Risk assessment and epidemiology of dental caries: review of the literature

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Abstract

The epidemiology of dental caries in the pediatric population demonstrates that caries is no longer pandemic in the US population. The incidence is confined to a subset of the total population of our children. The disease is also increasingly isolated to specific teeth and tooth morphology types in both the pediatric and the mixed dentitions, with pits and fissures being the predominate diseased sites. This sequestration of the disease into specific populations, individuals and tooth sites mandates a risk assessment strategy. In the past, universal preventive strategies were appropriate because of the extensive penetration of caries in the population. Our health care system does not have adequate resources to treat the entire population when a substantial portion of the population is not at risk for this disease process. Validated risk assessment strategies may prove adjunctive for the practicing dentist. Certainly knowledge of the known risk factors will assist the practitioner in performing risk assessment within their patient populations. This paper presents a review of the known risk factors for dental caries in child and adolescent populations.(*Pediatr Dent*. 2002;24:377-385)

KEYWORDS: DENTAL CARIES, RISK ASSESSMENT, EPIDEMIOLOGY, PEDIATRIC DENTISTRY, LITERATURE REVIEW

According to the recently released Surgeon General's report, dental caries continues to be the most common infectious disease of childhood.[1] Perhaps nothing is more important regarding our current knowledge of this infectious disease process than the knowledge that the disease is no longer pandemic, but, rather, endemic in specific sectors and individuals within our populations. Overall, there has been: (1) a decline in prevalence and severity of caries in children; (2) an increasingly skewed distribution, with most disease now found in a smaller number of children; and (3) a concentration of caries in pit and fissure lesions.[2]

In cases where disease is not uniformly distributed in a population, and there are effective interventions, risk assessment can play a significant role in the treatment of infectious diseases like dental caries. Rather than expending resources on an entire population, many of whom are not at risk for a disease, targeting preventive and interceptive strategies to at-risk populations is a sound public health and private practice strategy. This paper presents our current knowledge of risk assessment and the epidemiology of dental caries focusing on pediatric and adolescent populations.

Background

Dental caries (tooth decay) has been a highly prevalent and costly disease in the United States and the world. As recently as 60 years ago, this disease was a significant threat to the majority of the population. Conscription for World War II gave the United States an observation point of the dental status of our young men. What the Armed Services found was appalling. They had difficulty fielding young men from our population with 6 opposing teeth as a minimum requirement for induction. (Note: Although women participated in the uniformed services in WWII, they were volunteers and not conscripts.) Partially as a result of that experience, the United States Congress saw fit to fund the formation of the National Institute of Dental Research (NIDR).[3]

Today, tooth decay is increasing in incidence in the elderly in the United States and elsewhere as aging populations retain more of their dentition, making more tooth surfaces available for the disease processes. It is also increasing in incidence across all ages in many developing countries, including China and India. Tremendous progress in child populations have been made in this country. Yet, the recent

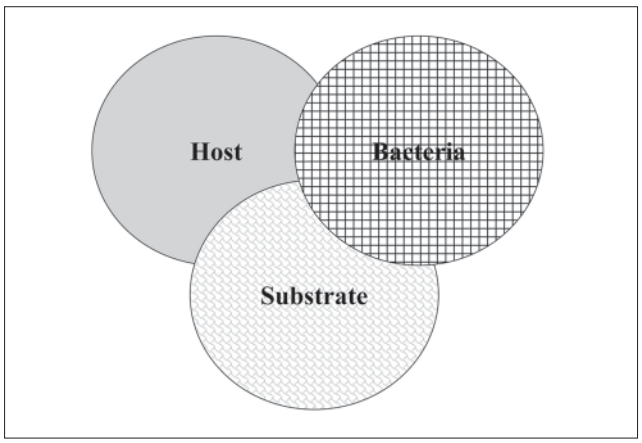

Fig 1. Keyes diagram of dental caries

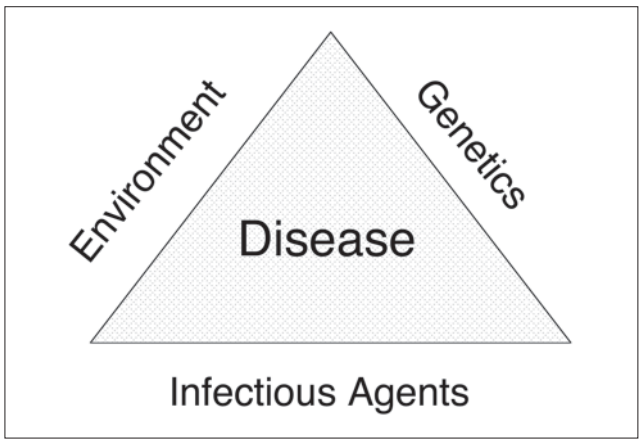

Fig 2. Non-exclusive contributory disease model

United States Surgeon General's report shows that 45% of children ages 5 to 17 have cariously affected teeth, and the problem is particularly severe among children in specific populations.[1] Total spending on dental services in the United States will be over $65 billion this year, with arguably about half of this a direct or indirect result of dental caries.[4]

Pathobiology

Cavities are the clinical manifestation of a chronic bacterial infection dependent on a limited number of species of cariogenic bacteria, susceptible individuals[5] and specific dietary patterns (such as sugar ingestion). This interrelationship of factors was first diagramed Paul Keyes.[6] The Keyes diagram inherently contains the dimension of time, in that an infection needs to be active over time to exert influence.

Streptococcus mutans has been shown to be *the* major cariogenic bacterium.[7] The bacterial cells of *S mutans* and other organisms colonize the surfaces of the teeth and form a complex biofilm commonly called "dental plaque." When provided a suitable metabolic substrate, this infection produces organic acids (primarily lactic acid) that are capable of dissolving the mineral calcium from the crystalline enamel matrix. Continued and repeated dissolution leads to frank cavitation and the subsequent need for repair.[8]

Risk assessment

Gaining clarity on what is meant by "risk" is an important part of gaining consensus. In everyday language, the word "risk" connotes the probability that some adverse event will occur in the future. In the world of health services, "risk" may be understood as the probability that a particular outcome will occur due to the presence of specific risk factors or after exposure to a particular action or event.[9] Identifying "risk" is more difficult in chronic diseases that are caused by multiple factors and events over a longer period of time in individuals in the population. In these cases, a "risk factor" is not just an element or action that is statistically related to some outcome.

The more important question is whether the factor is "causal" or merely "associated" with the outcome. A factor is considered causal if it satisfies various conditions.[10] Among others, these include: biological plausibility; similar distribution of the factor in the population as the outcome; the factor must temporally precede the outcome; and as the level of the factor changes (amount or length of time), the incidence of disease also varies. In chronic diseases, their multifactorial nature means that, while a factor may be necessary to produce the outcome, it is not "sufficient." "Associations," on the other hand, may be seen as 2 events occurring in a relationship where the first of temporal events is not necessary for the subsequent outcome.

In the case of dental caries, the profession knows that bacteria capable of producing acids as a metabolic byproduct are necessary to produce the clinical manifestations of the disease. However, they are not in and of themselves "sufficient" to produce a cavitated tooth. Also needed is a susceptible host and a metabolic substrate on which these bacteria can act over time. Therefore, for the purposes of this presentation, the working definition proposed by Burt[9] will be used.

"Risk factor is an environmental, behavioral or biologic factor confirmed by temporal sequence, usually in longitudinal studies, which, if present, directly increases the probability of a disease occurring. If absent or removed, it reduces the probability. Risk factors are part of the causal chain, or they expose the host to the causal chain. Once disease occurs, removal of the risk factor may not result in a cure."[9]

This definition fits nicely with a previous look at the Keyes diagram. The Keyes diagram is a specific application of a general model of disease termed the "*nonexclusive contributory disease model*." This model, seen in Figure 2, has 3 elements: "environment," "genetic" and "infectious agent."

This paper will use the more general "*nonexclusive contributory disease model*" as the basis for risk assessment for dental caries because it offers better opportunities to examine this multifactorial disease. By its very nature, this disease model states that any specific element may contribute to, but does not necessarily "cause," the specific outcome of a cavity. This fits our current understanding of this infectious disease process and will help provide a way to engage the risk-assessment process.

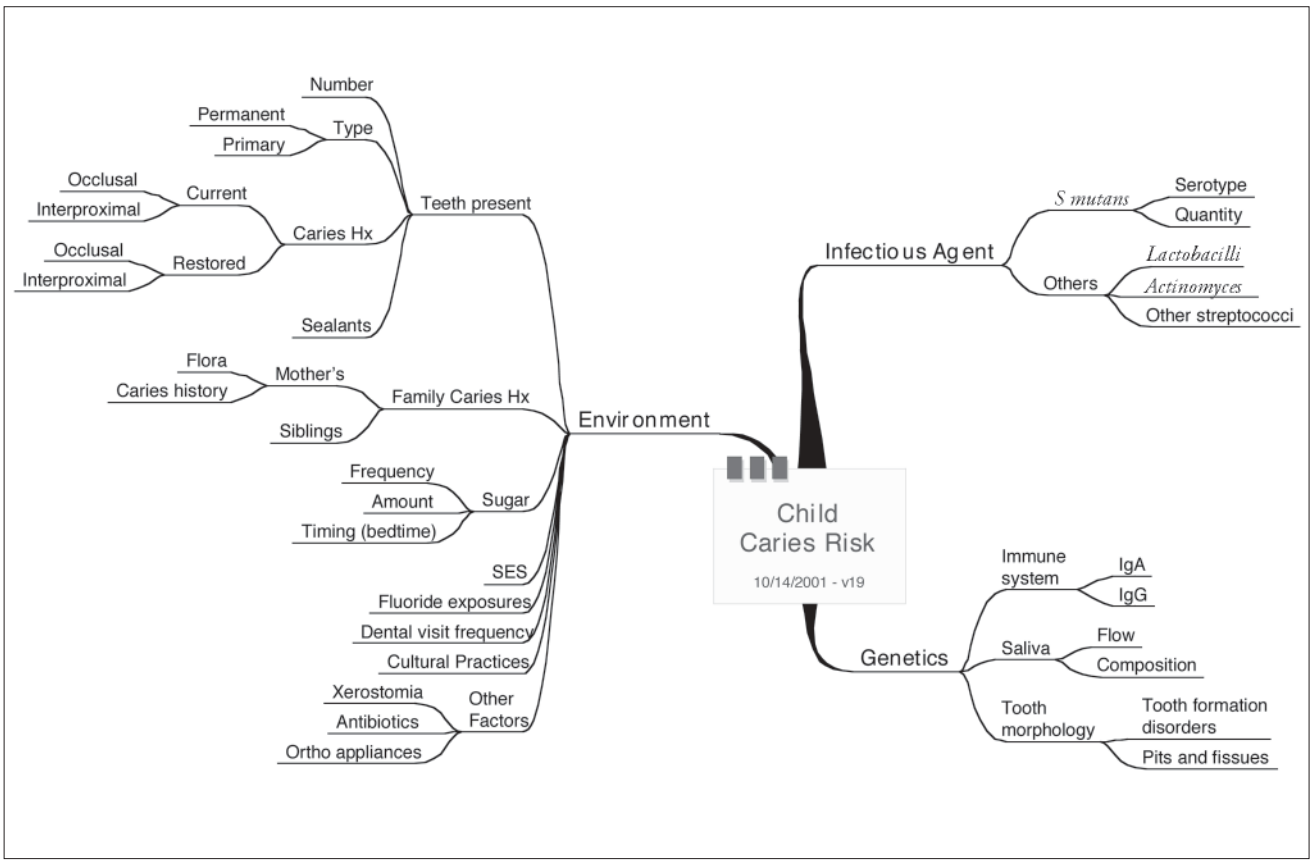

Fig 3. Diagrammatic view of caries risk factors in children using the "non-exclusive" contributory disease model classifications. The 3 primary factors form the primary arms of the diagram. Extensions of the primary arms are subcategories of risk.

Figure 3 is not an exhaustive picture of caries risk nor are all the elements covered in this paper due to the limitation of time and space. Individuals may also contest the categories in which the author has placed various putative risk factors. In spite of these limitations, the picture does facilitate the discussion of risk assessment.

Infectious agents

Mutans streptococci are infectious organisms that colonize the teeth. They help form an intraoral biofilm commonly referred to as "dental plaque" through their ability to adhere to tooth structure by laying down specific glucans, thereby creating a highly "sticky" bacterial environment.[11] Figure 4 shows the spatial distribution of immuno-florescently labeled *S mutans* in human biofilm (plaque) using confocal microscopy.

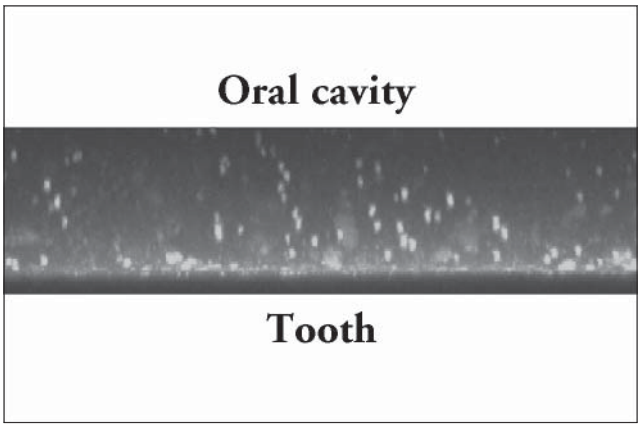

Fig 4. Fluorescent labeled *S mutans* as distributed in a human plaque biofilm. Courtesy of Dr. Wenyuan Shi, UCLA, School of Dentistry.

We know from previous studies that levels of *S mutans* above 500,000 colony-forming units per milliliter (cfu/ml) of saliva are associated with higher levels of smooth surface caries,[12,13] while the presence of lower numbers of *S mutans* in saliva or their presence in fissure systems are predictive of fissure caries.[14]

Generally, mothers transmit the *S mutans* infection to their children.[15-17] Knowing of this vertical transmission route in risk assessment suggests that, in the case of infants, it may be in the clinician's and child patient's best interest to sample the mother for the presence and severity of a *S mutans* infection. The *S mutans* bacteria group competes favorably for a niche in the ecosystem when it does not face a mature biofilm.[18] With the proper nutrients, *S mutans* forms glucan and levan polymers that are quite adhesive, providing for high selection of the organism to uncolonized tooth surfaces. This mitigates for early colonization by the organisms in pits and fissures as well as on smooth surfaces as teeth erupt.

Other infectious organisms may also contribute to dental caries, although none have demonstrated the same degree of risk for initiation of the caries process as that exhibited

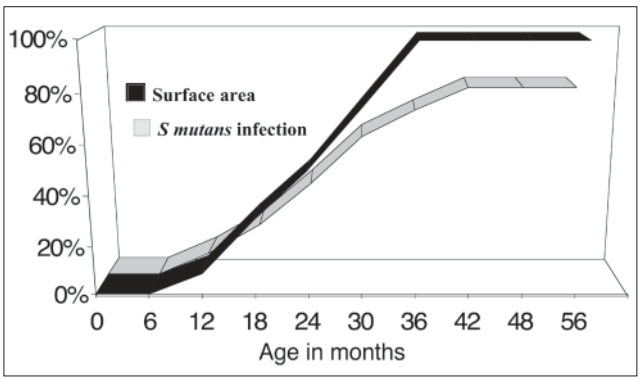

Fig 5. Cumulative surface area of the primary dentition and acquisition of *S mutans* over time

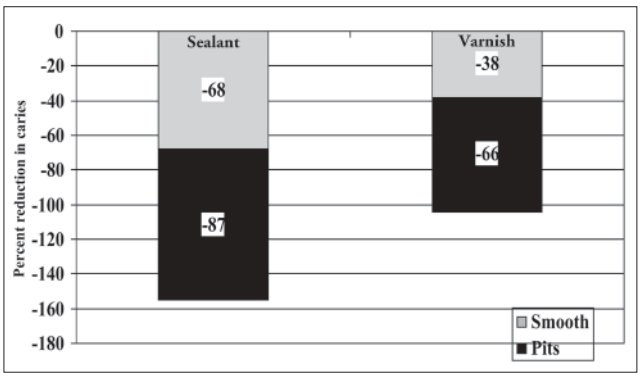

Fig 6. Reduction in caries as a result of 2 preventive schemes: sealants or fluoride varnish[31]

by *S mutans*. The next most studied potentially causative bacteria are *Lactobacilli*. In general, the results of *Lactobacillus* studies indicate that these organisms do not participate in the original colonization of the teeth and are more associated with carbohydrate consumption[19] and the progression of the disease process.[20] Other potential caries pathogens organisms are not covered in this presentation since they are generally associated with dental caries in different age groups.

Genetics

With the advent of the development of the tools to examine the genetics of individuals and the aggregation of those data into population studies, there is increasing evidence that there are genetic risk factor relationships between dental caries and an individual's phenotypic expression.[21] This is clearly manifest in the transmission of the *S mutans* infection between mothers and their children.

If a mother harbors a significant infection, then there is prima fasciae evidence that the particular strain of *S mutans* resident in the mother is able to successfully compete with the mother's immune response. If it were susceptible to the mother's immune system, it would have been defeated and hence not observed in high concentrations in the oral cavity. In saliva, the primary immunologic response is secretory immunoglobulin A (sIgA), which exhibits its influence by binding selectively to the surface of *S mutans* at areas used by the organism to attach to a solid surface.

We also know that, in large part, children manifest the mother's immune system for the first months of life. This immunity is transferred both by the placenta and via colostrums found in lactation products.[22-25] Since the child's temporarily acquired immune system is not particularly effective against the strain of *S mutans* resident in the mother, acquisition is more likely to occur from the mother than others.

Inherited disorders that affect tooth development or salivary flow and immune system competency increase the incidence of dental caries.[26] Dental enamel that is not well mineralized, such as in cases of ectodermal displasia, may also be more susceptible to dental caries.[27] Other inherited disorders and other genetic factors are not considered in this paper.

Environment

The environmental issues considered here are: (1) the teeth present, including their sealant status and caries status/history; (2) the family caries history; (3) carbohydrate (sugar) consumption; (4) socioeconomic status; and (5) fluoride exposure.

The teeth that a child or adolescent has resident in the oral cavity are associated with the risk for caries. Until the first teeth erupt, there is no chance for cavities. Once the teeth erupt the risk of acquiring the infection that contributes to dental caries increases with the increasing cumulative surface area of the dentition, as seen in Figure 5, which is adapted from the work of Caufield et al.[28]

We also know that one of the best predictors of future cavities is the presence of current caries or evidence of past caries in the form of existing restorations.[29,30] The specific location of current or previous caries also helps predict future caries incidence. Patients that exhibit only pit and fissure caries and not smooth surface caries have less risk of future caries than those who have extension of the disease onto smooth surfaces.

Another indicator of caries risk is the presence or, more precisely, the absence of sealants. Bravo and colleagues demonstrated that the presence of sealants reduces the risk of both pit and fissure and smooth surface caries.[31] The protective nature of sealants and fluoride varnishes on both fissures and smooth surfaces can be seen in Figure 6, adapted from Bravo's work.

As previously noted, the child's mother provides the most probable source of transmission of the caries organisms.[5,16] The level of the infection in the mother correlates directly with the potential for transmission of the organisms to their children and their subsequent development of dental caries.[32] The higher the mother's level of *S mutans*, the greater the probability of transmission to the child.

Clearly, one of the major environmental factors for caries is the frequency, level and consistency of exposure to refined carbohydrates.[33] In general, the greater and more

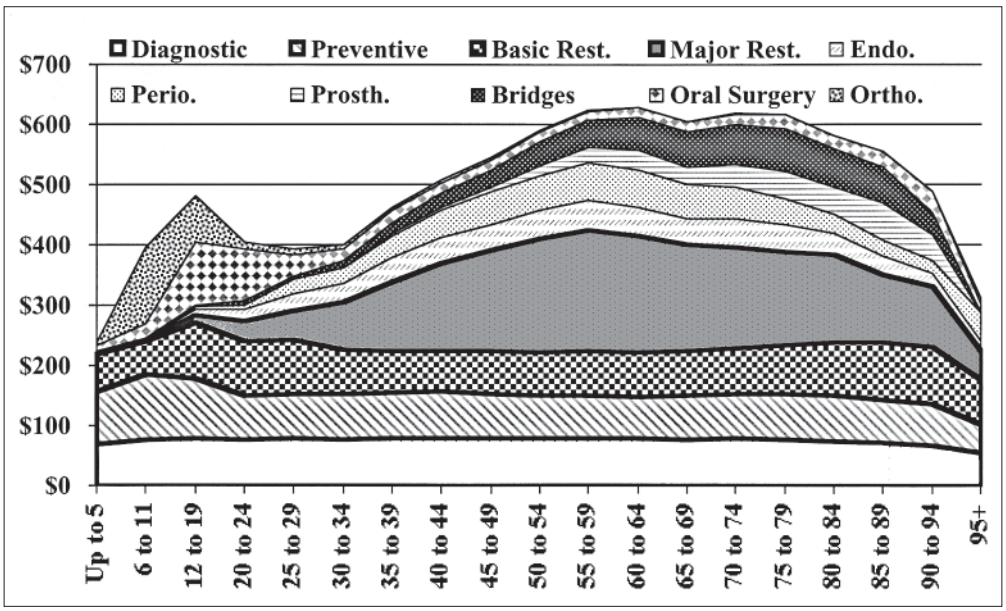

Fig 7. Cost of dental services in the year 2000 stratified by age

frequent the exposure, the greater the risk of developing caries although this risk appears to be at least partially offset by twice-per-day brushing with a fluoridated dentifrice.[34]

Socioeconomic status (SES) is moderately well associated and inversely related to risk for dental caries in children.[34, 35] It is noteworthy that almost all studies of this environmental relationship are cross-sectional in nature and do not discriminate well on the actual at-risk tooth surfaces. When studies capture the fluoridation status of the water supply, fluoride significantly attenuates the SES risk.[36]

Many other environmental factors can be considered for their contribution to the risk of dental caries. A review of many of these is provided in the most recent NIH Consensus Development Conference on Diagnosis and Management of Dental Caries Throughout Life.[9] The key risks have been covered here.

Keeping an eye on the goals

It is useful to remember that the purpose of risk assessment in the current context is to make available to the clinician information that will alter his or her future preventive and treatment strategies with the prospect of diminishing future disease. Since clinicians have effective strategies for childhood caries, risk assessment is critical in the use of targeting their resources. Assessment strategies or practices that do not meet these goals are of little use to the clinician.

Caries risk assessment

Using the non-exclusive contributory model expressed in Figure 3 and including the background information presented above, it seems useful to construct an example of how this information can be used to determine caries risk in pediatric or adolescent patients.

Given the nature of this disease as an infection, a model will be constructed that starts with ascertaining the presence or absence of *S mutans*. If present, the degree of the infection should also be noted. The diagram's logic can then be employed, as can knowledge of the various factors to render a reasoned view of a patient's risk of incurring future dental caries.

Recently introduced techniques for the qualitative detection and quantitative description of *S mutans* are making assessment tools available to those outside of academia.[37] As established above, the bacteria are "contributory." An important aspect of the predictive capacity of these tests is that they have high "specificity." That is, the absence of a positive test for the presence of the organisms accurately predicts that the child's risk for future dental caries is low.[38,39] Even though less predictive, the "sensitivity," expressed as specific levels of *S mutans*, still has a positive correlation to increased caries.

In general, the higher the number of risk factors for an individual, the greater the probability that he or she will incur the clinical manifestations of the *S mutans* infection—cavities. Clearly, the weighing of all risk factors is problematic for each individual and, hence, the clinician's cognitive skills are currently relied on to relate these factors. This process of correlating and relating the factors is called "clinical judgment."

Clinical judgment is not a precise, formula-driven science. Rather, it is the application of inductive and deductive reasoning processes to a presenting clinical situation. The tools are only now being acquired that will move "clinical judgment" in risk assessment from the "informed subjective" toward codified "objective" decisions. The profession's present "risk assessment" armamentarium is a collection of rather imprecise measures with which clinical judgments are rendered.[38,40]

In this simple case, the child is at increased risk for caries if he: (1) has salivary counts of *S mutans* above 250,000 cfu/ml of saliva; (2) has a pit and fissure morphology that provides a potential ecologic niche for the *S mutans*; (3) has a mother with a dental history of numerous restorations; lives in an unfluoridated community; and (4) does not brush twice per day with a fluoridated dentifrice. In this case, the

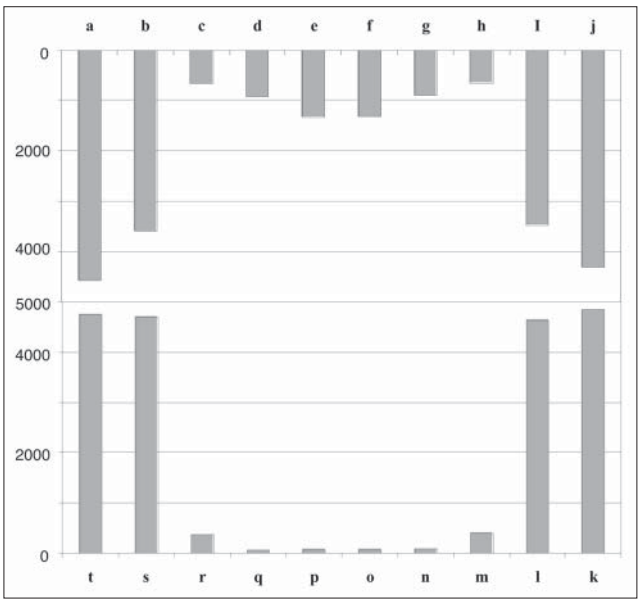

Fig 8. Distribution of restorative services in the primary dentition of 0- to 6-year-old patients (n=12,951)

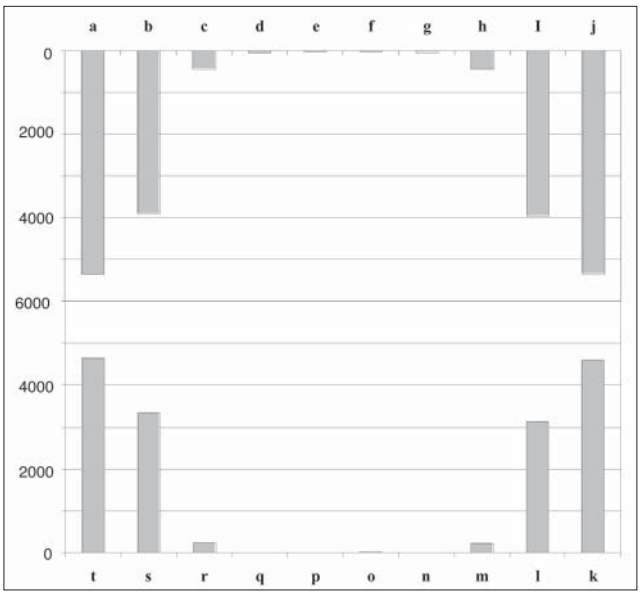

Fig 9. Distribution of restorative services in the primary dentition of 7- to 13-year-old patients (n=16,085)

risk assessment should lead the clinician toward the application of dental sealants and training in twice-per-day brushing with an ADA-approved, fluoridated dentifrice.

We can create an infinite set of risk factor mixes and test the model extensively. The great utility of this review is to refresh and renew our understanding of the caries process and the risk factors associated with the disease.

In concluding this section, the author has attempted to give the reader a list of the risk factors associated with dental caries in the pediatric and adolescent populations. By using the reviewed materials and the risk factor diagram, clinicians may exercise their clinical judgment and determine the relative risk an individual bears for developing dental caries.

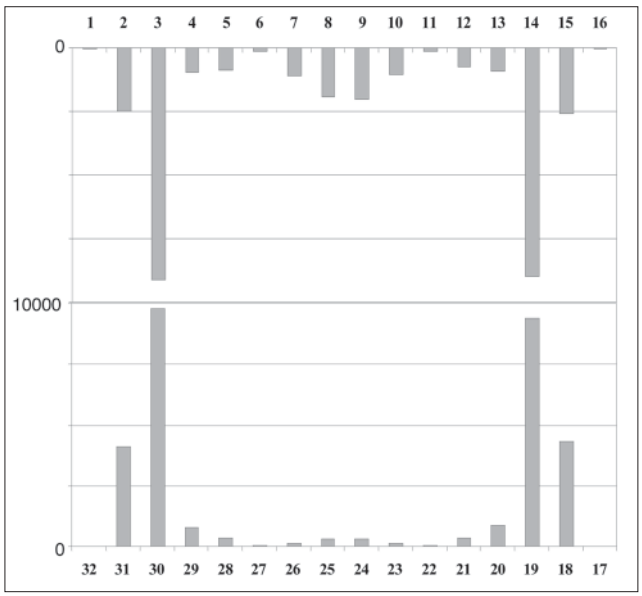

Fig 10. Distribution of restorative services in the permanent dentition of 7- to 13-year-old patients (n=29,054)

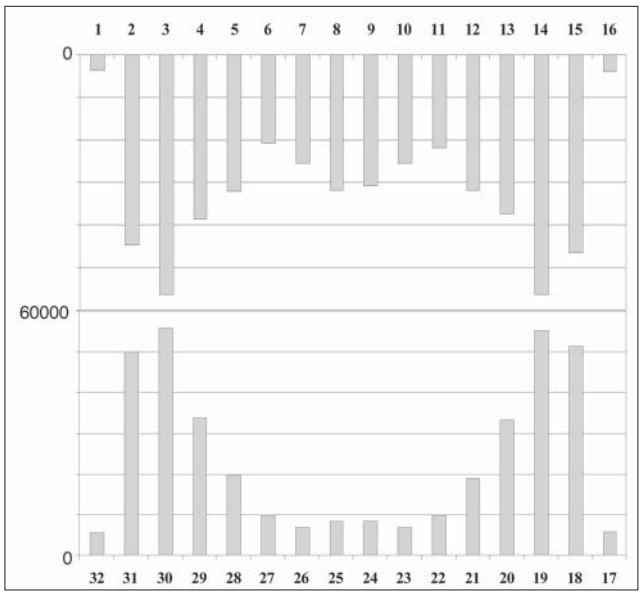

Fig 11. Distribution of restorative services in the adult dentition for all ages (n=288,467)

The epidemiology of dental caries in children and adolescents

The recent Surgeon General's report on the nations oral health outlines in great detail the prevalence of dental caries in our population. Much of this has been discussed previously in this paper. It is instructive to look at the services rendered to populations to see how our dental resources are employed. As part of his work at Delta Dental Plan of Washington (also known as Washington Dental Service, WDS) the author has developed a data warehouse, employed a staff and partnered with numerous academicians to analyze the archived data. Data warehouses are integrated, business-oriented computer databases with long-term time horizons of "frozen" snapshots of treatment and other data.

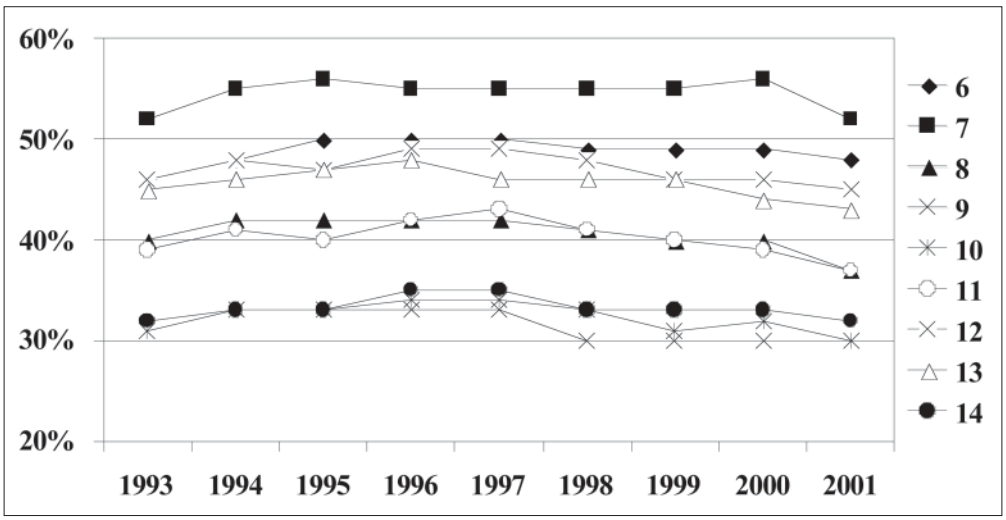

Fig 12. Sealant application pattern by age for the period of 1993-2001 in the insured population of WDS (n=25,000 per year)

They may contain very large data volumes, depending on the size of the populations being captured and the granularity of those data.

The WDS data warehouse contains longitudinal data on 1.6 million individuals, beginning in 1993, with a level of detail down to the individual tooth surface. Using these data, one is able to augment the data from the Surgeon General's report by providing specific information on how dental services are provided to the populations of interest.

Figure 7 examines the services provided to covered child and adolescent patients, based on the dollars expended, including the patients' copayment as recorded for the year 2000.

Since the cost associated with restorative services is primarily the result of dental caries, it is useful to examine how and where (intraoral location) these services are provided. Figures 8 through 11 show the distribution of services by tooth number in the primary, mixed and adult dentitions. The population sample size for each graph is included as a footnote.

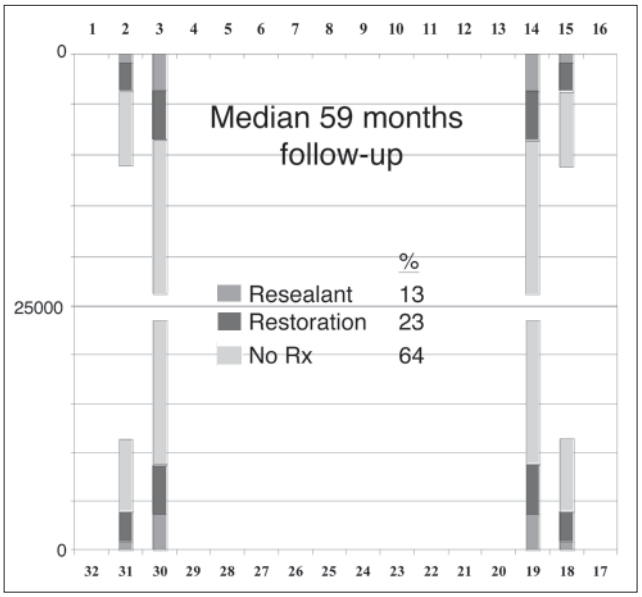

Fig 13. Distribution and fate of sealants applied to the permanent molars

As previously noted, sealants play an important role in the prevention and treatment of dental caries. Multiple authors have demonstrated the effectiveness of sealants for the preservation of the tooth's occlusal surface.[41,42] Bravo and his colleagues demonstrated an overall reduction in caries when sealants were employed in the populations being treated, as seen previously in Figure 6.[43] These data corroborate our own findings that when a child receives sealants, they are 72% less likely to receive restorative services over the next three years than their peers who do not receive sealants. Dentists need to remember that an administrative database like the warehouse does not capture treatment need or diagnostic status associated with the use of sealants. While acknowledging the limitations in these data, this is still a remarkable finding, given that, outside the operator, sealants are the only consistent variable.

The use and fate of sealants in specific populations has been examined as a cost-effective strategy.[44,45] Application of the risk-assessment strategies discussed in the first part of this paper can provide guidance for the clinical application of sealants. The data would suggest that those children with multiple risk factors and a tooth fissure morphology that provides an ecologic niche for *S mutans* should have their molar teeth sealed prophylactically. The actual use, retention, replacement and restoration of sealants is shown in Figures 12 and 13.

Figure 12 shows the percentage of available children that received sealants in the population stratified by their age at the time they received the sealant. Figure 13 shows placement and the outcomes of sealant placement in this population.

Summary

This paper presents the best current information on risk assessment and the epidemiology of dental caries in pediatric and adolescent populations. While the profession has made great strides in reducing the amount of disease in the population through the wide application of fluoride, there remains a significant amount of work to do in the area of risk assessment and its application to practice. One of the critical elements for the future that is not discussed in this paper is altering the clinician's behavior to take advantage of increasing knowledge of risk assessment. It is through consensus conferences such as this meeting that the profession begins the task of altering clinical behaviors to match the emerging science.

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AAPD and ASDC Merge!

Members with e-mail addresses were notified on July 16, 2002 that the AAPD had officially merged with the American Society of Dentistry for Children (ASDC). The AAPD Web site home page (www.aapd.org) includes the press release on the merger, as well as background information for ASDC members (both pediatric dentists and general dentists).

This merger, which is the result of several years of discussion and negotiation between both organizations, will lead to many exciting future opportunities for the AAPD. The press release indicated that the AAPD is now the unified voice for children's oral health, which will greatly enhance our efforts with federal and state governments, foundations, other health and dental-related organizations and child advocacy groups.