Review

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Using Universal Patterns of Caries for Planning and Evaluating Dental Care

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Key Words

Dental care planning · Dental caries patterns · DMF indexes · Epidemiology trend lines

Abstract

There are universal patterns of caries, in terms of prevalence, incidence, frequency distribution and rates of progression, in permanent teeth that can be considered working rules that can be applied when planning dental care. The universal patterns are: (1) caries levels follow trend lines; therefore, knowing the caries level at one age can be used to predict the levels at later ages in that cohort by looking at the trend line for that cohort; (2) the distribution of dental caries of a population exhibits the following characteristics: as the mean DMFT increases, the percentage of caries-free individuals falls and the caries distribution widens; this changing relationship between the mean DMFT and prevalence is not limited to a subgroup of the population who already have had some caries experience; (3) there is a specific mathematical relationship between the mean DMFT and mean DMFS; (4) there is a hierarchy of caries susceptibility by tooth type and sites on teeth; for a given DMFT or DMFS, there is a specific intra-oral pattern of caries by tooth type; (5) changes in mean DMFT scores for individuals and groups are not linear, but 'stepped'; there are groupings of teeth and tooth sites that may have similar 'resistance' to caries; (6) as the mean DMFT declines, the posteruptive time for initiation of caries

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Accessible online at: www.karger.com/cre increases and the progression rate of caries through enamel decreases. This is true regardless of the presence of fluoride. Any improvement in dental health will cause this effect.

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This paper sets out to show that there are universal patterns of caries, in terms of prevalence, incidence and rates of progression in permanent teeth, which can be considered working rules that can be used as a decision tool in determining the appropriate treatment needs and preventive strategies and agents to use for a given level of caries. Yet, despite the availability of so much epidemiological data on dental caries, there have been very few attempts to operationalize the data so that they can be readily used to plan dental care. This review sets out to present data on patterns of caries in the permanent dentition in a form accessible to planners and decision makers. The review is based on abundant data on the life history and patterns of dental caries in the permanent dentition available from a wide range of countries which, when compiled and analysed, could be a sound basis for rational planning of dental care for populations and individuals.

Massler et al. [1954] compiled epidemiological studies on dental caries and formulated 'rules' about the relationship between levels of caries and the teeth affected for each level. McDonald and Sheiham [1992] corroborated

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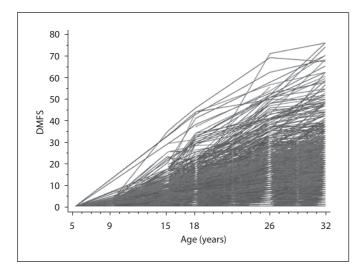


Fig. 1. Simple join of DMFS scores by age: the Dunedin study [from Broadbent et al., 2008, reproduced with permission].

their findings when compiling dental caries data from different periods and countries. Recently, their findings have been elegantly substantiated by Broadbent et al. [2008], who showed that there were clear trajectories of caries from the age of 5 to 32 years (fig. 1). These 3 studies showed that levels of caries at 6 years of age, for individuals and groups, predict the caries levels in adulthood. Another rule of caries was proposed by Knutson [1958]. He demonstrated that there was a defined relationship between the prevalence of caries attack, measured as having 1 or more teeth with decay, and the intensity of caries attack, measured as DMFT, and also the relationship between DMFT and DMFS. Knutson's findings have been confirmed but seldom used [Kortis et al., 1978; Järvinen, 1983]. Later, McDonald and Sheiham [1992] suggested another working rule based on patterns of teeth and tooth sites affected at different levels of DMFT. They claimed that 'as caries prevalence falls, the least susceptible sites (proximal and smooth surfaces) reduce by the greatest proportion, while the most susceptible sites (occlusal) reduce by the smallest proportions'. Different teeth and tooth sites are affected differentially at different levels of dental caries. This truism may appear obvious but it is not used to decide on appropriate treatments, such as whether to use fissure sealants and which teeth to seal, and the length of recall intervals and to evaluate the effectiveness of dental treatment. Another example of a rule of caries was the analysis, based on the finding of Rose and Day [1990] that the population mean predicts

the number of deviants. Applying their concept, Batchelor and Sheiham [2002] showed that there was a regular relationship between the population caries mean and the shapes of the frequency distributions of caries.

Such knowledge of the life history and robust patterns of caries attack rates in permanent teeth, within populations and individuals, could be used to plan and assess interventions. This review paper is not based on a systematic review of all available information but uses key examples from large population samples with differing levels of caries in the permanent teeth and gleaned over the past 50 years. The review does not extend to caries patterns in primary teeth or the relationship between caries in the two dentitions because there are few robust studies available on those subjects. The objective of this review paper is to demonstrate that there are some well-established characteristics about dental caries patterns in permanent teeth that are of use for interpreting data and planning dental services for populations. We shall demonstrate that for different mean DMFT scores one can predict the following: (1) the mean DMFT levels at later ages from current levels because caries levels for groups follow predictable trend lines, 'tracking', if environmental conditions are reasonably stable and where there was no effective intervention; (2) the shapes of the frequency distributions of children with different levels of caries; from those distributions, one can calculate the frequency distributions of DMFT at different mean levels of DMFT and the percentage of cavity-free children; (3) the relation between the mean DMFT and the DMFS; (4) the intraoral pattern and hierarchy of caries susceptibility of teeth, which teeth and tooth surfaces will be affected at given DMFT levels and the mean DMFS by types of sites affected, which groupings of teeth and tooth sites are likely to develop caries at different levels of caries within a given time period; (5) the rate of progression of caries through enamel and the time after eruption that caries is likely to be initiated and cavitation occurs.

The evidence for the above-mentioned working rules of caries is presented in more detail below.

The Relationship between Current Caries Levels and Mean Caries at a Later Age

One of the remarkable features of a number of chronic health conditions such as blood pressure is that the blood pressure levels at one age predict the levels at a later age [Chen and Wang, 2008]. The phenomenon is known as 'tracking' because an individual or group with

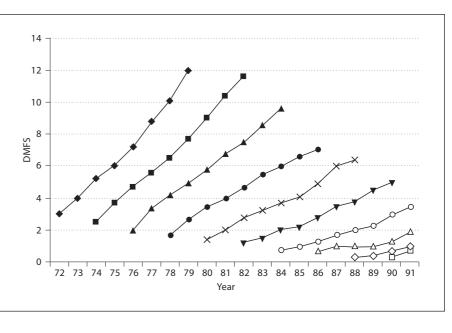


Fig. 2. Caries development (DMFS) in Danish children from first to ninth school grade [Schwarz et al., 1994].

a particular level of blood pressure follow a 'track' allowing their blood pressure at a later age to be predicted from their earlier level. The same tracking phenomenon occurs with dental caries. Massler et al. [1954] proposed that groups with a particular severity level of caries at 6 years of age would follow a 'channel' or track representing their dental caries increment. The groups with the higher severity level at 6 years of age would follow a higher 'channel' or track representing the higher dental caries increment. Based on annual incremental data they proposed that the level of severity of the caries attack, if known at certain ages, would allow the prediction of dental-cariessusceptible groups and teeth. The allocation to the 'channel' was based on a hierarchical method of caries attack within a mouth. If only the lower first molars were involved at 8 years of age, the attack was said to be mild. If, at the same age, all 4 first molars were affected, the attack was moderate. The idea of individuals being in susceptibility 'channels' was also highlighted by Carlos and Gittelsohn [1965]. Tracking is also obvious in large national population studies such as those in cohorts of Danish children who were examined annually during childhood [Schwarz et al., 1994] (fig. 2). Danish cohorts commencing school in 1972 had a DMFS of about 3, and by the age they left school their DMFS was 13. The 1982 cohort had a DMFS below 2 and a DMFS of about 4.5 when examined 8 years later. The slopes of the trend lines changed as the caries levels decreased (fig. 2). In a longitudinal study, Broadbent et al. [2008] showed that levels of caries in

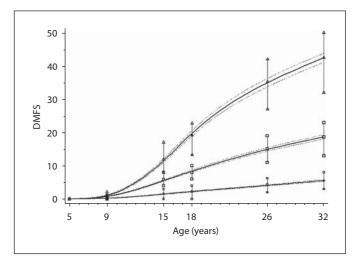


Fig. 3. Trajectory plot of mean DMFS scores for a 3-group caries trajectory analysis model: Dunedin Study [from Broadbent et al., 2008, reproduced with permission]; symbols indicate the 3 groups.

5-year-olds tracked into adulthood; there were clear trajectories of caries for individuals and groups, from the age of 5 to 32 years (fig. 1, 3). By assigning individuals to 3 groups they showed clear trend lines for each group. The plots of the 2 higher trajectory groups were S-shaped while the lowest trajectory group was linear (fig. 3). Similar trends were observed in a number of DMFT cohorts (fig. 4) [McDonald and Sheiham, unpubl. data].

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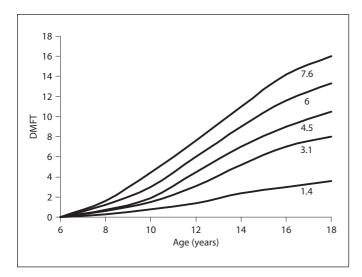


Fig. 4. Tracking of DMFT from the age of 6 to 18, by DMFT at 12 years [McDonald and Sheiham, 1992].

The Frequency Distribution of Caries for a Given DMFT Is Relatively Uniform

Universally, the frequency distribution of dental caries within populations exhibits some predictable characteristics that can be modelled. For a given mean DMFT level, the shape of the frequency distribution is similar for all populations with that DMFT. There is a predictable relationship between the mean DMFT and the prevalence. In other words, there is a particular percentage of the population distributed at each DMFT/DMFS interval for a given prevalence mean DMFT. As the percentage at each DMFT/DMFS is standard for that DMFT/DMFS, it is easy to calculate the percentage of subjects who are cavity free. The percentages at certain DMFT scores are not as predictable as at others. The changes are more predictable, for example, between DMFT scores of 1 and 3, compared to 1 and 2. Similar percentages of individuals are found at particular DMFT scores, for example DMFT 3 and 4, 5 and 6, and 7 and 8.

The frequency distribution of caries in populations shifts to the left as mean scores decrease (fig. 5, 6) [Seppä et al., 2000; Cypriano et al., 2008]. As the mean of caries decreases, the whole distribution shifts to the left pulling the tail with it so that the so-called high-risk group becomes smaller and smaller (fig. 7) [Armfield et al., 2009]. At high levels of caries, the distribution is normal with a longish right tail. It is obvious that as the mean DMFT score increases, the percentage of the population who have caries also increases. The reverse is also true. As the prevalence decreases, the mean DMFT/S decreases. This changing relationship between the mean DMFT and prevalence is not limited to a subgroup of the population who already has had some caries experience. The increase occurs throughout the child population. There is a population shift in the frequency distribution. The next question is whether the population shift is a constant, namely, as the mean increases are the changes equally distributed? This question can be answered by looking at the distribution around the mean. If the standard deviation remains the same, then the effects of any change are distributed equally among the population. If the standard deviation increases with the mean, then the gap widens between those at one end of the tail and the other. As the mean DMFT increases, the standard deviation also increases. The distribution of caries experience widens within the population as the mean increases. The change in caries risk is not equal for a given population: for some members of the population, the increase is greater than in others [Batchelor and Sheiham, 2002]. If the defined relationship between the mean and frequency distribution can be established, individuals responsible for strategy selection would be able to calculate the proportion of the population with no or low caries levels and those with high levels. Knutson [1958] used such an approach to estimate the mean caries levels in a population from estimates of the percentage of subjects who were caries free. For a given caries prevalence, the mean is relatively constant. The distribution of dental caries within a population has a relationship that can be described mathematically. The relationship between the variance and the mean DMF is that as the mean increases, the variance also increases [Batchelor, 1998]. This indicates that when the prevalence of caries increases, everybody increases their risk of developing caries. Batchelor and Sheiham [2002] found that there is a remarkable similarity in mathematical formulae describing the relationship between the mean caries score and its associated standard deviation for 12- and 14-year-olds. For the 12-year-olds, the formula is $y = 1.53 \cdot x^{0.55}$, where y is the standard deviation, x the mean DMFT score, and for the 14-year-olds, $y = 1.64 \cdot x^{0.57}$ [Batchelor and Sheiham, 2002]. The results demonstrate that there is a clear relationship between caries prevalence and mean DMFT score and between the mean score and the associated variance. These distributive laws apply to all populations and do not appear to have changed over time. For example, given the relationships outlined, the distribution of caries in a population where 80% of the caries occurs in 20% of a population, the mean DMFS will be about 1.0.

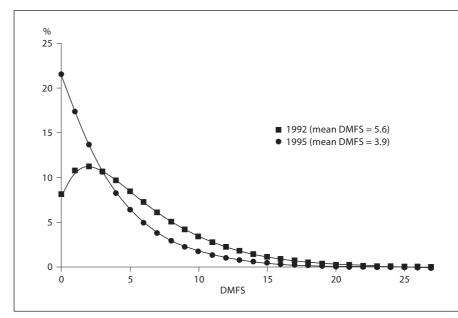


Fig. 5. Distribution of 15-year-olds with different levels of DMFS, in Jyväskylä, Finland, in 1992 and 1995 [Seppä et al., 2000].

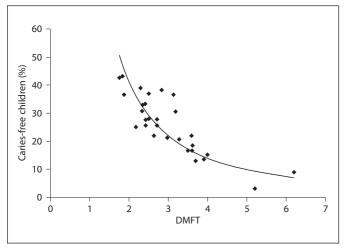


Fig. 6. Correlation between DMFT and percentage of caries-free 12-year-old children in São Paulo, Brazil [Cypriano et al., 2008].

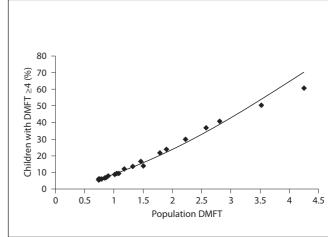


Fig. 7. Trends in DMFT of 11- to 12-year-old Australian children from 1997 to 2002 [Armfield et al., 2009].

[2000] reported that DFT scores of 6.5, 16.0, 15.4, 17.8, 16.2 and 16.5 corresponded to DFS scores of 11.2, 38.9,

There is a standard relation between the mean DMFS and DMFT. Knutson [1958] demonstrated that there was a mathematical relationship between DMFT and DMFS. Järvinen [1983] developed Knutson's concept by analysing the relationship between age-specific DMFT and DMFS. Others have demonstrated the relationship between DMFT and DMFS. In Norway, Holst and Schuller

38.5, 55.5, 52.1 and 52.9, respectively. In Sweden, at the ages of 12, 15, 19, 22 and 26-27 years, DFT scores of 3.1, 4.7, 6.0, 6.7 and 6.9 corresponded to DFS scores of 3.5, 5.8, 8.1, 9.6 and 10.0 [Mejare et al., 2004]. Applying the above-mentioned findings to the rela-

tion between DMFT and DMFS, from knowing that the DMFT is 3.0 at 12 years of age, the DMFS is likely to be

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5.6, 3.3 on occlusal, 0.94 on proximal and 1.6 on smooth surfaces and the percent being caries free 14%. The DMFT at 15 years for that cohort will be 5.2, the DMFS 9.76 and 3% would be caries free. At the age of 6, the mean DMFT would have been 0.2 and DFS for occlusal 0.3 and smooth surfaces 0.1; 85% would be caries free. Moreover, because of the stability of the relationship between DMFT and DMFS, there is no need to record caries by tooth surface in surveys as the DMFS can be calculated from the DMFT.

There Is a Hierarchy of Caries Risk by Tooth Type and Tooth Sites and Groupings of Teeth of Similar Caries Susceptibility

There is a hierarchy of caries susceptibility by tooth type and tooth surface within a mouth [Massler et al., 1954; Marthaler, 1966]. Different teeth and tooth surfaces are affected differentially at different mean levels of caries [Marthaler, 1966; Poulsen and Horowitz, 1974]. The most caries-susceptible sites are pit and fissured surfaces followed by approximal surfaces on posterior teeth. The least susceptible are the approximal surfaces on anterior teeth. McDonald and Sheiham [1992] took this argument a step further. They proposed that there was a relationship between vulnerability of differing sites and overall dental caries severity. Furthermore, this relationship was independent of the presence of fluoride. A working rule of caries is that as caries in populations declines, caries in the least susceptible surfaces (proximal and smooth surfaces) decreases considerably more than in the most susceptible surfaces (pits and fissures) [McDonald and Sheiham, 1992]. Batchelor and Sheiham [2004] confirmed that the most susceptible tooth surfaces to decay are occlusal surfaces of first molars and buccal pits of lower first molars. If all the first molars have caries, then there is a high probability that the second molars will be affected. The occlusal surfaces of the second molars and the buccal surfaces of the second lower molars are the second most susceptible sites for caries. At higher DMFS, the mesial proximal surfaces on the upper molars are the next sites to be affected, then the lower proximal surfaces. These are followed by the occlusal surfaces of the first premolars and proximal surfaces of first molars, then the occlusal surfaces of second premolars and the proximal surfaces of second molars. At about DMFS 15, the occlusal surfaces of the second premolars are attacked and then the upper first premolars. At higher levels of caries, all surfaces of canines, smooth surfaces of premolars and

incisors are affected. Examples from Switzerland and Brazil illustrate the hierarchy of caries susceptibility. Marthaler [1966] showed the caries attack levels for different predilection sites for 15-year-old Swiss children at various mean DMFTs, by tooth site type. At DMFT 6.2, the attack pattern was 6.0 in pits and fissures, 3.2 in proximal, 0.2 in smooth surfaces in molars and 0.35 in anterior teeth. At DMFT 2.2, the sites affected were 2.2 pits and fissures, 0.5 proximal, 0.07 smooth surfaces in molars and 0.10 in anterior teeth, respectively; only 2.9 of the 92 caries predilection sites were affected at DMFT of 2.2. The idea that the mouth could be zoned, so enabling a more efficient data collection method, was also suggested by Viegas [1969], Grainger [1967], Poulsen and Horowitz [1974] and Hannigan et al. [2000]. Further examples of affected teeth at different DMFT levels among 12-yearolds are: at DMFT = 1.23, tooth 46; DMFT = 1.6, teeth 46 + 36; DMFT = 1.62, teeth 46 + 36 + 16; DMFT = 1.67, teeth 46 + 36 + 16 + 26; DMFT = 1.74, teeth 46 + 36 + 16 + 26 + 37; DMFT = 1.81, teeth 46 + 36 + 16 + 26 + 37 + 47; DMFT = 1.73, teeth 46 + 36 + 16 + 26 + 37 + 47 + 14[Pereira et al., 2009]. Broadbent et al. [2006] examined the caries patterns and tooth loss in the Dunedin cohort for adults between the ages of 26 and 32 years with a DMFS of 13.9 at the age of 26 and 17.7 at the age of 32. Most of the caries occurred in the lower first molars and lower second molars, upper molars and upper second premolars. There was little caries in anterior mandibular teeth and moderate levels in anterior maxillary teeth.

Another feature of caries attack patterns is a left:right side symmetry; the risk of attack is similar for the left and right sides of the mouth [Berman and Slack, 1972; Poulsen and Horowitz, 1974; Hujoel et al., 1994]. There is also a degree of symmetry in the risk of caries both between the upper and lower jaws in the posterior sextants [Berman and Slack, 1972]. For the anterior sextants, the upper teeth are more prone to attack than the lower teeth. Symmetry in caries susceptibility is so well accepted that some dental caries examining systems recommend examining one side of the mouth and then doubling the score to give the total DMFS [Marthaler, 1966]. Marthaler [1966] used a hierarchical method of assessing dental caries severity that reflects the overall caries levels. That saves time compared to full mouth examinations.

The fact that caries occurs bilaterally in the same type of tooth suggests that any decline or increase in caries in individuals occurs at least in steps of two. If the left tooth is affected, so will the right. So changes in caries levels are stepped; they increase or decrease in pairs [Batchelor and Sheiham, 2004]. As there are standard patterns of caries, the mean DFS for occlusal, proximal, buccal and lingual, pit and fissure or smooth surfaces can be predicted for each DMFS level [McDonald and Sheiham, 1992]. Another phenomenon is that for different levels of DMFS there is a change in the ratio of smooth to pit and fissured surface caries [Burt, 1985; Dummer et al., 1990; Vehkalahti et al., 1991]. For example, at a DMFS of 1, the ratio of pit and fissured to smooth or proximal surfaces is 99:1. At a DMFS of 10, the ratio had changed to 3:1.

Another pattern of caries within mouths is that groups of teeth and tooth sites have similar propensities to caries. Changes in mean DMFT are not linear but stepped [Batchelor and Sheiham, 2004]. This stepped pattern of caries change suggests that there are groupings of teeth and surfaces that have similar levels of 'resistance' to caries. When resistance in one surface in a group is increased, for example by fluoride, then all surfaces within that resistance group with similar 'resistance' levels will also benefit and not go carious.

The groups, in order of susceptibility, are: (1) bilateral occlusal surfaces of first molars and buccal pits of lower first molars; (2) bilateral occlusal surfaces of second molars and buccal surfaces of lower second molars and occlusal surfaces of all second premolars on both sides of the mouth; (3) bilateral occlusal surfaces of first premolars, palatal surfaces of upper lateral incisors, proximal surfaces of first molars, lingual surfaces of lower first molars and buccal surfaces of upper first molars and palatal surfaces of upper second molars; (4) all proximal surfaces of second premolars, all proximal surfaces of upper first premolars, mesial and lingual surfaces of lower second molars and distal and buccal surfaces of upper second molars, proximal surfaces of upper central incisors, some proximal surfaces of upper and lower lateral incisors, all proximal surfaces of lower central incisors and distal proximal surfaces of upper canines, and proximal surfaces of second molars; (5) all surfaces of lower canines, buccal and mesial and labial aspects of upper canines, all smooth and proximal surfaces of lower first premolars, smooth surfaces of lower central incisors, proximal surfaces of lateral incisors; such groupings by resistance may explain the rapid stepped rates of decline of caries in the past 30 years [Batchelor and Sheiham, 2004].

A major reason for the rapid decline in caries since the 1970s can be explained by the fact that there are groupings of teeth or tooth sites having a similar propensity to decay. With an increased resistance to caries from fluoride, or a reduction in the challenge from sugars, all teeth in the group are affected.

Rates of Progression of Demineralization through Enamel and Dentine Vary by DMFT Levels

The rate of progression of caries through the enamel and into dentine is of fundamental importance for deciding on the frequency of dental recalls. The majority of proximal lesions in permanent teeth progress slowly and often regress, with an average proximal lesion taking at least 3 years to progress through enamel to dentine [Pitts, 1983]. The rate of progression of dental caries in the proximal surfaces varies by DMFT levels; the lower the DMFT levels, the slower the rate [Mejare et al., 1999, 2004; Lith et al., 2002]. When the DMFT is low, the rates of progression are slow whilst they are more rapid in higher DMFT populations. The speed of progression decreases when adolescents become young adults [Mejare et al., 2004]. This was illustrated in a longitudinal radiographic study in Denmark. At the age of 12, the mean DMFS was 1. Of all the examined occlusal surfaces, 93% had the same diagnosis at the age of 14 and 16 years. Out of the sound occlusal surfaces at the age of 14, 1.2% developed caries in outer dentine and 5.1 were filled. Of the occlusal surfaces that had caries in outer dentine at the age of 14, 36.7% had the same diagnosis, and the remaining were filled. Of all the examined proximal surfaces, 86% maintained the same diagnosis. At the second examination, of the sound approximal surfaces, 8.9% had caries in enamel, 1.4% caries in outer dentine, 0.02% caries in inner dentine and 0.5% were filled. At the second examination, out of the approximal surfaces that had caries in enamel at the age of 14, 4.6% were sound, 64% had the same diagnosis of caries in enamel, 22% had caries in outer dentine and 8% were filled [Hintze, 1997].

In a Swedish population of 12-year-olds with a DMFT of 3.2, the rate of progression of caries in dentine was almost 4 times higher than progression from inner enamel to outer dentine. In the same population, 20.3 out of 100 surfaces at risk progressed to outer dentine every year. Of the proximal lesions that had reached the enamel-dentine border but revealed no radiolucency in dentine, 50% progressed into dentine within 3 years. Of the 50% that progressed into dentine, 20% did so in 1.2 years [Mejare et al., 1998, 1999]. In areas with water fluoridation, the progress rates of caries were slightly slower [Lawrence et al., 1997; Lith et al., 2002]. The distal surfaces of the lower first molar and upper second premolars are at higher risk for a relatively faster progression of caries from enamel to dentine [Mejare et al., 1999]. The slower rates of progression of caries appear to be related to cavitation of proximal surfaces. When rates were more rapid, cavitation occurred earlier in the caries process.

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Discussion

This review shows that there are predictable patterns of caries in permanent teeth. Whilst some of these patterns may be known to dental epidemiologists, they do not appear to be widely used by planners of dental services. No example of their use could be found in the available literature or reports following national dental surveys. One of the most important patterns is that caries levels follow track lines. So, if a caries level is known at one age, one can predict caries levels at later ages. The tracking of caries indicates that the present caries experience embodies the cumulative effects of all risk factors to which the individual has been exposed. On the basis of the tracking pattern, one can predict the mean caries at a later age and what the caries level was at an earlier age, from knowing what the mean of the group was at any particular age. Therefore, it is not surprising that caries experience is the best single predictor of future caries increment [Demers et al., 1990]. People with high caries rates at a young age are likely to develop much more caries. This simple universal phenomenon can be used to predict what the caries level for a particular group will be in say 5 or 10 or 20 years' time. Such information can be used to decide on appropriate treatment regimens and recall intervals as they reflect the severity of caries attack and the likelihood of new caries developing within different intervals and which teeth and sites will be affected [Tan et al., 2006]. For example, being able to predict future caries levels by age can inform planners when newly erupting teeth are likely to develop caries as the likelihood of caries developing months or years after eruption is related to the intensity of the caries attack. The trends in caries can also be used to evaluate the effects of treatment regimens. An important fact to bear in mind is that there is a relatively constant rate of new caries over the life course from childhood to middle age [Broadbent et al., 2008]. The Dunedin study highlighted that there was no apparent drop-off in the rate of increase in percent DMFS with increasing age indicating that most caries occurs after the age of 18 years. So caries susceptibility is not a childhood phenomenon [Broadbent et al., 2008]. Therefore caries-preventive measures are necessary in adults as well as children.

The finding that the frequency distribution varies by mean levels of DMFT is unsurprising as that phenomenon is based on characteristics of frequency distributions. Therefore one can estimate the percentage of cavity-free children and children at each level of DMFT within a population at different mean DMFT levels from the frequency distribution of each population mean DMFT. From a planning point of view, the rule that for a given DMFT or DMFS there is a specific pattern of distribution of caries within a population allows planners to deduce from the mean DMFT the percentage of caries-free individuals and the teeth and sites affected in persons with specific levels of caries. If one knows the mean DMFT, because the distribution at a given DMFT is standard for a given mean, one can estimate the percentage of children in a population with different levels of caries, from 0 to 32. A review of some recent publications suggests that as a reasonable rule one can say that at 12 years of age the percentage being caries free at DMF = 1 would be $\pm 60\%$, at DMF = 1.25, 50%, at DMF = 1.5, 40%, at DMF = 1.75, 35%, at DMF = 2.00, 30%, at DMF = 2.25, 30%, at DMF = 2.50, 25%, and at DMF = 3.00, 15-20%. These relationships between DMF and prevalence of caries-free children were observed in different populations and in adults. For example, Cypriano et al. [2008] used dental survey data from 29 cities to examine the relationship between DMFT and percentage of caries-free 12-year-old children. In the low-prevalence area 32% were caries free, and the DMFT was 2.3, in the moderate-prevalence area, 22% were caries free, and the DMFT was 3.36. In the highprevalence area, 6.9% were caries free, and the DMFT was 5.54 (fig. 6). These estimates can be extrapolated to 15-year-olds because there is a good relationship between caries levels at 12 and at other ages as shown in trend data. Among adults aged 26, with a DMFT score of 1.5 the prevalence of caries was 48% [Javali and Pandit, 2009].

Assessment of the posteruptive time when caries manifests itself is important as it would determine, for example, whether and when to place sealants. Bohannan [1983] and King et al. [1980] both reported a sustained dental caries attack rate that neither proves nor disproves the hypothesis that a tooth surface is more susceptible in a given posteruptive period. Recently, Broadbent et al. [2008] challenged 'the commonly held belief that childhood and adolescence are periods of special risk for caries and/or that caries immunity may be acquired during late adolescence or early adulthood'. They found a relatively constant rate of new caries. There was a linear relationship between rate of increase of percent DMFS with no drop-off in the rate of increase with increasing age from the ages of 5 to 32 years.

These conclusions highlight a number of very important issues when proposing the development of cariespreventive regimens. First, there is an order to the development of dental caries: certain sites and teeth are more prone to caries than others. At low DMF scores, it is the pit and fissured surfaces of molar teeth that will develop caries. Second, as the attacking force decreases, the effect may not be constant. A decline from high levels of caries would be easier to achieve than at lower levels of caries. For example, to obtain a reduction of 5 at a DMFS of 20 is easier than at a DMFS of 6. These issues influence the selection of preventive agents and strategies to be used in a scientific approach to the prevention of dental caries.

Dental epidemiologists know that there is a hierarchy of caries risk by tooth type and tooth sites in permanent teeth. What is not generally known is that there are groupings of teeth of similar caries susceptibility. Batchelor [1998], using large data sets from studies done in the USA and UK, showed that there were groupings of teeth and tooth sites by susceptibility to caries. The practical implication of that finding is that if the groupings by susceptibility are known and one tooth site in that grouping develops caries, then, in the absence of any effective preventive measure, it is highly likely that over time all the other sites in that grouping will develop caries.

Dental planners are very fortunate to have abundant data on caries patterns that can be used to plan the types of dental services needed and the details of what preventive measures to apply for given populations based upon their mean DMFT levels. The data have not been collated in an accessible form until now. In this review evidence has been presented that the patterns of caries in permanent teeth in children and adults can be modelled because they display what can be considered rules. Much time and effort can be saved by using the rules outlined here. By using these rules much more use can be made of existing epidemiological caries data without resorting to large costly cross-sectional surveys. Moreover, future population treatment needs can be estimated and preventive measures evaluated by assessing to what extent they 'bend the trend lines' and the patterns of teeth and tooth sites affected by caries.

In summary, from knowing the mean DMFT level for a particular age group, the mean caries levels at later ages can be predicted because caries levels for groups follow predictable track lines. Moreover, the percentages of cavity-free people and with different levels of caries can be calculated because for a given mean DMFT there is a standard frequency distribution. Added to that, one can predict which teeth will manifest caries at a DMFT level for those at the different caries levels as there is a hierarchy by tooth type of caries susceptibility. Last, but not least, because there is a relationship between DMFT and the rate of progression of caries through enamel and the time after eruption that caries is likely to be initiated, planners can vary the length of recalls based on the groups' DMFT. Based on the patterns of caries and rules outlined here, computer programs and spreadsheets can be developed that will facilitate the applications of the copious epidemiological information available and thereby save much time and resources.

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