropriate for older children and adults in selected cases.

Diet and Plaque Control

This chapter takes a critical look at the role of dietary approaches to preventing oral disease, at the potential for caries control through the use of sugar substitutes, and at the most appropriate place for oral hygiene in caries control.

Nutrition and oral diseases

What is meant by a cariogenic food?

- Sugars and other fermentable carbohydrate are part of the etiologic chain in dental caries. The phrase other fermentable carbohydrates is used a lot in the literature, and it sounds both broad and vague. What are these other fermentable carbohydrates? The terms refers to the cooked of milled starches in the refined flours used in making cooking, biscuits, sweet rolls, croissants and other processed foods. Their dental significance is that as simple carbohydrates they can be broken down furher by the salivary enzyme amylase while still in the mouth and then metabolized by cariogenic bacteria just as sugars are. For that reason these simple carbohydrates are considered potentially cariogenic. Some evidence suggests that starch sugar mixtures are more cariogenic than sugars alone.
- Strach is branched or unbranched polysaccharide chain of glucose molecules. Large molecule carbohydrates such as those found in potatoes, broccoli other fruits and vegetables and whole grains. These are all carbohydrates that have been viewed as essentially noncariogenic because they break down very little in the oral cavity. These sugars are part of the structure of fruits, vegetables, and milk and as such are called intrinsic sugars. Intrinsic sugars are considered virtually noncariogenic when eaten in moderate amounts. Added sugars, sometimes called extrinsic sugars, are held to be the sugars that are metabolized by cariogenic plaque bacteria and trigger the events that lead to demineralization. Some of the literature in this subject expands the terms sugars to nonmilk extrinsic sugars.
- There is no important difference in cariogenicity between refined sugars and brown sugars. Despite the earlier comment on intrinsic sugars, adherence to a high fruit does not necessarily protect from caries. However as an after school snack, fruits have considerably more nutritional value than the average candy bar.

- The concept of a cariogenic food was too broad to be of practical use in caries control so attempts were made to determine the cariogenic potential food caries in humans under conditions conducive to caries formation.
- The swiss government has been testing the cariogenic potential of snack foods since 1982 and has permitted snack foods there to be labeled Zahnfreundlich (which means 'tooth friendly'and implies nonerosiveness as well) if they do not lower the pH of interdental plaque below 5.7 for up to 30 minutes after consumption. Under this well-accepted program, tests of food products are carried out telemetrically with a plaque electrode. Accepted products are usually confectionery items sweetened with the sugar alcohols xylitol, sorbitol, mannitol or martitol or with lycasin, a hydrogenated starch derivative. Fructosa does not pass the test.

Soft drinks

- Soft drink consumption is associated with caries and that high consumption of soft drinks increases the risk of caries. Therefore caries control calls for modest consumption of soft drinks, but in the current social environment it is difficult to promote that message. One obstacle is America's insatiable thirst for carbonated soft drinks and the nonstop advertising that goes along with it. Even though soft drink consumption has leveled out in the United States over recent years, it average 54.2 gallons per person in 2002. For years all of this sugar consumption was seen as a matter of no concern, but more recently the high consumption of soft drinks has been linked with the global obesity epidemic, an epidemic that is well recognized in the United States. One response to such a problem should be promotion of good nutrition in infancy but instead the trend in recent years has gone the other way, with juices and soft drinks replacing breast milk, formula and cow's milk. This change generally is not beneficial, and too many children are already overweight when they begin school.

Consumption of sugars

- The material known by the lay term sugar is sucrose, adisaccharide that is the most common from of sugar consumed by humans. Sucrose and the other sugars, both monosaccharides and disaccharides, are added to a wide variety of processed foods, labels on supermarket staples like canned soups, salad dressings, and processed meats frequently put sugars high on the list of

ingredients. The ingredients on a label are listed in order of relative proportions. So the higher on the list an ingredient appears, the more of it there is in the product.

- Most monosaccharide consumed in the United States is High-Fructosa Corn Syrup (HFCS), widely used in place of sucrose in processed foods and soft drinks. HFCS consists mostly of fructose, glucose and other oligosaccharides.it is to be so much more cariogenic than other sugars. However, later research has suggested that the differences between sucrose and the various monosaccharides in terms of cariogenic potential are less than originally believed.
- One could speculate whether America's reduced consumption of sucrose has been a factor in the sharp reduction in approximal and smooth surface caries relative to the overall caries decline. This speculation is based on the fact that the production of extracellular polysaccharides in plaque depends on sucrose and that smooth surface caries will only develop with plaque that adheres by means of extracellular polysaccharides.

Noncariogenic Sugar Substitutes

- The most commonly used sugar alcohol in the United States has been sorbitol, which is the standard sweetener in several sugarless chewing gums and over the counter medicines. The advantages of sorbitol over sugars, in terms of cariogenesis is that in small amounts it does not lower the pH of plaque to the point at which enamel demineralization occurs. Sorbitol is considered to have low cariogenicity rather than to be noncariogenic, however, because when larger amounts are counsumed both the acid production in plaque and the number of sorbitol fermenting microorganisms learn to metabolized sorbitol when their sugar supply is restricted, a form of adaption to sorbitol that has also been also demonstrates in animals. Several clinical trials of sorbitol chewing gum, however, have shown that these problems do not occur when consumption levels are low, around two sticks of gum per day. Use of sorbitol gum at this level at least does not promote caries and may help to reverse early demineralization of a lesion.

- The sugar alcohol that has received most research attention is xylitol. Xylitol like other sugar alcohol, is caloric but has been shown to be noncariogenic and to possess the properties of a marketable sweetener.
- Subsequent field trials of xylitol-sweetened gum and confectionery products have continued to give impressive results. Other field studies, in one instance with fluoride added to xylitol gum, have yielded acceptable positive results only slightly clouded by questionable study design and data analysis. In comparisons between sorbitol and xylitol, xylitol has yielded better result, probably because of its antibacterial properties. Maternal consumption of xylitol has also been shown to block the transmission of mutans streptococci from mother to child.
- Xylitol can not be metabolized by cariogenic microorganisms and thus does not reduce the pH of plaque. Further analysis of data from the turku studies, suggest that xylitol may promote remineralization, and there are also reports that xylitol can arrest established dentin caries. This evidence has led to the possibility that xylitol may be more than noncariogenic and may actually be therapeutic or anticariogenic.
- Xylitol has been approved for special dietary use in the United States since 1963 although it remains hard to find in chewing gums and other snack products. Xylitol is much more expensive than sucrose, however, and because it is destroyed by heat it cannot be used in cooked food product.

Cleansing and Protective Food

- One food with reported protective factors is cheese, there is evidence in humans to show that finishing a meal with cheese reduces the acidity of plaque and therefore presummably its cariogenicity. Animal studies, which of course can be more tightly controlled than studies in humans, support this finding.
- Nutritious and fibrous foods are naturally to be recommended for good general health. There is not any evidence to support chewing of carrots, celery, or apples as a means of cleaning plaque from teeth. This form of dietary counseling should not become the centerpiece of dental health education, although dental personnel should always encourage healthy food choices by their patients.

Plaque Control

- Even with all the knowledge gained from modern research, however, the relation between caries incidence and level of oral hygiene can still be confusing.Despite historically mixed research evidence linking oral hygiene was piqued by a series of reports in the 1970s which concluded that caries incidence in children could be virtually eliminated by meticulous plaque removal carried out by trained dental auxiliaries at frequent intervals. Benefits of this protocol probably came from a combination of (1) Plaque control (2)Intensive use of topical fluoride paste and (3) Dental health education and oral hygiene practices at home, although the researchers concluded that most benefit came from the oral hygiene procedures. Caries reductions of 98% were reported over 2 years.
- Studies carried out by Scandinavian researchers with small groups of children continue to relate good oral hygine to low caries experience. Poor oral hygiene is also a clear risk factor for root caries in older people. On the other hand, despite extensive improvements in the caries status of Quebec children between 1977 and 1990, no improvement in oral hygiene could be found and oral hygiene levels were not associated with the progression of white spots to dentinal lesions. Even if a poor level of oral hygiene does promote caries, extensive professional care to clean up a dirty mouth may not be time well spent if the underlying reasons for poor oral hygiene are not addressed.
- The main purpose of regular toothbrushing, in terms of caries prevention, is to introduce fluoride into the mouth regularly via the toothpaste. The plaque-removal effect appears secondary in caries prevention, although it can have primary benefits in controlling gingivitis. Regular toothbrushing with a fluoride toothpaste should be encourage as a regular daily routine for all people , whether susceptible to caries or not.

Fluoridation of Drinking Water

Optimal fluoride concentrations in drinking water

- The US Public health Service recommends that drinking water in the United States be fluoridated to a level between 0,7-1,2 ppm F, depending on climate. This recommended have been found unsuitable for Asian and African regions. Hongkong, for example, reduced its water F levels to 0,5 ppm F by the mid 1990s because of increased fluorosis and an expert committee of the World Health Organization has recommended a range of 0,5 – 1.0 ppm F for all parts of the world.
- The recommended levels for the United states have been in place since 1962 and are due for revision, because much in our lifes styles and exposure to F has change since then. For example :
  - Air – conditioning is widespread and has been a major factor in promoting population growth in hotter parts of the country. Because we live in an increasingly climate controlled environment, people drink more water in hotter parts of the country becomes weaker.
  - There has been a huge increase in consumption of soft drinks and bottled waters. These may or not may not be made with fluoridated water and products from the same plant can be distributed to both fluoridated and nonfluoridated areas.
  - An increase in the prevalence of fluorosis since the time the guidelines were developed indicates that young people are ingested more F than they used to. Increased use of commercial juice and drinks in place of milk and water could be a factor.
- Reducing the disparities, disparities refers to the differences in health status between favored and unfavored groups in our society, usually racial ethnic groups and those different socioeconomic status. Disparities are complex and persistent problem, with economic costs as well as political and moral overtones. Fluoridation has the major social advantage of benefiting children in lower SES areas relatively more than those in higher SES areas.

Caries Reductions in Children

- The many fluoridation studies conducted in different parts of the world have varied in quality of design and operation, but their results have still been remarkably uniform. In Britain, where water fluoridation is not widespread, reduction of around 50% are still found in primary and mixed dentitions. Similar result have been reported in Brazil, in Australia, where comparison were based on school dental service data for groups of 9000 or more children, in New Zealand and in Ireland
- Where fluoridation is widespread, as in the United States, differences in caries experience between children in fluoridated and nonfluoridated communities are now estimated to be more on the order of 18% - 35 %. However, how much of F's benefits is due to water fluoridation, rather than overall F exposure, really cannot be estimated with any precision now for the following reason :
  - The development and almost due universal use of fluoridated toothpaste and other dental products containing F, plus the presence of F in food and beverages processed with fluoridated water, means that there are many different F exposure for the average American
  - The extreme mobility of the US population means that most people have had varied exposured to fluoridated water at different times of life. When this is added to the diffusion effect, it is clear there is no way of finding true control subject in the United States.
  - History of exposure to fluoridation is difficult to document in research studies. There are no biomarkers for lifetime exposure, and interview data can be unreliable.

Caries reductions in adults

- Studies of fluoridation's effect in adulthood began early with McKay's recognition that 45-year old adults benefitted from consuming fluoridated water. Adults born and raised in naturally fluoridated Colorado Springs were found to have 60% lower means DMF scores than their counterparts in nonfluoridated Boulder.Resident of Colorado Springs also had far fewer teeth missing. Similar findings among adults came from aurora,illnois, a city with 1.2 ppm F naturally occurring in its drinking water.

- A later study did use more detailed statistical analysis in evaluating data from lordsburg (3.5 ppm F) and Derming (0.7 ppm F) New Mexico. SES status was higher in Deming. Results still favored the community with the higher F level. After other important variables were controlled for, Deming adults were found to have two more restored teeth per person than did those in Lordsburg.
- Root caries is also less prevalent in fluoridated areas than in nonfluoridated areas. This findings is important, because with increasing tooth retention in an aging population the amount of root caries would otherwise be expected to increase and become a greater treatment problem in the future. It is not yet clear whether F's protective effect against root caries is due to topical action on exposed root surfaces, to incorporation of F into cementum before root exposure in the oral cavity, or to a combination of both.

Prenatal benefits

- Some support for a prenatal exposure benefit came from the Evanston fluoridation study, in which children who were exposed to fluoridation water in utero as well as postnatally were reported to have fewer carious lesions than those who receive it only postnatally. Other authorities, however, did not find this effect. A comprehensive 1981 review concluded that there was probably no benefit in prenatal F exposure, though it was not ruled out completely. If the offspring derives any benefits at all from prenatal F exposure, such benefit would be slight.
- Benefits or lacks of them aside, it is clear that fluoridation is quite safe for the developing fetus. No special precautions are therefore necessary for expectant mothers in fluoridated areas.

Effect on the primary Dentition

- Early fluoridation studies reported caries reductions in the primary dentition of about the same range as was found in the permanent dentition. The primary dentition clearly benefits from exposure to fluoridated drinking water.

Partial exposure to fluoridation

- What happens in those community with water naturally fluoridated at suboptimum levels such as 0.4-0.7 ppm F ? The US and Canada are highly mobile societies. Many people have therefore spent their lives in a fluoridated area and part in a nonfluoridated area. The benefit of a partial exposure to fluoridated water in adulthood has not been documented, but there is evidence that partial exposure in childhood reduces caries experiences in proportion to the length of exposure. In nonfluoridated Coldwater,Michigan, children who had moved to coldwater after some residence in a fluoridated area had lower caries experiences than did those who had lived in coldwater all their lives. A British study demonstrated a 27% reduction in caries incidence over 4 years among children who were 12 years old when fluoridation began in their community, relatively to the evidence in control children in nonfluoridated areas.
- The evidence regarding partial exposure to fluoridated water indicated that a cariostatis benefit will be received that is more or less proportional to the extent of the exposure. Maximum benefits naturally comes with lifetime exposure.

Caries patterns when fluoridation ceases

- The continued decline in caries experience after fluoridation ceases would be expected in communities in which there is regular and frequent exposure to F form toothpaste and other sources, whereas a caries increase would be expected if drinking water were the only F source. These findings emphasize yet again that regular exposure to low concentration if is what leads to reduce caries experience,not necessarily exposure to any one F vehicle. Water fluoridation is the most efficient way to bring F to a community, but other exposure methods work as well.

Saving in treatment costs

- In the Newburgh Kingston study, initial dental care for 6 years old children cost 58 % less in fluoridated Newburgh than ini nonfluoridated Kingston.
- Children with an obviously low caries attack rate in a fluoridated area do not need to visit the dentist as often as they used to, and they require bitewing radiographs less frequently. Data have also shown that F gel application in an

insured population, most of whom lived in a fluoridated area, bore no relation to caries experience. If dentist continue to see such children twice a year and apply the full battery of diagnostic and preventive services each time, the substantial savings that can be realized in the cost of restorative treatment will be drastically reduced.

Fluoride : Human Health and Caries Prevention

This chapter deals with the issues of how fluoride`s caries inhibitory potential was first discovered, how the human body physiologically deals with fluoride when the material is ingested, how fluoride affects our health and how it works to prevent caries.

Environmental Fluoride

- Fluoride is one of the most reactive elements and therefore is never found naturally in it's elemental form. The F ion, however, is abundant in nature and occurs almost universally in soils and waters in varying, but generally low, concentrations. Seawater contains 1.2 – 1.4 ppm F. Fresh surface water generally has very low concentrations, 0.2 ppm F or less, whereas concentration in deep well water in Arizona and concentrations of over 40 ppm in boreholes in Kenya.

Sources and Amounts of Fluoride Intake

- Human absorb F from air, food and water. F intake from air is usually negligible, around 0.04 mg F/day. Exceptions can occur around some industrial plants that work with F rich material, such as alumunium smelters with inadequate safeguards to prevent the escape of F-containing compounds.
- For most people, water and other beverages provide some 75% of F intake, whether or not the drinking water is fluoridated. This may occur because many soft drinks and fruit juices are processed in cities with fluoridated water, or it may reflect variable F content of the ingredients. One brand of grape juice in North Carolina, for example, was found to contain more than 1.6 ppm F, even in soft drinks of the same brand, F levels can vary considerably due to production at different sites.

Fluoride Physiology

- Although the use of F is a contribution to the public's health of which dentistry can be proud, F compounds must be handled responsibly and with respect. Everyone in dentistry should understand how the human body handles ingested F so that the material can be used safely and efficiently.

Absorption, retention and excretion

- Ingested F is absorbed mainly from the upper gastrointestinal tract, and some 95% of ingested F is absorbed. Absorbed F is transported in the plasma and is either excreted or deposited in the calcified tissues. Most absorbed F is excreted in the urine, a single ingestion of 5 mg F by an adult is absorbed and cleared from the blood in 8-9 hours.
- F balance is the net result from the accumulated effects of F ingestion, degree of F deposition in bones and teeth, mobilization rate of F from bone and efficiency of the kidneys in clearing absorbed F.

Optimal Fluoride Intake

- Frank McClure, a biochemist with the US public health service, estimated in 1943 that the average daily diet contained 1.0 – 1.5 mg F, or about 0.05 mg F/kg body weight per day in children up to 12 years of age. McClure's estimate somehow came to be interpreted as the lower limit of the range of optimal F intake.
- The National Research Council, the body that establishes recommended dietary allowances for the United States, classified F as a 'beneficial element for humans' because of its positive impact on dental health. The council at one time

considered F an essential nutrient, but it backed away from that position because an essential role for F in human growth studies could not be confirmed and because the physiologic mechanism by which F would influence growth was unknown. Available evidence did not justify classifying F as an essential element by accepted standards. Nutritional requirements became recorded as dietary reference intakes in the late 1990s, with an adequate intake level for F set at 0.01 mg/day for children 0-6 months. For all ages above 6 months the adequate intake was set at 0.05 mg F/kg/day, so the absolute intake amount increases with increasing weight to a maximum for adults age 19 or older of 4 mg/day for males and 3 mg/day for females. It was not clear on what these intake levels were based.

- The discussions about optimal intake are vague about what this intake is optimal for. The implication is that this degree of ingestion is optimal for caries resistance, but as will be described later in this chapter, ingested F plays only a minor role in caries control relative to intra oral F. It is also worth nothing that McClure's 1943 comment was observational, although it somehow was turned into a recommendation over time. Empirical evidence suggests that F intake of 0.05 – 0.07 mg/F/kg/day in childhood is a broad upper limit if unesthetic fluorosis is to be avoided. There is no evidence to link this range of F ingestion with caries inhibition, so we suggest that the term optimal intake be dropped from common usage.

Fluoride and Human Health

Early Studies

- The first study relating bone fracture experience to the F concentration in home water supplies (a subject revisited in the 1990s) concluded that there was no relationship.
- Higher F intakes were likely in communities such as Bartlett, texas, however, where community water carried about 8 ppm F. A long-term study of the residents of barlett, conducted by a U.S. public Health Service team, began in 1943. Apart from severe dental fluorosis, the study found no adverse effects of long-term ingestion of this high F water, although postmortem bone F concentrations were high. Numerous animal studies in the early years of water fluoridation supported the results from studies in human population.
- Although not every possible hypothesis regarding F and human health was tested prior to initiation of controlled fluoridation, there was sufficient research evidence to provide reasonable assurance that controlled fluoridation, with up to 1.2 ppm F in the drinking water, could be instituted in North America without creating any public health hazard.

Mortality

- For the United States as a whole, no differences could be found in 1949-50 death rates between 32 cities with 0.7 ppm F or more and 32 randomly selected nearby cities with 0.25 ppm F or less in the drinking water. Mortality rates were similar for cancer, heart disease, intracranial lesions, nephritis, and cirrhosis of the liver. No differences between fluoridated and nonfluoridated communities were found.

Cancer

- The 1979 mortality study was conducted as a response to testimony before a congressional committee in 1975 by Burk and Yiamouyiannis that fluoridation led to an increase in cancer deaths. But a number of independent analyses of

the same data were conducted in both Britain and the United States and employed more detailed age-sex-race adjustments. None could find a link between cancer incidence and consumption of fluoridated water.

- In response, the assistant secretary for health incorporated the NTP (National Toxicology Program) findings into a broader review of F and the environment, conducted by a special committee appointed by the U.S. Public Health Service. This committee reached the following conclusion on cancer risk :
  Optimal fluoridation of drinking water does not pose a detectable cancer risk to humans as evidenced by extensive human epidemiological data available to date, including the new studies prepared for this report. While the presence of fluoride in sources other than drinking water reduces the ability to discriminate between exposure in fluoridated as compared to non-fluoridated communities, no trends in cancer risk, including the risk of osteosarcoma were attributed to the introduction of fluoride into drinking water in these new studies. During two time periods, 1973 -1980 and 1981 – 1987, there was an unexplained increase of osteosarcoma in males under age 20. the reason for this increase remains to be clarified, but an extensive analysis reveals that it is unrelated to the introduction and duration of fluoridation.

Down Syndrome

- A claim that water fluoridation caused an increase in the congenital malformation known as Down Syndrome was put forward in a series of papers in the mid-1950s. the studies claimed to show from birth records in Wisconsin and Illinois that the incidence of Down Syndrome was higher in fluoridated than in nonfluoridated areas, but there were errors was the assumption that the city of birth was the place of residence of the mother, which is clearly not the case for hospitals serving a large rural population. More rigorous independent studies in the United States and Britain have subsequently failed to show any correlation between fluoridation and Down Syndrome. A systematic review in 2001 reached the same conclusion.

Bone Density, Fracture Experience, and Osteoporosis

- Bone fragility conditions have been treated for years with high doses for F combined with calcium, estrogen, and vitamin D. Controlled clinical trials have shown that high doses of F (30-60 mg/day), administered under medical supervision, can increase vertebral bone mass and reduce the vertebral fracture rate. These favorable changes do not come without problems, however, for the new bone can be imperfectly mineralized, and a good proportion of patients do not respond to treatment. Treatment also seems ineffective in preventing further fractures in patients who already have a fracture at first presentation. The main concern is that the positive effects seen in the vertebral column, which is mostly cancellous bone, are not seen in the appendicular skeleton, which is mostly cortical bone. Indeed, fracture rates in the appendicular skeleton have actually been shown to increase with intensive treatment. Although an international conference in 1988 recommended F treatment for vertebral crush syndrome, high dose F treatment (up to 80 mgF/day) for bone fragility conditions is no longer recommended in the United States, nor is F theraphy seen as a measure that can prevent fractures resulting from osteoporosis.
- In more recent times, a series of ecologic studies to assess the risk of bone fracture in those drinking fluoridated water have produced mixed results:

Decreased risk, no association,, and increased risk. Given the lack of precision that is part of ecologic studies, it is not surprising that this body of research does not yield clear conclusions for either increased risk or a protective effect of F. Extensive reviews of the literature have also reached the conclusion that no relationship can be discerned between bone fracture experience and exposure to water with 1 ppm F.

Fluoride Toxicity

- There is a world of difference (literally) between a single intake of 5.0 g F and constant intake of 1-3 mg F daily. F is thus like many other nutrients: beneficial in small amounts, toxic in high amounts.
- Information on F toxicity levels cannot, of course, be taken from controlled studies with humans. Available data come from a mix of case studies of various kinds and research on workers in certain industrial processes. Some workers had been employed as long as 31 years. Under these conditions a number of toxic effects were observed, principally gastric complaints and osteosclerosis.
- Ingestion of a single dose of 5-10 g of NaF by an adult male (32 – 64 mg F/kg body weight) results in a rather unpleasant death in 2 – 4 hours if first aid is not rendered immediately. From that lower limit of 32 mg F/kg body weight, the estimated equivalent dose for a 10 kg child (12 – 18 months old) is 320 mgF. Crippling skeletal fluorosis can eventually occur in an adult with daily ingestion of 10 – 25 mg F over a period of 10-20 years.
- If an individual is known or suspected to have taken a potentially toxic amount of F, first aid is to induce vomiting as quickly as possible or to have the person ingest a material to bind F, milk is usually the most readily available. The American Dental Association recommends, as a safety precaution.

Fluoride and Caries Control : Mechanism of Action

- F works best to prevent caries when a constant, low level of F is maintained in the oral cavity. Its most important caries-inhibitory action is posteruptive and takes place at the plaque enamel interface.
- It has long been held that preeruptive exposure to F inhibits caries to some degree. In this preeruptive model, F said to act by becoming incorporated into the developing enamel hydroxylapatite crystal and thus reducing enamel solubility. It has been argued that preeruptive benefits are especially important for reducing pit and fissure lesions. This caries prevention action by F was assumed for many years to be F's primary effect, but the actual supportive evidence is largely associative. Any preeruptive effect on caries inhibition is likely to be minor, the evidence for posteruptive F action is much stronger.

Three Principal Mechanisms by Which Fluoride Inhibits the Development of Dental Caries

Posteruptive

1. Promotion of remineralization and inhibition of demineralization of early carious lesions.
2. Inhibition of glycolysis, the process by which cariogenic bacteria metabolize fermentable carbohydrates.

Preeruptive

3. Some reduction in enamel solubility in acid by preeruptive incorporation of fluoride into the hydroxyapatite crystal.

Fluoride and plaque

- Plaque contains 5-10 mgF/kg wet weight in low –F areas and some 10-20 mg

F/kg wet weight in fluoridated areas. The bound F can be released in response to lowered pH, and F is taken up more readily by demineralized enamel than by sound enamel. The availability of plaque F to respond to the acid challenge leads to the gradual establishment of a well-crystallized and more acid-resistant apatite in the enamel surface during demin-remin. This means that F can be incorporated into the enamel crystal, but more through cycles of demin-remin than through preeruptive absorption.

- F in plaque also inhibits glycolysis, the process through which fermentable carbohydrate is metabolized by cariogenic bacteria to produce acid. F from drinking water and toothpaste concentrates in plaque, where its concentration is governed by the concentration of plaque calcium. Plaque contains higher levels of F than does saliva.
- In addition to these mechanisms, high-concentration F gels may have a specific bactericidal action on cariogenic bacteria in the plaque. These gels also leave temporary layer of a material resembling CaF2 on the enamel surface. At lower concentrations, streptococcus mutans has been shown under laboratory conditions to become less acidogenic through adaptation to an environment in which F is constantly present. It is not yet known whether this ecologic adaptation reduces the cariogenicity of acidogenic bacteria in humans.

Fluoride and Enamel

- In the early days of F research, it was assumed that F's inhibition of caries depended on its preeruptive incorporation into developing dental enamel, which thus reduced enamel solubility in demineralizing acids. In most of the early fluoridation studies, greater caries reductions were found in children who were born after fluoridation began than in those for whom fluoridation began after birth. But it was also clear that caries inhibition occurred in teeth that had already erupted when fluoridation began and in first molars that were erupting when fluoridation began.
- The converse also holds, it has been observed that higher concentrations of enamel F do not necessarily mean than caries will not occur.

Fluoride and Saliva

- Resting salivary F concentrations are low, although they are some three times higher in fluoridated than in nonfluoridated areas. In a fluoridated area, salivary F levels averaged 0.016 ppm, whereas they were 0.006 ppm in a nonfluoridated area. Fluctuation of salivary F levels is normal, and after tooth brushing with an F solution, salivary F levels can rise 100-1000 fold. This level rapidly drops back to normal, and the saliva is likely to be an important source of plaque F during this time. The role of saliva in caries inhibition is still not well defined.

Effective Use of Fluoride

- As stated earlier, the evidence shows that the most effective community wide use of F is in frequent, low concentration intraoral exposures such as in drinking water or toothpaste. Less frequent application of high concentration gels has its place in the care of highly caries-susceptible patients.

Restricting the Use of Tobacco

- Tobacco use is a major risk factor for many disease and it is the leading cause of preventable mortality. The bare statistics are brutal, more than 430.000 deaths occur each year attributable to tobacco use in the United States, and some 3000 children and adolescents become new smokers every day. More than 10 million Americans lost their lives prematurely to tobacco-caused diseases during the twentieth century.
- Regard to oral effects, tobacco use of all kinds is a major risk factor for oral cancer, it is also a major risk factor for periodontitis.
- Political action to reduce exposure to tobacco is not easy because the tobacco industry is formidable opponent. Likewise, including patients to change establish tobacco addictions are powerful and there are usually strong social or psychological reasons why tobacco habit was adopted in the first place. However, as described here, programs are in place that can help.

Prevalence of Tobacco Use

- Prevalence of cigarette smoking in the United States is now around 23 % among adults and has remained around that figure for some years.
- Heavy marketing of ST (smokeless tobacco / spit tobacco) products, principally targeted to adolescent and young adult males. The concerns about ST's appeal to youth seem well founded, 1995 national survey data revealed that 11.4 % of high school students had used ST within the previous 30 days, 19.7 % of males and 2.4 % of females. The 1991 national survey found that 8.2 % of males ages 18-24 years were regular ST users, the high proportion of any age-group. Even if there is some overreporting, presumably by individuals who wish to appear more 'macho', these figures are high. Usage of ST is highest in the South,in rural areas, and declines with increasing education.
- Use of ST is extensive in the military and is particularly heavy among Native Americans, a study conducted in seven western states found that 56 % of Native Americans in the Ninth and tenth grades reported that they were regular users, as were 28.1 % of sixth-graders.
- In one study of Navajo adolescents, over 25 % of ST users were found to have leukoplakia, compared to only 4% of nonusers. The duration and frequency of use were highly significant risk factors for leukoplakia.
- Prevalence of ST use is also widespread among highly visible professional baseball players, surveys carried out with major and minor league teams found that 39%-46% of players were regular users. Another study of baseball players in 1988 found that ST users had 60 times the risk of developing leukoplakia compared with nonusers.

Pathologic Effects of Smokeless Tobacco

- ST is sold in several forms. The main concern is with snuff, a powdered tobacco product, which is used by placing a 'dip' between the cheek and gum. Dry snuff contains high concentrations of N-nitrosamines, evidence is strong that compounds in this group are carcinogens, especially for oral cancers.
- A consensus panel of the National Institutes of Health found strong evidence that use of snuff causes oral cancers. Nicotine is absorbed from ST in amounts similar to those absorbed from cigarette smoke, which makes ST a potential

risk factor for the same disease that result from smoking. That could be why ST users face a relative risk of 2.1 for cardiovascular disease compared to nonusers. The relative risk for smokers compared to nonsmokers in the same study was 3.2

Restricting Smokeless Tobacco Use

- Voluntarily breaking the ST habit, given that nicotine addiction is involved, seems to be no easier than breaking the cigarette habit. Of 25 adolescent habitual ST users who participated in an intensive program of ST cessation, only 4 had remained successful in quitting 3 months after the program. On the other hand, in another intervention study the number of young male ST users who quit was 50% above the normal rate when participants viewed a 9 minute videotape, were given a self-help manual, and received an explanation of the risks and 'unequivocal' advice to quit.With this mixed evidence on quitting, strategy should be aimed at preventing young people from starting to use ST, although it is obvious that health education programs need to go well beyond the admonition to 'just say no' if they are to be successful.

What Dental Professionals Can Do

Dental professionals obviously have a potentially major role to play in educating patients about the hazards of ST use and in helping patients who are already addicted to quit.

Conclusions and Recommendations on Ways of Health Professionals Can Help Patients Stop Smoking

1. Tobacco dependence is a chronic condition that often requires repeated intervention. However, effective treatments exist that can produce long-term or even permanent abstinence.
2. Because effective tobacco dependence treatments are available, every patient who uses tobacco should be offered at least one of these treatments :
   - Patients willing to try to quit tobacco use should be provided with treatments identified as effective in this guideline.
   - Patients unwilling to try to quit tobacco use should be provided with a brief intervention designed to increase their motivation to quit.
3. It is essential that clinicians and health care delivery systems (including administrators, insurers, and purchasers) institutionalize the consistent identification, documentation, and treatment of every tobacco user seen in a health care setting.
4. Brief tobacco dependence treatment is effective and every patient who uses tobacco should be offered at least brief treatment
5. There is strong dose-response relation between the intensity of tobacco dependence counseling and its effectiveness. Treatments involving person-to-person contact are consistently effective, and their effectiveness increases with treatment intensity (minutes of contact)

Use of these sources of information should be part of the routines of every dental office, for tobacco use cessation must be seen as the first treatment priority for almost any oral disease. After all, tobacco use is a serious matter, in fact, it is a matter of life and death.