Causes and Prevention of Dental Caries: A Perspective on Cases and Incidence

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Purpose: The purpose of the present paper is: 1) to review recent concepts of the causes of caries; 2) to illustrate parameters of causes of cases and of incidence; and 3) to outline the consequences for caries prevention and oral health promotion. The paper is divided in three sections. Section 1 reviews recent theories of the causes of caries and summarizes that it is necessary to make a clear distinction between the causes of a case of caries and causes of occurrence of caries in populations. Cases relate to the reasons why individuals get sick, while incidence relates to why so many (or so few) within a population get sick. In section 2 the difference between the causes of a case and of the occurrence in a population is illustrated. A new social epidemiology moves beyond the focus on individual level risk factors to a multi-level perspective. Applied to oral diseases the paradigm of social epidemiology bridges our understanding of the biological determinants of caries with an understanding of the societal determinants of caries. Social epidemiology is the branch of epidemiology that studies the social distribution and social determinants of states of health. Individuals are embedded in societies and populations. In section 3 the insight acquired above is applied to the choice of disease-preventive and oral health-promotive strategies. Prevention of caries and promotion of oral health must be rooted in the understanding of caries as it occurs in populations.

Key words: dental caries, causes, cases, incidence, prevention, promotion

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Caries is a chronic disease that progresses slowly in most individuals (Fejerskov et al, 2003). Caries is a very widespread oral disease. Caries is considered to be ubiquitous since metabolic fluctuations are always present in the biofilm in all populations (Fejerskov, 1997; Kidd and Fejerskov, 2003).

The parameters of caries initiation are the biofilm and the metabolic activity that constantly takes place within it. Dental caries may develop on any tooth surface in the oral cavity where a microbial biofilm is allowed to develop and remain for a period of time (Baelum and Fejerskov, 2003). The presence of a microbial biofilm does not necessarily result in a caries lesion, but is a necessary factor. However, the activity within the biofilm results in numerous minute pH fluctuations at the interface between the tooth surface and the microbial deposits. In simplified terms each single pH fluctuation may cause a loss of mineral from the tooth when pH drops, or a gain of mineral from the tooth when pH increases (Baelum and Fejerskov, 2003). The cumulative result of these demineralisation and remineralisation processes may be a net loss of mineral, leading to dissolution of the dental hard tissue and possibly a caries lesion or a case of caries.

The scale of the problem of caries in a population is determined by the occurrence of caries. The parameters of occurrence, such as incidence rate for a particular disease, are not constants of nature (Miettinen, 1985). Rather their magnitudes generally are functions of a variety of characteristics of individuals, their behaviors, and contextual and environmental conditions.

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The relation between occurrence parameters and determinants can be viewed as either descriptive or causal. In a descriptive problem, a parameter of occurrence is related to a determinant without any view to causal interpretation of the relation. Descriptive occurrence relations are of interest for risk assessment of prognosis, diagnosis, allocation of case findings and other services, service evaluation and other purposes (Miettinen, 1985). Etiologic insight and rational intervention, by contrast, rest on information about occurrence relations that may be interpreted in causal terms (Miettinen, 1985). Better causal insight of the occurrence of caries has a potential both in itself and for the control of the disease. According to Holst et al (2000) it is necessary to make a clear distinction between the causes of caries as such and of the occurrence of caries in populations. The occurrence of a caries lesion and of caries in a population has different causal candidates and causal patterns. Some of the disagreement among researchers as to the causes of caries may be rooted in lack of a causal precision.

The purpose of the present paper is: 1) to review recent concepts of the causes of caries; 2) to illustrate parameters of causes of cases and of incidence; and 3) to outline the consequences for caries prevention and oral health promotion. The paper will be divided in three sections accordingly.

RECENT CONCEPTS OF THE CAUSES OF CARIES

Several researchers have developed causal models for dental caries. Keyes (1962) founded the basis for most models described over the past 40 years. He used the host-agent-environment model to describe the occurrence of dental caries. Dental caries only occurs when the three factors are present simultaneously. Newbrun (1989) postulated that the factor time should be considered in any discussion of the etiology of dental caries; Geddes (1991) added saliva as a protective factor to Keyes's model; and Nikiforuk (1985) described the etiology of dental caries by primary factors such as susceptible host, a suitable substrate and cariogenic microflora, and secondary factors such as oral hygiene, saliva, fluoride, diet, nutrition, oral sugar clearance, composition of the enamel, morphology of the enamel, age of the teeth and the crystalline structure of the enamel. In his model, Nikiforuk (1985) noted an interplay between primary factors and many secondary factors that modify the causal process.

Fejerskov and Manji (1990) introduced a model to emphasize the relationship between dental plaque (the etiological factor) and multiple biological determinants that influence the likelihood of a caries lesion developing. According to their model a determinant is any factor that may influence the outcome, but in itself cannot cause loss of mineral. A determinant may influence the rate of development and progression of mineral loss. In Fejerskov and Manji's model the determinants are time, fluoride, diet, microbial species, saliva flow rate and composition, sugar (clearance rate), and buffer capacity. Further distant to the process the authors have positioned a variety of socio-economic and behavioral factors (social class, income, knowledge, attitude, behavior and education) and indicated that these should not be considered etiological factors or determinants, but confounders.

In a recent presentation Baelum and Fejerskov (2003) revised the description of the model of caries and included confounding variables as determinants. They emphasized that dental caries may be considered from three different perspectives, owing to the fact that the tooth surfaces and the teeth cluster in individuals, whereas individuals cluster in populations. "Caries is a question of unfavorable localized biological processes. However, these unfavorable processes are determined by environmental and societal factors that may be more amenable to change than are local biological processes" (Rose, 1985).

Silverstone et al (1981) restricted etiological factors to the interplay between bacteria, saliva, foodstuff and crevicular fluid in shaping a cariogenic plaque. Based on Silverstone et al's model of caries, Johnson (1991) and Brathall (1996) restricted the etiology of caries and the causes of caries to the biological process. According to Brathall's cariogram, diet, bacteria and host susceptibility represent the immediate action on a tooth surface. Behind each factor there are several other factors that determine the actual contribution of diet, bacteria and host susceptibility. Bokhout et al (2000) used Rothman's sufficient cause model to develop a biological caries model that distinguishes between causal and protective factors, effect modifiers and confounders. The philosophy behind the 'sufficient cause' model is that the cause of any disease must consist of a constellation of com-

ponents that act in concert. Dental caries can only occur when three component causes are simultaneously present: teeth, fermentable carbohydrates, and micro-organisms with a cariogenic potential. Bokhout et al (2000) underscored that other factors affect the actual disease occurrence. Whether dental caries is actually formed also depends on saliva, fluoride, oral hygiene and diet. These factors interact with the component causes of dental caries in either a positive protective or a negative risk increasing way. Sex, age, and race are not included in the model because they are considered to be effect-modifiers and confounders. In a pragmatic conceptual analysis of causal relations and caries, Scheutz and Poulsen (1999) suggested that "a factor is a cause of disease when changes in the factor inevitably lead to changes in morbidity". The latter appears to be an unnecessary restriction of Rothman et al's (1998) definition of sufficient and component causes. A potential cause can be part of a causal web and conditionally dependent upon other component causes, and may therefore be a cause in a chain of reactions without necessarily leading to a change in outcome (Dean, 1993). Eriksen and Dimitrov (2003) have suggested analyzing caries and oral health in the perspective of complex theory by which the social and psychological characteristics of the human mouth are better understood.

It is clear from the literature above that the specification of the causes of caries depends in part on the frame of reference of the investigator and the scope of his inquiry, that is, on the conceptualization of the study (Susser, 1973). Differences in concepts and in the frame of reference determine the type of causes included. The main criticism of the suggested causal models of caries is that they do not take into account the difference between the parameters of caries initiation that is the etiology of a single caries lesion on a tooth surface, and the parameters of occurrence of caries in time and space; the latter meaning in a specific population at a specified time. This is equiralent to the distinction between explaining a single tubercle lesion in a lung and the occurrence of tuberculosis in a population. It is fairly obvious that explaining changing incidence rates of tuberculosis in a population involves other mechanisms than the actual pathological development of a lesion. Not to make the distinction creates uncertainty and unnecessary disagreement among researchers. Holst et al (2001) discussed the necessity of an overarching framework in order to understand occurrence of caries within and across populations. The necessity of making a distinction between the causes of a case of caries and causes of occurrence of caries in populations is rooted in the famous distinction by Rose (1985) between causes of cases and causes of incidence. Cases relate to why individuals get sick while incidence relates to why so many (or so few) within a population get sick.

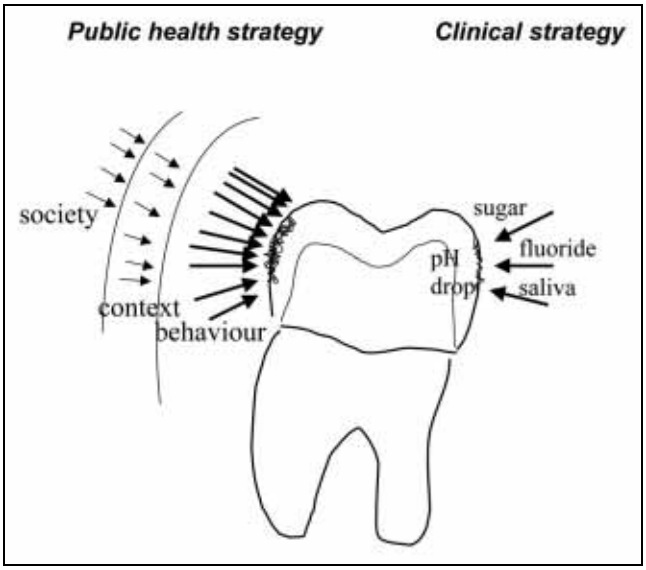

Fig 1 Caries as a social, contextual, individual and biological process.

THE PARAMETERS OF CASES AND OCCURRENCE OF CARIES IN A POPULATION

Fig 1 illustrates a case of caries and the determinants of occurrence of caries in populations. A metabolic process is developing on the right side of the drawing, involving the biofilm, bacteria, substrate in terms of sugar, saliva. If fluoride is present demineralisation is delayed and remineralisation is favored. In a biological context variables like these are the causes of a case of caries. The variables explain the onset of a cariogenic process. These factors alone do not explain the occurrence of caries; that is how much caries a person gets and how many persons are affected by caries in a population. It is therefore necessary to make a distinction between the causes of a case and the causes of incidence or occurrence in a population (Rose, 1992).

The left side of the figure illustrates the causes of occurrence. In order to understand caries in populations one must ask why plaque gets old and sour and why is it not removed, why pH drops many times during the day, and whether or not fluoride is present. The arrows show the number of times carbohydrates are eaten and thus the number of times the tooth is attacked and pH drops. The more attacks, the more arrows, the more demineralisation. The arrows are behaviors, such as dietary behaviors, forgotten or neglected oral hygiene. With inadequate decisions and behaviors of individuals the attacks happen more often during the day. The identification of causes on the left side of Fig 1 includes behaviors of oral hygiene, diet, between meal eating, drinking sugar-containing soft drinks, etc. These are some of the intermediate parameters of caries occurrence in populations.

The causal investigation does not stop here, unless it is implied that the behaviors and decisions are made solely by individuals. By doing so it will be overlooked that behaviors are not performed in a vacuum and people are blamed who are the victims of societal conditions that are unfavorable to health (Sheiham, 2000). Even if individual behaviors were successfully changed, each year thousands of new people would continue to behave the old way as long as the roots of these behaviors are not modified. Rose (1992) said it very clearly: "When you try to change individual behavior you modify the causes of cases, not the causes of incidence in the whole population."

Over the past two decades growing evidence has demonstrated that health is highly sensitive to the local context and to societal conditions (Fig 1). Attributes such as stressful living conditions, supportive social relationships, childhood environment and relative disadvantage have emerged as key determinants of health and of oral disease. A new epidemiology is now gaining attention (Berkman and Kawachi, 2001). In order to understand disease and its distribution in the population, social epidemiology suggests that it is necessary to have a view that bridges the entire range from social and economic policies through institutions, neighborhoods and communities, living conditions, social relationships, individual risk factors, as well as genetic and patho-physiological pathways (Susser and Susser, 1996; Berkman and Kawachi, 2001). Applied to oral diseases the new paradigm of social epidemiology bridges our understanding of the biological determinants of caries with our understanding of the societal determinants of caries. Social epidemiology is the branch of epidemiology that studies the social distribution and social determinants of states of health (Berkman and Kawachi, 2001). Individuals are embedded in societies and populations. The crucial insight provided in 1992 by Rose's population perspective is that an individual's risk of illness cannot be considered in isolation from the disease risk of the population to which he or she belongs.

Rose's insight has broad applicability to a range of public health problems ranging from aggression and violence, mental health problems, to effects of poverty and material deprivation on health. The implication of Rose's theory for social epidemiology is that the social context must be incorporated into explanations about why some people stay healthy while others get sick.

A causal model of caries occurrence in populations comprises variables from the inner level to the outer level (Fig 1). The most distant variables exert their effect on caries via indirect effects. Attempts to estimate direct effects of such variables may lead to weak or zero empirical association that are wrongly interpreted and therefore excluded or strongly underestimated. Distant variables may be very important for disease occurrence through their necessary relationship somewhere in the chain. This is in contrast to predictive studies where researchers seek relatively strong predictors of an outcome without any demand on causality (Miettinen, 1985). Variables that have been scientifically justified to be included in a causal web are not confounders of relationships taking place closer to the outcome. Confounders are variables that cause change in the dependent or outcome variable and are statistically associated with a hypothetical causal variable (Susser, 1973). In studies of hypothetical monocausal associations the confounders appear as uncontrolled third variables that are associated with both the independent and the dependent variable. However, in a multilevel causal model all variables included have an assumed role in the web leading to the outcome.

CONSEQUENCES FOR PREVENTION AND ORAL HEALTH PROMOTION

The choice of disease preventive and health promotive strategies is decisively dependent on the causal perspective. If it is assumed that the causes of

caries can be identified in the oral environment and on the tooth surface, preventive activities will be selected among such factors. This implies, for example, that the focus will be on bacteria, and plaque will be removed either chemically or physically, the intake of carbohydrates will be restricted, and the tooth surface strengthened by fluoride. As shown in Fig 1 these measures do not interfere with the societal and contextual conditions that threaten oral health. Neither do clinical measures interfere with the behavioral decisions to rinse, brush, eat and snack, and therefore the arrows will not be reduced in number. Adding fluoride locally or in toothpaste will compensate the effect of the arrows, but will not remove them. This may also be the reason why Hausen and et al (2000) and Källestål et al (2004) showed that there was no effect of a high-risk strategy intervention in Finland and Sweden, respectively. The interventions did not reduce the social causes of caries but only tried to defend the tooth surface; and that does not work well enough against social forces. The preventive potential lies in supporting individuals and families to provide them with enough self-motivation and strength to take appropriate decisions with regard to what they eat, drink and snack. Sheiham (2000) suggests that individual lifestyles are expressions of the social and cultural circumstances that condition and constrain behavior in addition to personal decisions the individual may make. Three approaches to oral health are suggested: a population rather than individual approach; a common risk factor approach; and multidisciplinary working. Health policies that provide health, social and welfare support can act as a springboard to assist the most vulnerable groups to achieve their full potential in society (Blane, 1999).

Furthermore, it is important to make another distinction, namely between preventing a case of caries by means of plaque and sugar control, and promotion of oral health in populations by means of strategies for maintaining health (Holst et al, 2000).

The value of this insight is that it results in a more effective programme for the promotion of oral health. This is not to deny the importance of working with individuals one at a time on a clinical basis. Every patient should receive individually tailored attention and clinical prevention. But prevention on the individual level does not change the roots of incidence of the disease in the population because it does nothing about the forces in society that cause the behaviors in the first place. Solely focusing on changing the lifestyle of individuals is both ineffective and very costly (Marmot, 1998; Syme, 2004).

Conceptually these perspectives are difficult to manage theoretically and practically. One approach is to take advantage of the patterned regularities in oral disease rates. The most impressive patterned regularity of all is social class. It is well known that people lower down the social class scale have higher rates of virtually every disease and condition. Despite this universal recognition, surprisingly little is known about the reasons for this phenomenon. The list of possible explanations is long and well known. It includes poverty, bad housing, unemployment, poor nutrition, inadequate medical care and poor education (Muntaner et al, 2000). The relative importance of these various factors is not known, because we do not study social class. Social class is of such overwhelming importance that epidemiologists typically hold it constant in order to study other things within each social stratum. This is done in order to see the role of other factors that would otherwise be drowned by social class. However, the interrelation between social class and these other important factors remain modestly understood.

In conclusion the paper has demonstrated the importance of understanding the difference between the causes of cases of caries and the occurrence of caries in populations. Causes that contribute to a case of caries cannot explain the occurrence of caries in a population. Prevention of caries and promotion of oral health must therefore be rooted in the understanding of caries as it occurs in populations.

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