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A brief introduction to oral diseases: caries, periodontal disease, and oral cancer

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Introduction

By the late 1990s, treating dental disorders cost more than it did to treat mental disorders, digestive disorders, respiratory diseases, and cancer, at least in Canada (Leake 2006). The only group of disorders that exceeded dental treatment in terms of direct cost of illness was cardiovascular disorders (Leake 2006). In dealing with disease, "prevention is better than a cure." Dental disorders are an enormous burden to society, especially when one now considers the connection between poor oral health and systemic illness, which is a topic that is becoming increasingly important and a focus of other scholarly books. Papananou and Behle (2009) describe the mechanisms linking periodontitis to systemic disease. Dentistry in the past has been treatment oriented, but we are witnessing an unprecedented interest in prevention. It is obviously better to prevent the disease in the first place, than treat it once it has taken hold. This is quite true for most diseases in medicine.

The three general disease categories of focus in dentistry are dental decay, periodontal disease, and oral cancer. In the case of oral cancer, associated with a high degree of mortality, preventive dentistry even saves lives. Figure 1-1 summarizes the general hierarchy of prevention in dentistry.

The goals of preventive dentistry are to avoid disease altogether. Maintaining a disease-free state (green) can result from primary prevention. When lifestyle changes are made early on, the risk for developing dental disease are minimized. Secondary prevention and early intervention (yellow) can be used to reverse the initiation of disease. An outcome of good health can still be achieved when incipient enamel lesions are reversed before cavities form, when gingivitis is reversed before periodontitis sets in, when dysplasia is found and excised before cancer develops, thus returning to good health and controlling dental disease is possible. Far too often though, dentists spend most of their time treating dental disease in an endless cycle of repeat restorations and surgery (red), which leads to tooth loss, and in the case of cancer, disfigurement and even death.

No one would disagree that it would be better to maintain oral health throughout life, never to have had any kind of dental disease. This is the goal of primary prevention (green area in Figure 1-1). Throughout the book we have used a 'traffic light' color system: "green is good," "yellow means caution," and red means "stop! fix the problem." A similar theme has been used commercially in buffering capacity tests and in risk assessment (Ngo and Gaffney 2005).

Primary prevention for dental diseases such as dental caries and periodontal disease could include eating a healthy diet, maintaining low intake of fermentable carbohydrates, practicing meticulous oral hygiene throughout life, and reducing the other risk factors, such as smoking, that would normally lead to dental disease. In the case of oral cancer, primary prevention might include successful smoking cessation counseling, where a patient has been smoking for quite some time. Obviously it would be better for the patient not to have smoked at all.

Secondary prevention ('caution') suggests that the disease has started but can be reversed, and good health can still be achieved. For example, incipient carious lesions (white spot enamel lesions) can be arrested and reversed using appropriate 'preventive' measures so that

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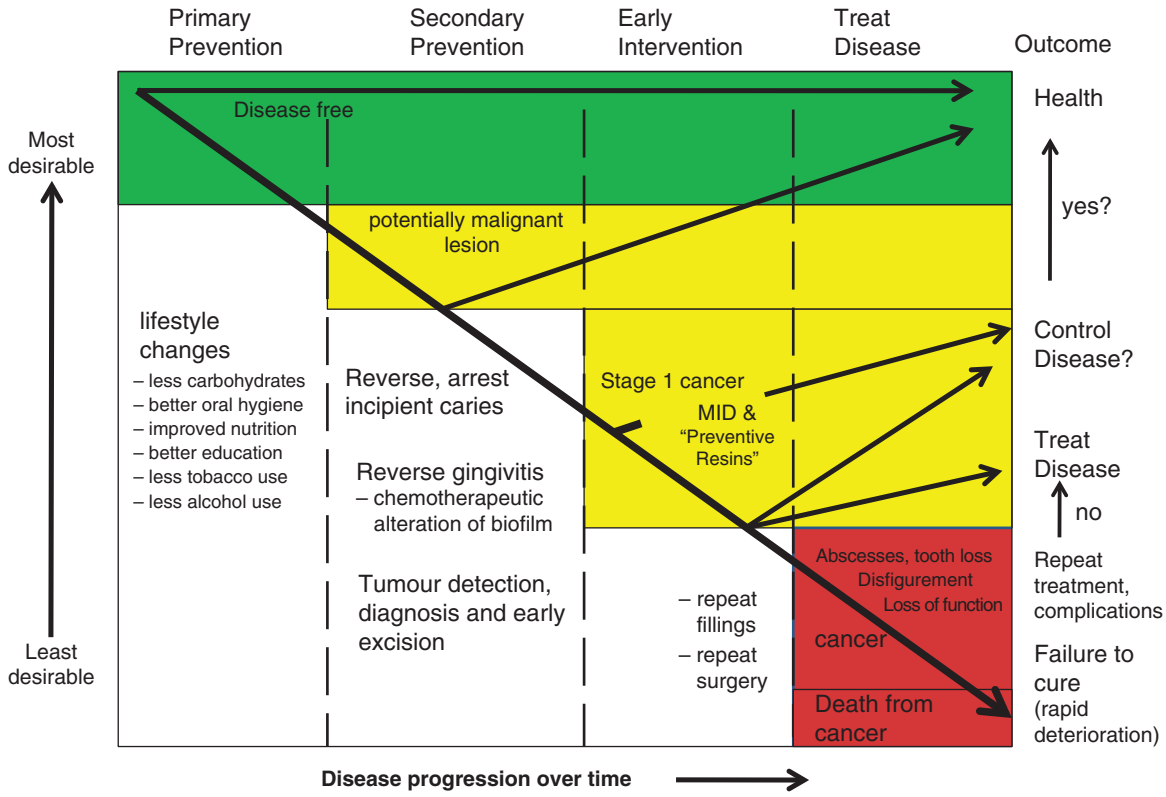


Figure 1-1 A hierarchy of prevention and treatment of oral diseases.

a full-blown carious lesion never develops. It was well established that frequent oral hygiene reinforcement by dental professionals can prevent caries, gingivitis, and periodontal disease (Axelsson and Lindhe 1978).

Secondary periodontal disease prevention might include other strategies such as the chemical elimination of bacteria known to initiate periodontal disease. Secondary prevention of oral cancer could include identification of dysplastic tissue and its removal as well as stopping the irritation that leads to the dysplasia.

It will be obvious to the reader that this book has attempted to be all-inclusive: a comprehensive text on prevention of oral diseases. Despite this ambitious goal, there is a heavy concentration and discussion around dental decay. It is important to realize that the literature on prevention of caries is quite extensive, compared to the prevention of periodontitis or oral cancer. If the reader is interested in the *treatment* of periodontal disease and oral cancer, it is suggested that the reader turn to more comprehensive reading material on the management of these diseases once they have developed. For example, two resources that are excellent reading material are books by Dibart and Ditrich (2009) and Tobia and Hochhauser (2010). Nevertheless, at least some approaches that have been successful in preventing periodontal disease and oral cancers are reviewed in this text.

The global burden of oral diseases

The World Health Organization's definition of health is "a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity" (World Health Organization 1946). One of the true indicators of a good quality of life, of true physical, mental, and social well-being, includes being in good general health. Oral health is an integral part of good general health. Unfortunately, it is the poor and socially disadvantaged that carry most of the burden of poor oral health (Karim *et al.* 2008). Table 1-1 summarizes some of the general overall risk factors known to be associated with oral diseases, the diseases that result, and their consequences.

People in poverty in developing counties face an overwhelming burden of chronic and severe caries, periodontal disease, tooth loss, oral cancer, and other oral disorders. These have detrimental effects on health and create negative behavioral situations that simply contribute more to the cycle of social deprivation. Perhaps only by improving the socio-economic status, education and literacy, oral education, and access to affordable dental care can the cycle of poor oral health be broken.

In most developing countries there are relatively few organized public health programs. If they exist, there

Table 1-1 Risk factors associated with oral diseases and their consequences

Risk factors	Oral disease	Consequences
• Malnutrition	• Rampant dental decay • Periodontal disease	• Pain and tooth loss • Compromised chewing with further nutritional deficiencies • Social isolation
• Poor oral hygiene • Lack of dental care	• Dental caries • Periodontal disease	• Pain and tooth loss • Compromised chewing with further nutritional deficiencies • Social isolation
• Poor quality drinking water	• Disturbed tooth development (e.g., fluorosis)	• Mottled teeth • Social isolation
• Tobacco products and alcohol in excess	• Caries in children • Periodontal disease • Oral cancer	• Pain and tooth loss • Disfigurement • Death
• Poverty • Illiteracy • Lack of access to dental care • Serious systemic illnesses (e.g., HIV AIDS)	• Dental caries • Periodontal disease • Oral cancer • Oral infections • Oral cancer	• Pain and tooth loss • Continued social isolation, poverty • Inability to thrive • Death

is an uneven distribution of these dental services (concentration of dentists in urban centers) and a lack of modern dental services. Clearly, as poor nations begin to improve their standard of living, they will be able to afford to spend money on dental disease prevention.

Dental decay (dental caries): global patterns

Most countries have seen a dramatic decline in oral diseases and are entering the new millennium with less oral disease to manage than in the previous century. Figure 1-2 shows how the prevalence of caries has changed over the decades. In nearly every developed country, there has been a steady decline in dental decay. It is interesting to note, however, that there was a period of extreme shortage of sugar during World War II resulting in an almost elimination of caries. As the supply of sugar returned, so did the caries. This observation was made not only in Japan but also Norway. The decline of caries started many years before the introduction of fluoride and may be related to numerous other factors, such as the introduction of penicillin, the increased use of sugar substitutes, and improved nutrition (which includes better access to calcium and Vitamin D). Experts believe, however, that it was primarily the introduction of fluoride therapies after the 1960s that had a huge impact on dental decay rates (Bratthall *et al.* 1996).

The reason for the decline in caries worldwide in most developed countries is multifactorial. Other factors may have had an influence on the caries rates. Sucrose has traditionally been used to make preserves of fruit when in season. The introduction of the electric refrigerator likely increased the consumption of fresh fruit and vegetables as well as fresh milk. Penicillin and Vitamin D-fortified milk were introduced during World War II (WWII). Both could have affected caries—penicillin, because it is effective against streptococci, and Vitamin D because its deficiency can lead to caries susceptibility, especially in those countries where there is little sunlight during the year (the northern countries). The first non-cariogenic sweetener (cyclamate) was introduced shortly after WWII, and then fluoridation and fluoridated toothpaste made their impact. The effects of fluoride were striking, according to researchers even today, but caries may have already been on the decline. Chlorhexidine, xylitol, and fissure sealants also had their role to play in reducing caries in the post-fluoride era. Separate chapters are dedicated to these agents in this book. The result is that cases of caries are now at an all time low throughout the developed world.

In 2003, Dr. Poul Erik Petersen, Responsible Officer for Oral Health, World Health Organization (WHO) in Geneva, reported on the oral health status of nations worldwide (Petersen 2003). The distribution of dental

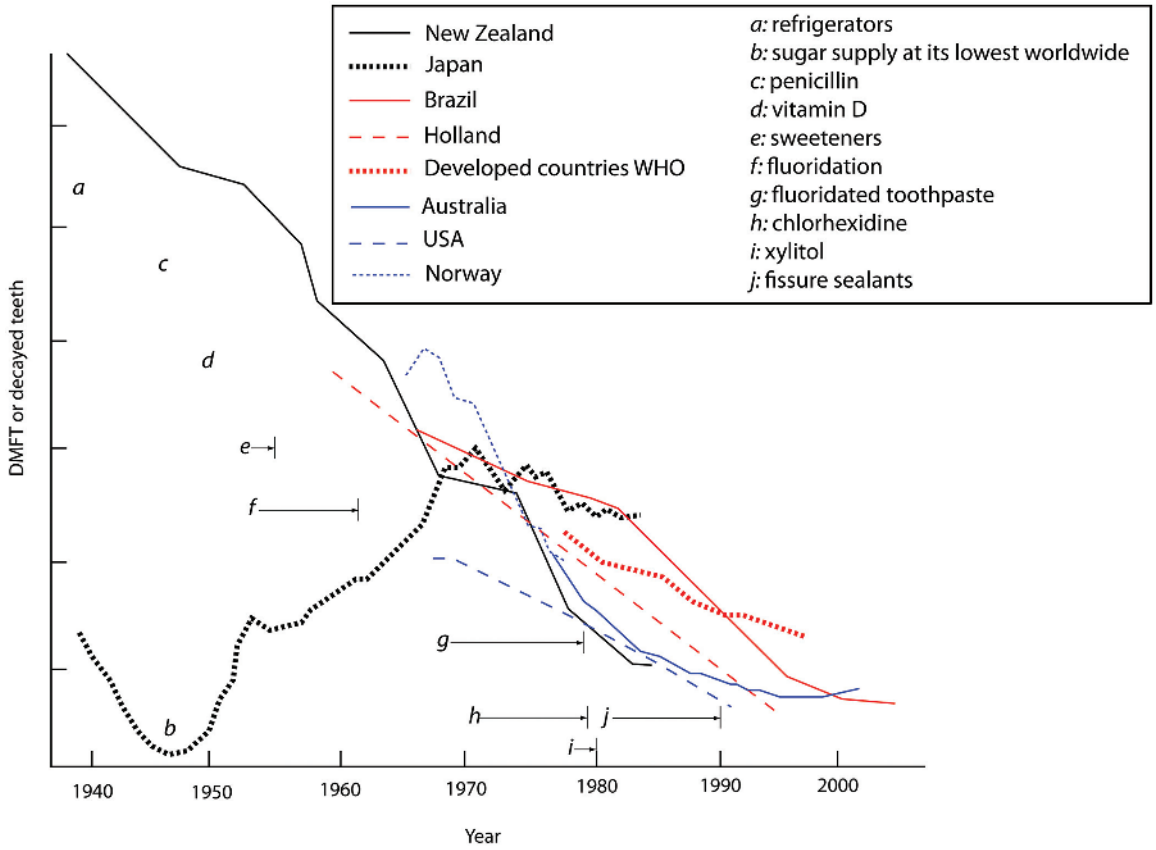


Figure 1-2 Global prevalence of caries from World War II to present. The relative (not to scale) decline in caries, represented by reported caries or DMFT (decayed, missing, filled teeth) are compared in this diagram from the start of World War II to the end of the twentieth century (each country, or countries, represented by different lines as shown). Also labeled are other factors that have contributed to the decline in caries worldwide (labeled **a** to **j**) and the approximate periods that they were introduced, represented by the horizontal arrows following the letters.
Sources:
New Zealand: Colquhoun (1997)
Japan: Miyazaki and Morimoto (1996)
Brazil: Cury *et al.* (2004)
Holland: Marthaler (2004)
Developed countries: The World Health Organization
Australia: Armfield and Spencer (2008)
USA: US Center for Disease Control
Norway: Von der Fehr and Haugejorden (1997)

decay throughout the world for children and adults is shown in a world map (Figure 1-3).

Despite this lowering of caries rates in children in most developed countries, the rate of edentulism remains quite high in the >65-year age group (Peterson 2003). The inevitable loss of teeth because of caries and periodontal disease is something that half of the population worldwide, on average, expects even today. The caries-free status of the younger population has been increasing however. As the younger population ages, dental professionals will witness a change in their dental practice profiles where their caries-free children, who grew up in the post-fluoride era, become adult and start raising another generation of children with very few caries. In the next 30 years, there will be at least two generations of adults where at least half of them are caries and filling free. The annual increment of caries in any given population and age group can be measured, and the projected caries for a certain age can be estimated. For example, in New Zealand, a cohort of children was followed from the time of their birth in 1972–1973, and their caries experience recorded until age 30 (Broadbent *et al.* 2008). This study appears to be the only dental study that followed a group from birth to adulthood. Based on the findings, one can conclude that, out of the 932 participants who consented to dental examinations from birth to age 32,

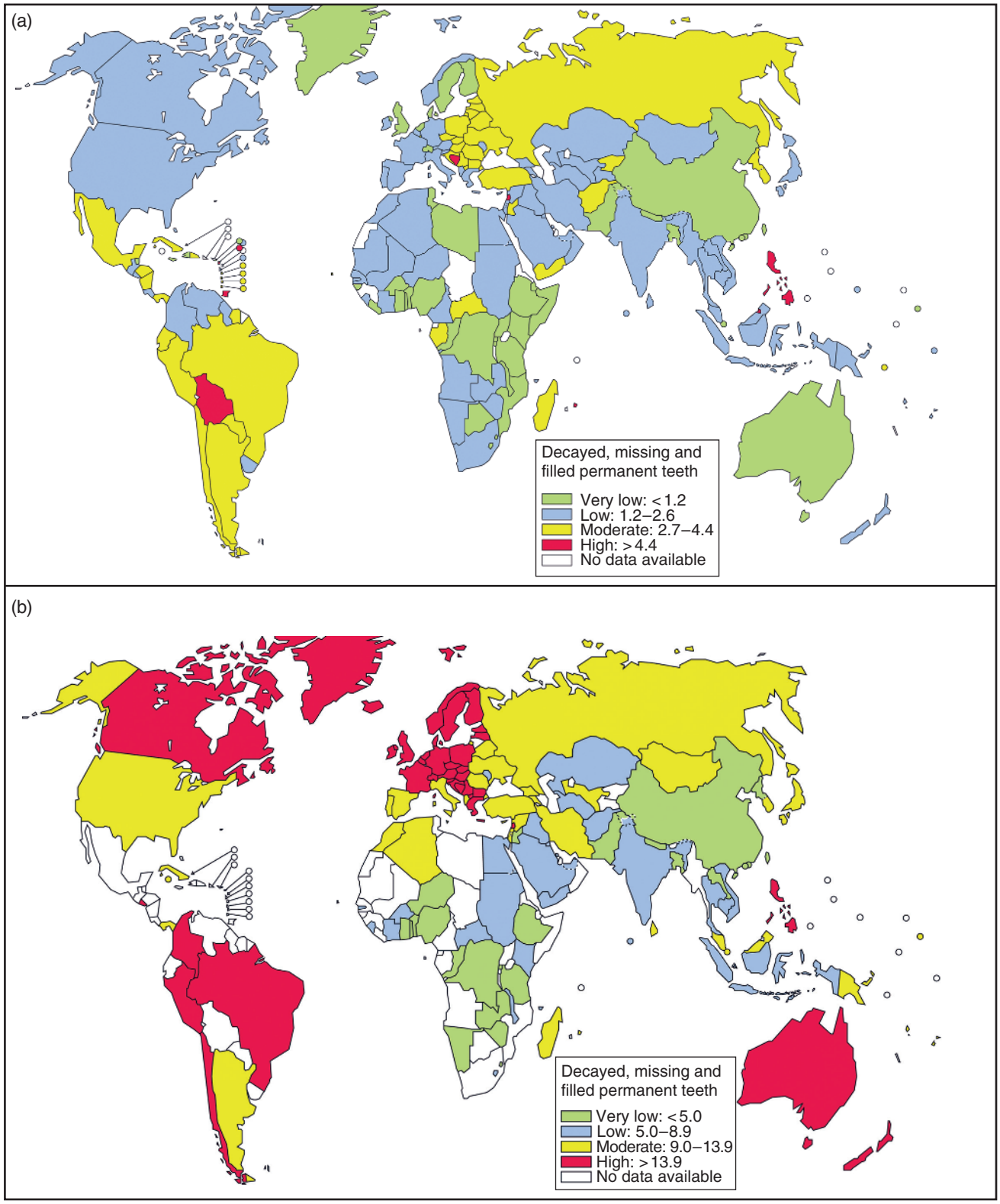


Figure 1-3 (a) Dental caries levels (DMFT) of 12-year-olds worldwide. (b) Dental caries levels (DMFT) of 35–44-year-olds worldwide. Reprinted from Petersen 2003, with permission from John Wiley & Sons, Inc.

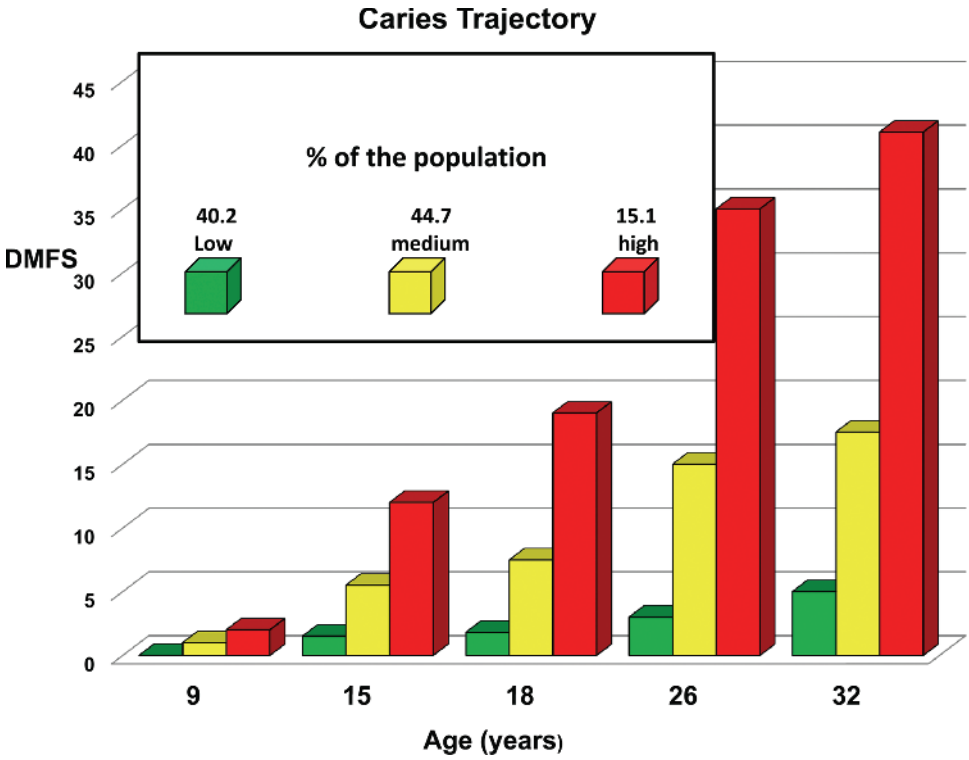


Figure 1-4 New Zealand caries incidence in a long-term clinical trial.

the trajectory of the caries is a linear one, with a minority of subjects (15.1%) experiencing a high increment of caries, 44.7% experiencing a moderate level of caries, and 40.2% experiencing a very low level of caries (Figure 1-4).

Nearly 1,000 patients were followed from birth to adulthood in this prospective clinical trial (no interventions). Approximately 15% of the population had the highest decay rates (red). The rest of the population was nearly equally divided between the low (green) and moderate (yellow) caries active patients.

By the end of the last century the edentulism rate from caries and periodontal disease was still high (see previous discussion). It is anticipated that as the current cohort of 30 year olds age toward their senior years, they can fully expect to keep their teeth. The rate of edentulism will decline dramatically as did the caries rates. Until that happens, a typical profile of the average dental office might include young adults with very little dental restorations and some older patients who have experienced extensive restorations and tooth loss (Figure 1-5).

Caries prevention: how far we have come in one century!

If one considers that the terminal stage of caries is the loss of a tooth, then early intervention (minimal intervention dentistry) is obviously desirable. When the disease has progressed significantly and more drastic measures are required (surgical intervention such as root canal therapy), one is still 'preventing' tooth loss. This was the goal in the early days of dentistry more than a century ago when Dr. G.V. Black proposed the "Extension for Prevention" concept during the restoration of teeth (see Figure 1-6) (Black 1875; Jokstad 1989).

It has taken over a century for dentistry to advance from the pioneering "extension for prevention" concepts proposed by Dr. G.V. Black. By removing a significant proportion of tooth structure so that only the easily cleansed tooth surfaces remained, there was a reduction in the need for further operative treatment. As dental decay rates began to fall worldwide in industrialized countries after WWII, a new concept of operative dentistry began to take hold. It is called minimal intervention dentistry, or MID (Mount and Ngo 2000).

Minimal intervention dentistry, as the term suggests, refers to a principle of treatment in dentistry in which early intervention minimizes tooth destruction because the disease is diagnosed prior to cavitation, and steps are taken to remineralize the enamel and arrest the decay. However, more than that, it is a whole philosophy of managing caries. Chalmers (2006) summed it up:

> "The main components of MID are assessment of the risk of disease, with a focus on early detection and prevention;

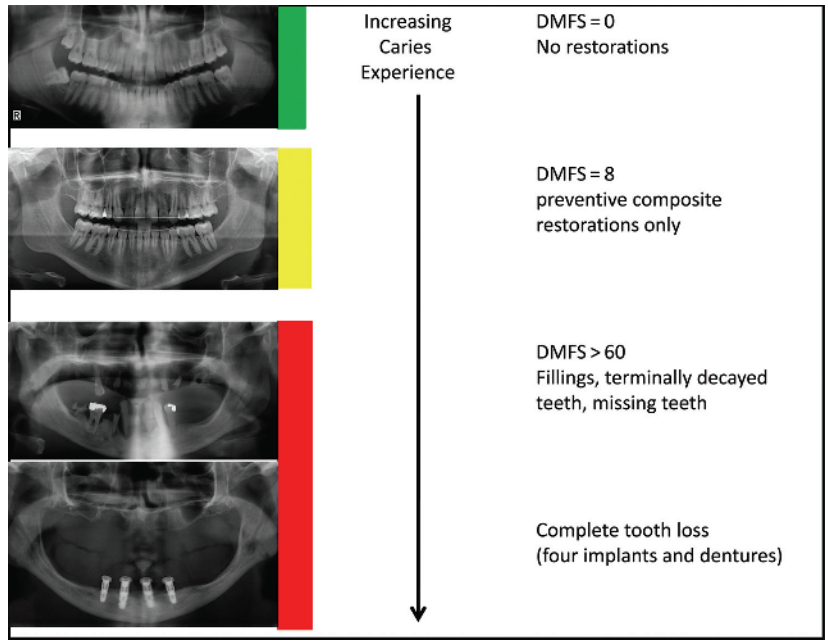
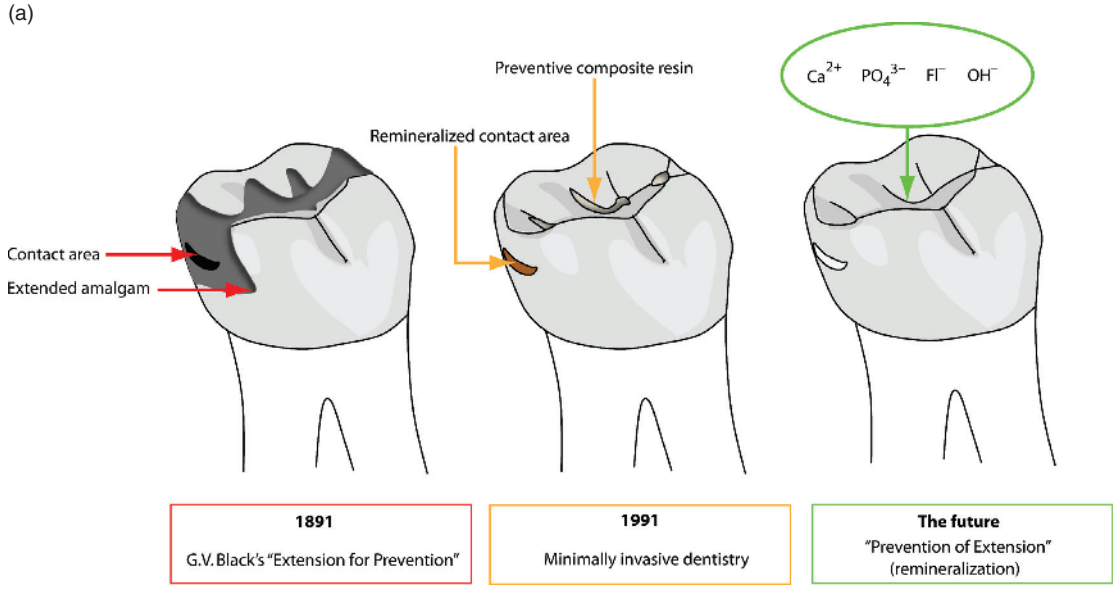


Figure 1-5 Radiographic profile of representative adult patients in a typical dental office in North America today. Most young adults will be either caries free (top, green) or have a select few fillings, many of them preventive resins, indicating a moderate risk for caries (middle, yellow). However, there is still a significant percentage of patients whose caries activity is extremely severe (red) leading to multiple root canals, extractions, and finally total tooth loss with teeth replaced with dentures or implant-supported prostheses (bottom). Radiographic images courtesy of Dr. Ray Voller of Pittsburgh, PA.

(a)



(b)

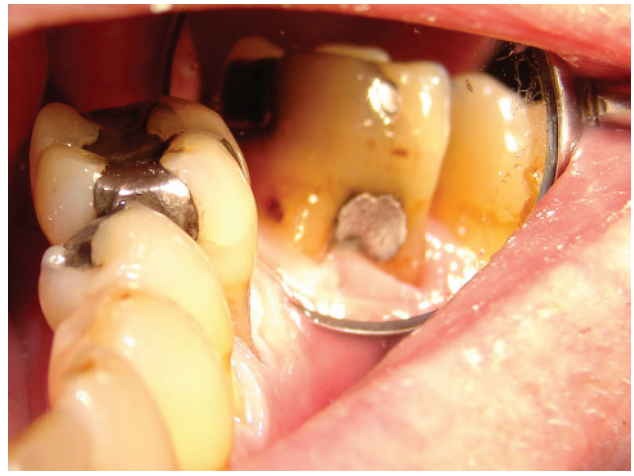


Figure 1-6 A century of caries prevention: (a) Illustration of the major changes in preventive dentistry. Left: G.V. Black's 'extension for prevention' showing a typical class II amalgam restoration. Middle: Minimally Invasive Dentistry 100 years later. Somewhat smaller restorations have been placed in a patient at moderate risk (yellow) for caries. Right: The latest concept in remineralization therapy in low-risk patients (green) ensures that the minerals are returned to the enamel before caries lesions start. (b) A clinical image of a typical class II amalgam restoration showing 'extension for prevention' as well as an amalgam restoration in the furcation area of the exposed root. Photo courtesy of Dr. Aaron Fenton, University of Toronto.

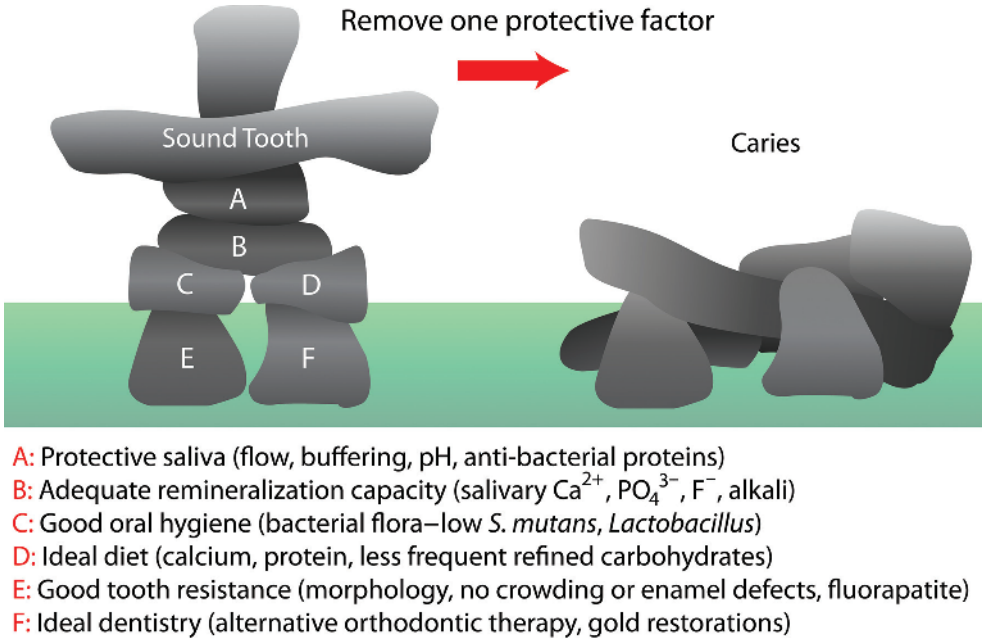


Figure 1-7 An illustration of how protective factors maintain sound tooth structure. This is an illustration of an Inuit Innunguat, a human figure made of stone by the native peoples who live in the arctic. The component rocks of the arctic stone figure are in delicate balance. Each protective factor (each stone) plays its part in keeping the human figure together. If any one of the parts is removed (loss of a protective factor), the structure collapses (caries results).

external and internal remineralization; use of a range of restorations, dental materials and equipment; and surgical intervention only when required and only after disease has been controlled."

Assessing caries risk can be done in several ways using many different approaches (see Chapter, Caries Risk Assessment). A popular approach is using Caries Management By Risk Assessment, or CAMBRA (Featherstone 2004) or Ngo and Gaffney's Traffic Light system (Ngo and Gaffney 2005), which has been adopted in this text.

A thorough analysis of patient history (social, medical, and dental), followed by a careful extra- and intraoral examination will provide the necessary background for assessing caries risk in order to determine the most appropriate preventive therapy. Changing dietary patterns, controlling the cariogenicity of the oral microflora, and providing a healthy environment for remineralization are primary goals of MID.

Humans have developed several defense mechanisms that are in balance with each other and protect teeth against damage. If any of these protective factors are disturbed, the balance is disturbed and caries will result.

In Figure 1-7 an Inuit Innunguat representing a human figure standing alone and precariously against the elements, illustrates how caries is in delicate balance. Each protective factor (each stone) plays its part in keeping the human figure (tooth) together. If any one of the parts is removed (loss of a protective factor), the structure collapses (caries results).

This is a convenient analogy to understand and is an offshoot of the classic Venn diagram (Figure 1-8) first introduced by Keyes (1962).

Caries results when all of the factors that contribute to caries overlap. One must have a tooth, plaque bacteria, fermentable carbohydrate, saliva, and enough time in order for a carious lesion to develop (red color, center). Several factors influencing each component, listed in the diagram, affect the rate and severity of the caries.

Dental professionals provide the initial care, reversing caries, but then they need to guide patients to maintain the good habits at home. A recognition that early enamel lesions can actually be arrested or reversed with various therapies (some practitioners go so far as to use the term 'healed'), has taken the MID concept to its ultimate level, where enamel, and even dentin, demineralization can be reversed with appropriate chemical therapy, resulting in carious lesions that have either been arrested or reversed. "Prevention before extension," a reversal of Dr. G.V. Black's idiom (Wesolowski 2008) has not yet found its way into the English dental literature, but it should be the goal of every dentist. Working together with the dental hygienist, prevention should be the primary focus of each dental office. Although in many dental offices, providing the preventive services is the sole responsibility of the dental hygiene team, 'prevention before extension' can only be achieved if the dentist

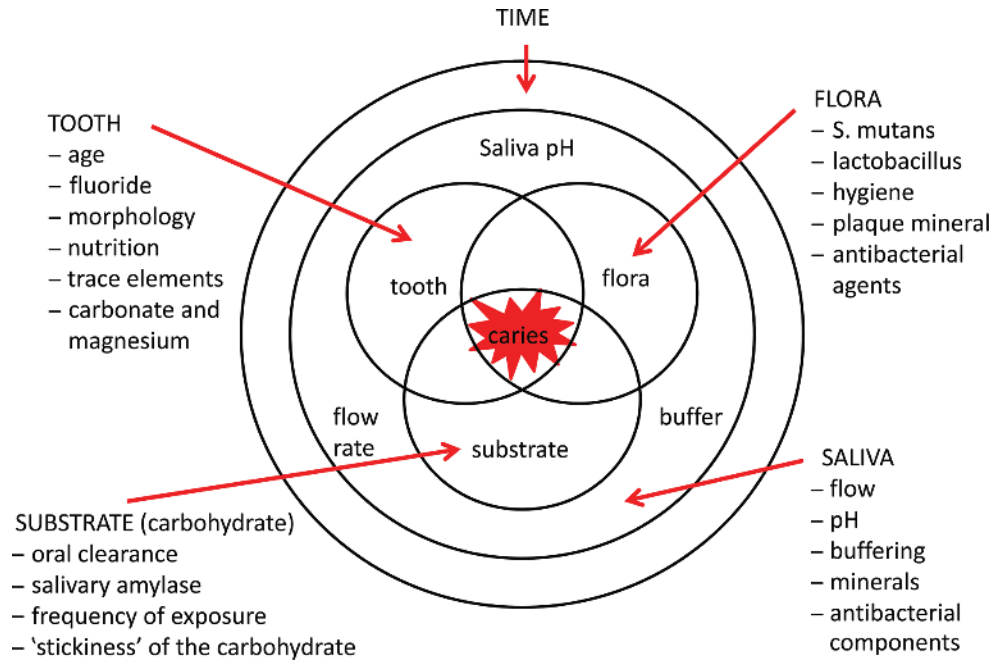


Figure 1-8 The classic Venne diagram of caries.

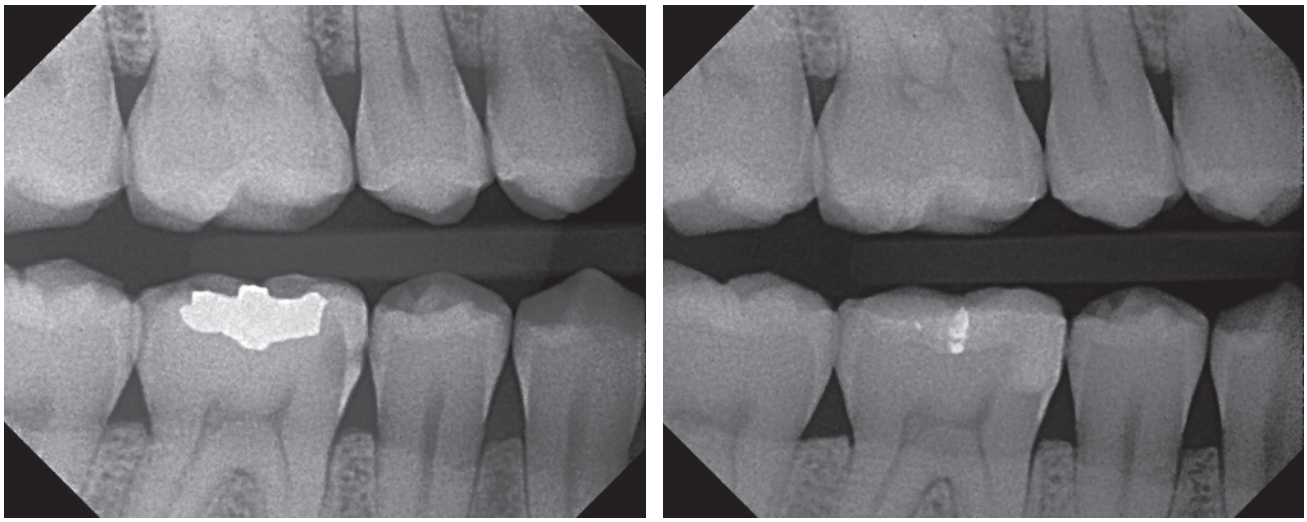


Figure 1-9 Caries as seen on bite wing radiographs. The left radiographic image shows a typical approximal carious lesion in the mandibular right first molar that has progressed into the dentin. The radiographic image of the same tooth on the right shows most of the previous amalgam removed and the caries treated with a posterior composite resin.

recognizes the value of providing this service. There is every reason to believe that, in modern times, each and every person should be able to expect to achieve a caries-free status.

An introduction to dental decay

Figure 1-9 shows a bitewing radiograph using a digital system identifying an interproximal carious lesion and the composite restoration that replaced the class I amalgam and repaired the carious dentin/enamel. Early carious lesions into enamel can be reversed. In this section we introduce the biochemistry and microbiology that leads to carious lesions.

Caries as an infectious disease

Dental caries does not occur in a sterile mouth. However, no mouth can ever be made sterile. The conditions in the oral cavity are ideal for the growth of bacteria that metabolize sugar to acids. The oral cavity is generally a warm place, at body temperature (37°C) encouraging the growth of bacteria.

Table 1-2 Salivary components and their role in caries

Classification of component	Ingredient	Function
Inorganic	Water (99%)	Dilutes and clears acid, wets teeth and mucosa, the vehicle for other ingredients
Inorganic, organic	Carbonate, phosphate, protein	Buffers acid
Organic	Amylase, lipase, protease, pyrophosphatase, lysozyme	Antibacterial
Organic	Mucins	Lubricant, calcium binding
Organic	IgA	Antibacterial

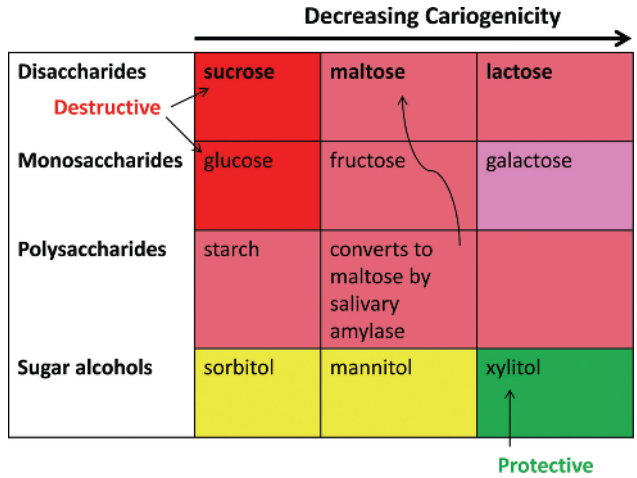


Figure 1-10 Cariogenic potential of carbohydrates. This chart summarizes the cariogenic potential (cariogenicity) of various carbohydrates. The sugars with the most cariogenicity are sucrose and glucose (red). Other carbohydrates (maltose, lactose, fructose, and starch) are less cariogenic. The sugar alcohols, such as sorbitol and mannitol, are the least cariogenic (yellow), and xylitol has even been shown to be anticariogenic (green).

Caries is an infectious disease that is actually transmissible, usually when the mother, infected with *S. mutans*, infects her infant when the child's first teeth appear in the oral cavity (Kulkarni *et al.* 1989). In fact, it has been shown that the caries rates of the offspring can be reduced if the parents' *S. mutans* are reduced and the child is not colonized by *S. mutans* until after age 2 (Isokangas *et al.* 2000).

The role of saliva

Saliva contains antibacterial proteins, electrolytes for remineralization but also the essential nutrients for bacteria to grow. However, it is the food that is ingested by the host that provides the dietary carbohydrates that are easily converted to energy and acids by the bacteria that leads to dissolution of dental hard tissues.

The main components of saliva and their function are shown in Table 1-2.

Because of its buffering capacity and ability to neutralize acids, a simple intervention such as stimulating the saliva with chewing gum can arrest white spot lesions and prevent cavities from forming (Stookey 2008).

The role of dietary sugars

Not all sugars are cariogenic. In a chart of cariogenicity (Figure 1-10), the more common dietary sugars are presented.

The disaccharide sucrose and the monosaccharide glucose, a component of sucrose, are most cariogenic and, with frequent ingestion, can cause severe damage to the tooth (Paes-Leme *et al.* 2006). Other dissacharides are less cariogenic, and the sugar alcohols are nearly neutral in their cariogenicity. Xylitol stands out as an anti-caries sugar, and more about this sugar will be discussed in Chapter 9.

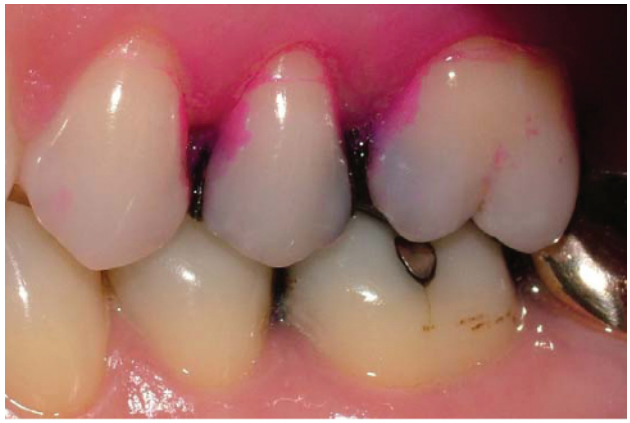
One of the strategies in prevention of caries is to limit access to the more cariogenic sugars and substitute them with the anti-cariogentic ones. As we saw in our discussion of the global patterns of caries, when the sugar supplies dried up during WWII, caries rates declined to almost nil. There is no question that carbohydrates are the main etiological reason for the development of caries. Not only does their conversion to acid result in enamel dissolution, but they also encourage the growth of more virulent cariogenic bacteria.

Plaque biofilms and their role in caries and periodontal disease

Biofilms responsible for caries and periodontal disease might occur in the same location (interproximally, at the margins of fillings, and at the gingival margins). The supragingival bacteria are dominated with streptococci and lactobacilli that can lower the plaque pH and induce decalcifications (white post lesions). Below the gingival margin and in the gingival sulcus, periodontal pathogens start to grow. They induce the formation of calculus and cause host immune responses that is initially inflammation, but as the bacteria migrate deeper into the periodontal pocket, the more virulent species cause host reactions that lead to the destruction of the periodontal attachment apparatus. In Figure 1-11, three different methods were used to visualize plaque.

(a)

(b)



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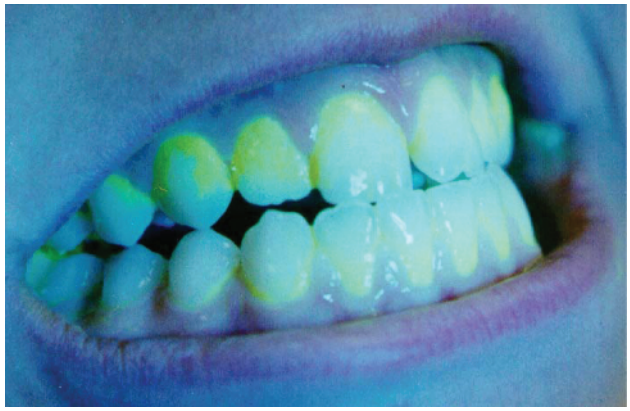

Figure 1-11 Three plaque disclosing methods: (a) 2-Tone. Plaque on a teenager revealed with Young's cherry-flavored 2-Tone Disclosing Solution. New plaque is stained red, and old plaque is stained blue to identify areas continually missed. (b) Red-Cote. Plaque revealed on adult teeth with Red Cote (Butler G.U.M.) disclosing solution. Chewable tablets produce the same effect. (c) Plak-Check. The plaque on this 16-year-old patient is revealed with Plak Check (Sunstar Butler G.U.M.), a sodium fluorescein dye, made more visible with a blue-filtered light source.

Disclosing agents such as 2-Tone (Young Dental Manufacturing) can reveal very thick, old plaque (blue color) and recently formed plaque (pink) (Pretty *et al.* 2005). Sodium fluorescein (Plak Lite, Butler) uses a blue filter and bright light to highlight plaque, which glows fluorescent yellow (Lang *et al.* 1972; Gillings 1977). With this technique there is no messy cleanup. The third method is the more common one and uses erythrosine dye (Butler Red Cote) (Gillings 1977). Erythrosine stains more plaque than sodium fluorescein (Gillings 1977).

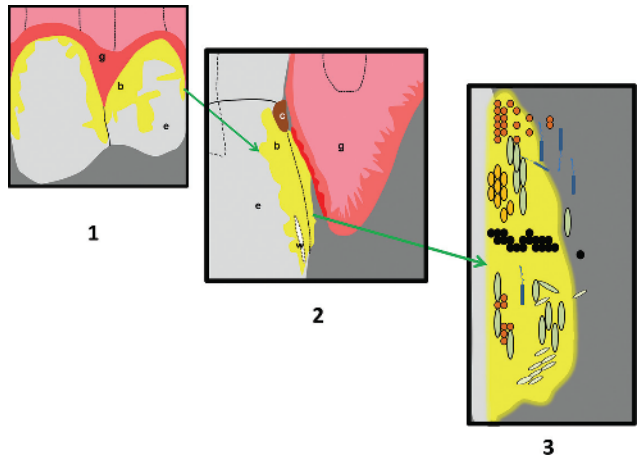

Figure 1-12 Illustration of plaque: (1) Plaque stained with sodium fluorescein: the enamel (e) has plaque biofilm (b) growing at the border of the inflamed gingival (g). (2) Same plaque as in 1 but a closer look. There is a 'white spot' lesion (w) developing at the margin of the gingiva, and brown calculus (c) developing in the sulcus attached to the tooth. (3) Close-up view of plaque. Sodium fluorescien not only stained the plaque biofilm bacteria, which consists of several species of bacteria (cocci, rods, motile spirochetes), but also the organic material (salivary proteins) and organic matter secreted by the bacteria (yellow-stained matter between bacteria depicted in 3).

Figure 1-12 shows an illustration of dental plaque at the gingival margin.

In this example, plaque is growing at the gingival margin attaching to the tooth surface and growing below the gingival margin as well (usually where tooth brushing is inefficient). The bacteria that are associated with caries differ from those that are associated with periodontal disease. The pathogens involved in periodontal disease are geographically located deep in the sulcus, and are different species with different metabolisms. The plaque that is responsible for caries is generally located supragingivally and is acidogenic.

Dental plaque is quite complex in composition and extremely dynamic (Marsh and Bradshaw 1999; Filoche *et al.* 2010). Early colonizers attach to the enamel pellicle, the salivary organic film that forms immediately on freshly cleaned enamel surface. This allows the attachment of other bacteria, and eventually several communities of bacteria form that adhere to each other and

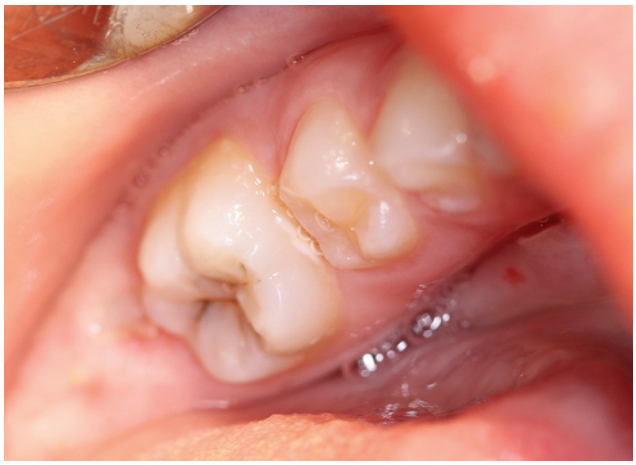

Figure 1-13 The enamel white spot lesion. This is a representative enamel white spot lesion at the mesial contact zone of the first maxillary right molar after exfoliation of the primary second molar. The premolar can be seen erupting into contact with the molar. These white-spot lesions are sometimes filled by dentists but can be remineralized.

interact with each other. People who consume sugars frequently in their diet increase the levels of streptococci and lactobacilli, the two bacteria species thought to be responsible for caries. These bacteria can be stimulated to grow in the right conditions, and they continue to thrive as the pH drops. If the plaque is not removed, eventually, the enamel starts to decalcify and an incipient 'white spot' lesion ensues, as shown in Figure 1-13.

The microflora associated with periodontal disease is really much more complicated, and researchers have been studying the virulent species for decades. It has been known for some time now which bacteria start to grow in unhealthy periodontal pockets. The main bacteria in health and disease are listed in Table 1-3, which shows a brief list of bacteria associated with dental disease. There are literally hundreds of microorganisms that are known to grow in unhealthy periodontal pockets (Listgarten 1994; Kumar *et al.* 2006).

Table 1-3 Bacterial species associated with dental disease

	Bacteria associated with health	Bacteria associated with disease
Caries	Normal flora	*S. mutans* and other low-pH streptococci (*Streptococcus oralis, Streptococcus mitis, Streptococcus anginosus*), *Rothia, Actinomyces, Lactobacilli Bifidobacterium* spp., *Candida albicans* Source: Filoche *et al.* 2010
Periodontal disease	*Streptococcus sanguis, Streptococcus mitis, Veillonella parvula, Actinomyces naeslundii, Actinomyces viscosus, Rothia dentocariosa.* Also *Veillonella* spp. oral clone X042 (Kumar *et al.* 2006), *Deferribacteres* clone W090, and clone BU063 from *Bacteroides, Atopobium rimae,* and *Atopobium parvulum*	*Porphyromonas gingivalis, Treponema denticola*
Gingivitis		*Actinomyces* species, *Streptococcus* species, *Veillonella* species, *Fasobacterium* species, *Treponema* species, *Prevotella intermedia*
Chronic periodontitis		*Treponema* species, *Prevotella intermedia Porphyromonas gingivalis, Candida* species, *Tannerella forsythia Peptostreptococcus micros, Campylobacter rectus, Aggregatibacter actinomycetemcomitans, Eikenella corrodens, Fusobacterium* species, *Selenomonas* species, *Eubacterium* species
Localized aggressive periodontitis		*Aggregatibacter actinomycetemcomitans*
Generalized aggressive periodontitis		*Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, Tannerella forsythia Campylobacter rectus, Eikenella corrodens*
Chronic/aggressive periodontitis		*Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, Prevotella intermedia, Tannerella forsythia Campylobacter rectus, Peptostreptococcus micros*

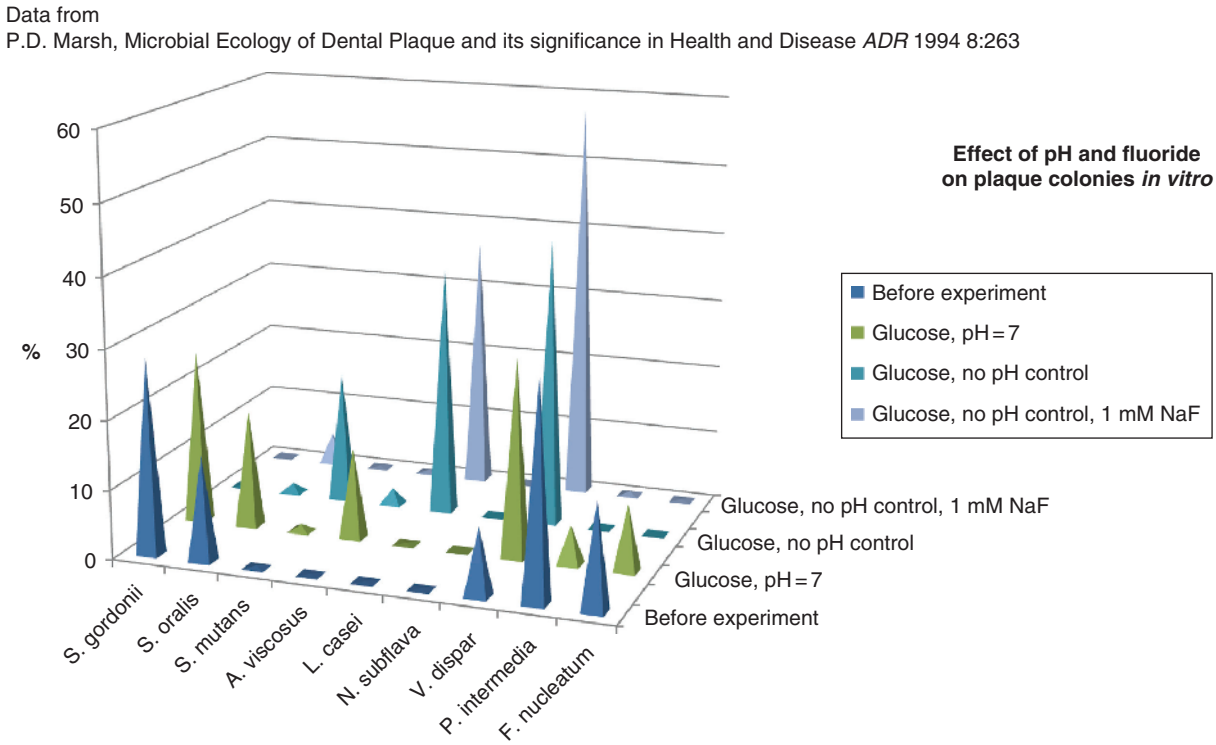

Figure 1-14 Changes in oral flora under controlled culture conditions.

In a series of elegant experiments, Marsh (1994) was able to show, at least in well-controlled chemostat cultures, that feeding mixtures of bacteria a meal of glucose can encourage the growth of cariogenic bacteria and suppress the growth of periodontal pathogens when the pH is allowed to drop (see Figure 1-14).

Nine different oral bacteria were cultured together in controlled conditions. Glucose rinses (second row) at neutral pH encouraged *A. viscosis* and *V. dispar* growth, but if the pH is not controlled and allowed to drop, the acidic conditions encourage the growth of *S. mutans* and *L. casei* but inhibit the growth of periodontal pathogens. There is an ecological shift to cariogenic flora. Fluoride at high concentrations inhibits SM but not LB.

In their experiments, the cultures were pulsed daily with glucose. To simulate a healthy mouth, the pH was maintained at neutral pH in some bacterial mixtures. In other mixtures the pH was allowed to fall as acid was produced from the glucose. As the pH dropped, the *S. mutans* were encouraged to grow. *In vivo, S. mutans* is able to secure sucrose and make an extra-cellular coat of glucan that favors its attachment to enamel and rapid growth. It can also tolerate low pH. *S. Mutans* thrives at low pH. The others don't do well at low pH. Thus, a cariogenic flora is encouraged to grow. Fluoride has to be at mM concentrations to significantly inhibit *S. mutans,* and it has no effect on lactobacilli. In separate experiments Marsh's group was able to show xylitol had inhibitory properties for both cariogenic and periodontal bacteria. These observations were made by other researchers as well (Ccahuana-Vasquez *et al.* 2007).

The demineralization–remineralization balance in caries

As plaque thickens *in vivo*, and becomes dominated by cariogenic bacteria, it can effectively keep the saliva from reaching the enamel surface. In addition, the more plaque there is, the more acid is produced. These acids have a longer time to penetrate into the enamel under thick biofilm. If the saliva reaches the acids they are washed away and neutralized by the salivary buffers. This allows the tooth to remineralize. The cycle repeats itself over and over with every sweet snack and meal containing fermentable sugars (Figure 1-15).

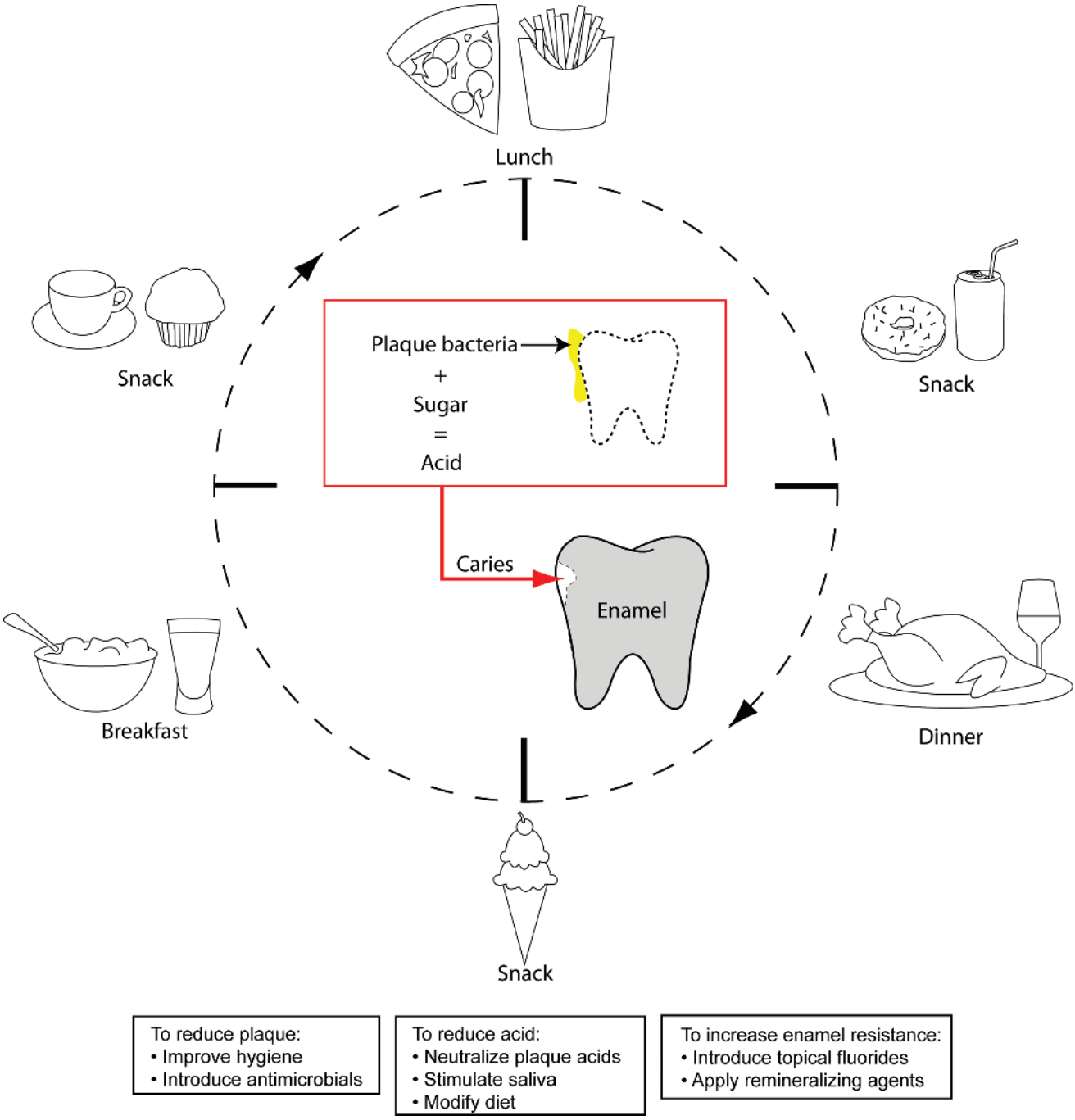

Figure 1-15 The repeated cycle of 'sugar attacks.'

Caries occurs when the frequency of sugar exposure during the day is high. There are many strategies in preventive dentistry to reduce the risk for caries from this frequent exposure to carbohydrates. One can limit how much plaque is on the tooth surface through better hygiene and antimicrobials, reduce plaque acids by introducing buffers, increase salivary flow, modify the diet (changing to less cariogenic foods), and increase the resistance of the tooth structure with topical fluorides and remineralizing agents.

Preventive interventions aim to modify the steps in the repeat demineralization and remineralization cycles.

1. Neutralize the plaque acids: This can be done by adding base or adding buffers such as sodium

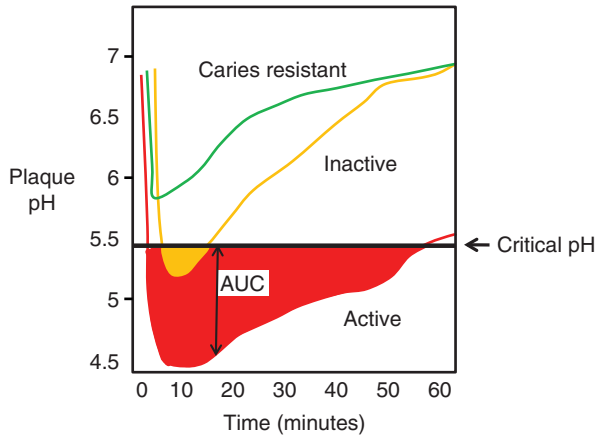

Figure 1-16 The classical Stephan Curve.

bicarbonate (baking soda) to the saliva to boost its ability to neutralize acids.

2. Improve hygiene: With bacterial levels low, less acid is produced. Also, plaque layers don't have a chance to grow thick; saliva can penetrate better to the enamel surface through thin layers of plaque.
3. Introduce antimicrobials: Since caries is a disease caused by bacteria, simply eliminating the bacteria or controlling their growth would go far to reduce the caries incidence. Chlorhexidine, xylitol, ozone, even experimental antibodies, have been used to control bacterial growth.
4. Stimulate saliva: Saliva contains numerous components that fight tooth decay (buffers, remineralizing minerals, antimicrobial enzymes, antibodies).
5. Topical fluorides: Fluoride added to the remineralizing incipient lesion increases the enamel crystals' resistance to dissolution by plaque acids.
6. Remineralizing strategies: Remineralization can be promoted with the use of calcium-phosphate complexes such and ACP-CPP.

The pH of dental plaque in response to glucose has been studied using the classic Stephan curve (Stephan and Miller 1943) (Figure 1-16).

This diagram illustrates the plaque pH response curves that have been obtained from patients with different risks for caries. A high-risk individual, when given a glucose rinse at time zero, will experience a dramatic drop in the plaque pH well below the critical pH of 5.5. The recovery to neutral pH in the high risk individual will be slow. The area under the pH-time curve (AUC) representing the time spend at pH lower than the critical pH is a better measure of total caries risk. The AUC for a high risk individual (red) will be very large. For a more moderate risk individual (yellow), the initial pH drop may only be a little lower than the critical pH, and the AUC will be much less. For a caries-resistant person (green), the initial pH drop of that person's plaque may not even reach the critical pH, and the recovery will be very quick.

In these experiments, the pH of plaque is monitored after a patient is given a glucose rinse. The degree to which the pH drops will depend on several factors and is governed by how quickly the acids are eliminated and neutralized. It can depend on how thick the plaque is and how deep the sugar penetrates into plaque. A theoretical model was even developed to demonstrate this (Dawes and Dibdin 1986). Some people are caries prone, others are caries resistant. In caries-resistant people, the pH drop in response to a rinse with glucose does not fall below pH 5.5, a pH thought to be a 'critical pH.' The concept of 'critical pH,' where there is net loss of calcium and phosphate from the enamel (Ericsson 1949), is actually a 'moving target' and is not the same for every person. The critical pH can be different for different people. If salivary phosphate and calcium levels are low, the critical pH, the pH when there is net loss of mineral, can be as high as 6.5. People that tend to have very high calcium and phosphate levels in their saliva (and plaque fluid) may have a lower critical pH, such as 5.1 (Dawes 2003).

What is crucial, really, is the time that the enamel surface is exposed to acid. This is quantified as the area under the curve (AUC) in the classic Stephan graphs. If this area is large, one can expect that more calcium and phosphate would have escaped from the enamel. If this is repeated on a daily basis several times (i.e., because the subject is constantly snacking on cariogenic foods or beverages) then the 'red' AUCs combine during the day, and there will undoubtedly be a net mineral loss. This is demonstrated in Figure 1-17.

In this hypothetical comparison, the person at low risk (green) may not snack at all and has three meals of low cariogenicity spread apart during the day to allow remineralization to occur. The person with moderate (yellow) caries risk might have three meals and one snack of moderate cariogenic potential on a daily basis, and the combined AUCs below the critical pH might result in a net loss of mineral. At this stage, remineralization strategies might work. The person with a high risk for caries (red) snacks frequently during the day, and the total AUCs clearly are excessive and will not allow remineralization to occur. If that daily trend continues, the person will undoubtedly experience dental decay.

Researchers have determined that it is not only the frequency of ingestion that is important, but it is also the type of fermentable carbohydrate that is ingested. (We discuss these issues more in Chapter 6, on diet and

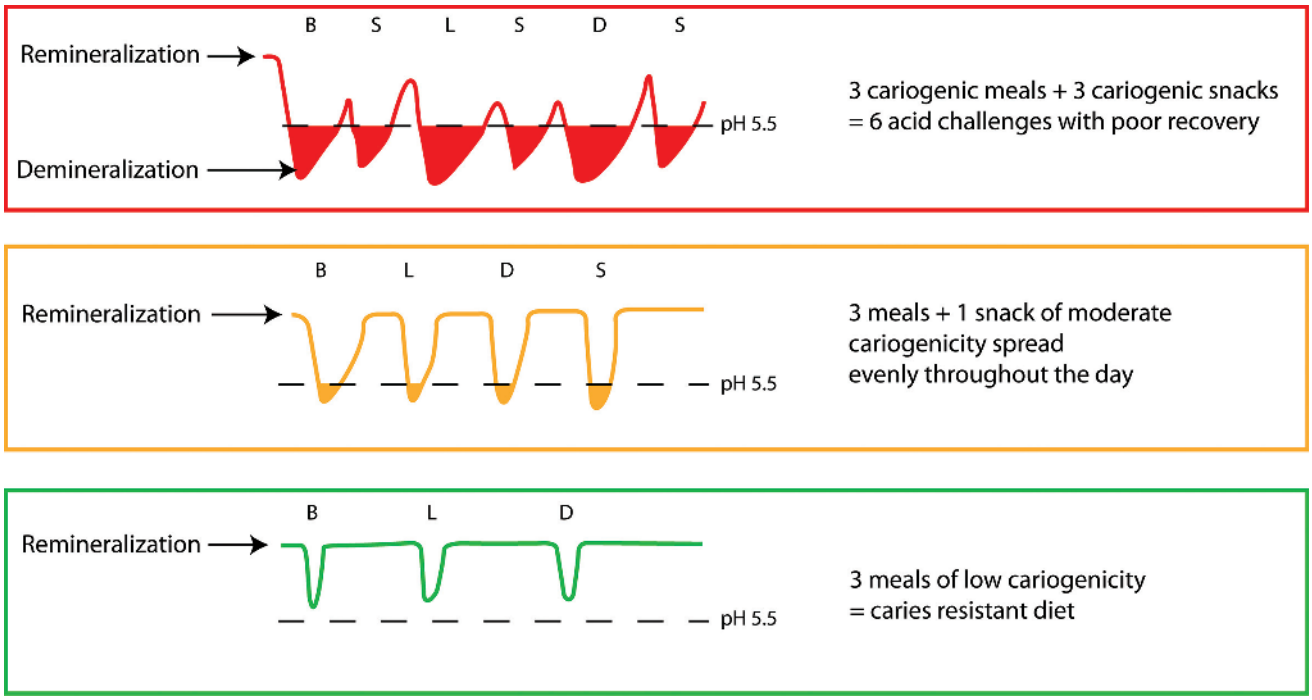

Figure 1-17 Daily repeat Stephan curves in low to severe risk individuals. Top: A high-risk individual (red) is exposed to several repeated acid challenges each day because of the many cariogenic snacks and meals that consumed in one day. The AUCs, which are large to begin with, all combine to add up to a net demineralization of tooth structure. Middle: An individual with moderate risk will have fewer daily cariogenic challenges, with less severe pH drops in the plaque, and may have enough time under remineralization conditions to allow for 'repair' of the enamel that could have lost some mineral. Bottom: The individual that is caries resistant is likely to be one who does not eat cariogenic meals or snacks more than three times a day. This person may never reach a plaque pH that risks the loss of mineral from the enamel.

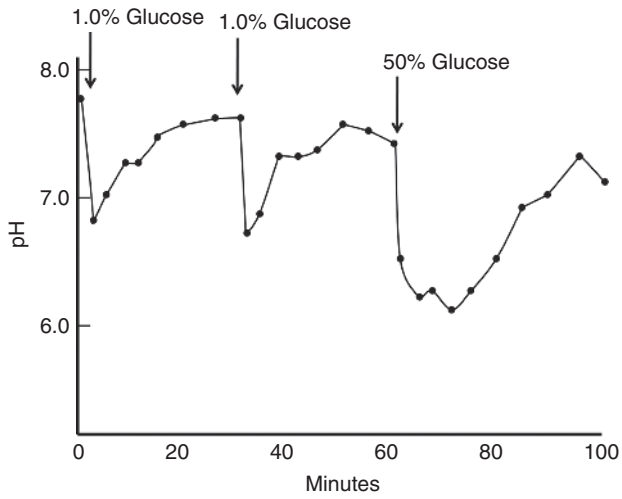

Figure 1-18 Plaque pH is dose dependent. Reprinted from Kleinberg *et al.* 1982, with permission from SAGE Publications.

caries.) For example Kleinberg *et al.* (1982) determined that increasing glucose concentrations results in lower pH drops (Figure 1-18).

In these experiments, it was shown that exposure to dilute glucose solutions lowered plaque pH but not too far for the saliva to neutralize the acid and return the plaque to neutral pH. However, when a concentrated solution of glucose is used, the plaque pH drops further and stays in the acidic range longer. This suggests that caries also depends on the concentration of sugar in the cariogenic foods.

Lingström *et al.* (1993) showed that the indwelling electrode was the most sensitive method for producing Stephan curves and discovered that potato chips were more cariogenic than soft white bread, which was more cariogenic than starch solutions or glucose solutions. This could reflect the fact that food impaction into pits and fissures as well as interproximal contact areas would prolong the retention of sold starches. The solid starches are then slowly converted to maltose by salivary amylases. The areas under the pH curve are more pronounced for retained solid foods.

Coronal versus root caries

Thus far we have discussed the general principles of enamel caries. Most of the coronal caries in modern times occur on the pits and fissures, making them ideal hiding places for bacteria since they are not easily

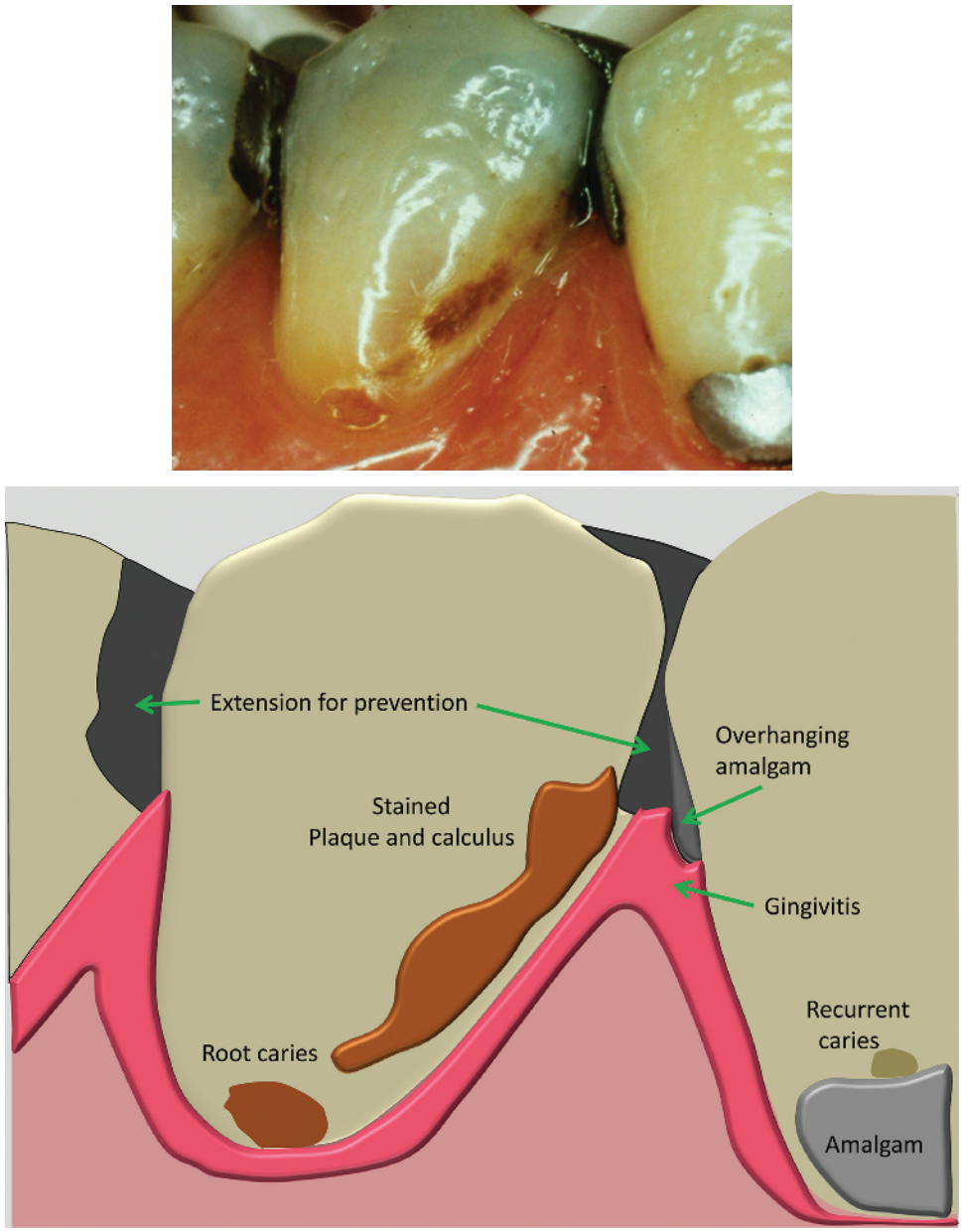

Figure 1-19 Example of root caries. This clinical image of a mandicular right first premolar demonstrates the start of cavitation at the demento-enamel junction at the gingival margin at the cervical region of the crown. Two brown stain areas are present but the most apical lesion is cavitated and an active root caries lesion. The illustration shows the various landmarks of this image.

disturbed with tooth brushing, and in the contact areas between the teeth where the plaque is sometimes left undisturbed for days because the toothbrush does not reach that area. It is only with flossing that the interproximal areas are disturbed.

Another form of caries is the caries that forms at the root surface that has been exposed due to gingival recession or periodontal disease (see the next section). The same bacteria are believed to be responsible for the decay of dentin (*S. mutans, Lactobacillus*), but *Actinomyces* species that are able to metabolize starch to sugars are also involved (Chen *et al.* 2001).

Root caries usually starts at the weakest point. The cement-enamel junction at the exposed root surface may or may not be hidden by plaque (Figure 1-19).

Cavitation occurs much more quickly; dentin has no critical pH and dissolves more quickly at low pH because the dentin tubules allow bacterial invasion, and the dentin crystals are smaller and readily dissolved at low pH.

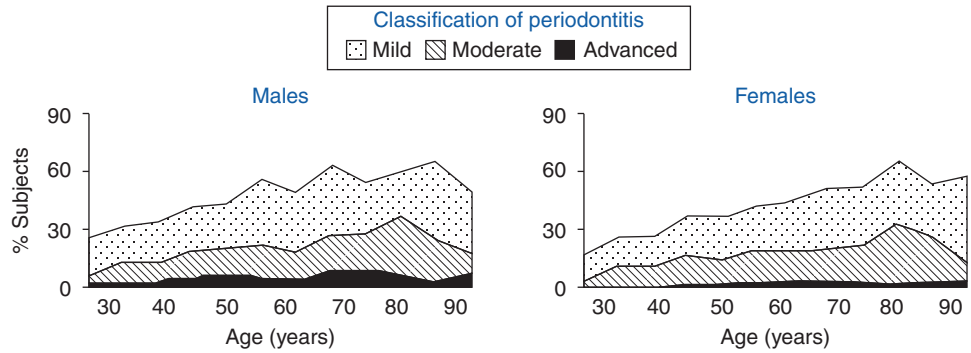

Figure 1-20 Percentage of individuals with advanced, moderate, or mild periodontitis among US adults examined from 1988 to 1994 by age and gender. Reprinted from Albandar, Brunelle and Kingman 1999, with permission from the American Academy of Periodontology.

Current patterns of periodontal disease

The prevalence of periodontal disease in the US has been monitored in a large-scale clinical study called the National Health and Nutrition Examination Survey (NHANES III). Mild periodontal disease is generalized, moderate periodontal disease is less prevalent, and severe periodontal disease is not very common. The prevalence increases with age in the adult population. A little more than one-third of the adult population has periodontal disease with 22% having mild periodontal disease and 12.6% having moderate to severe periodontal disease (as defined by pocket depths ≥3 mm and bone loss). The results showed that at the time of the NHANES III study (1988–1994), 21 million people had at least one site with ≥5 mm probing depth and 35.7 million persons had periodontal disease (Figure 1-20).

The World Health Organization developed a community periodontal index to measure the prevalence of periodontal disease in several countries. Gingival bleeding, periodontal pocketing, and loss of bone attachment were measured. Gingivitis, or gingival bleeding, was prevalent in all regions of the world. Severe periodontal disease (>6 mm pockets) is generally found in 10 to 15% of adults worldwide. The WHO identified periodontal disease risk factors that included poor oral hygiene, tobacco and alcohol use, stress, and diabetes. It proposed several preventive strategies to lower the risk for periodontal disease, and these were obviously aimed at reducing the risk factors (Petersen 2005).

Caries versus periodontal disease

Caries and periodontal disease are infections. They are caused by bacteria that can infect the oral cavity or by bacteria that are already present that become virulent. These bacteria reside in communities, sometimes in harmony, living in a symbiotic relationship, other times in conflict, competing for the same nutrients or resisting conditions that would result in their demise. On surfaces such as teeth, microorganisms usually live in communities called biofilms. It is now known that these biofilms change in their composition, properties, and adherence and that their inhabitants can change from being dominated by passive bystanders to those that over-run the biofilm and become aggressive pathogens. Commensal microorganisms, defined as those bacteria that live symbiotically with others, providing a benefit to themselves or the host, without affecting other organisms negatively, allow the body to function normally. It is estimated that there are 10^{14} cells in the human body and only 10% of them are mammalian (Sanders and Sanders 1984).

So many factors can disrupt this balance, and this can result in the host infection and pathological responses. Environment factors that affect the metabolism and numbers of active bacteria include, but are not limited to, oxygen tension, pH, energy supply, inorganic and organic chemical changes, inflammatory host response to foreign proteins/objects, and anti-bacterial agents (both intrinsic and extrinsic).

There are numerous surfaces in the oral cavity to which biofiolms adhere, each with their own unique characteristic. The mucosa of the inner lip, vestibule, attached gingival, tongue, and palate all have different families of resident bacteria that at any time can change in composition. The biofilms attached to the mineralized tissues (enamel, dentin, cementum) have bacteria with the ability to adhere to the salivary pellicle, a layer of proteins, lipids, and inorganic molecules derived from the saliva that make adherence to the mineralized tissue possible. This microflora is dominated by the facultatively anaerobic gram-positive bacteria, especially streptococci. There are more than 500 taxa of microbes normally found in the oral cavity, and these appear to be unique to the oral cavity since only about 29 of them end up in the feces (Moore and Moore 1994).

The bacteria in the gingival crevicular crevice are bathed not only in saliva but in crevicular fluid, a serum-like exudate from the sulcus of the periodontium. Both

are rich in protein, are neutral in pH, and are warm, perfect conditions to encourage bacteria to grow. As inflammation occurs, the crevicular fluid flow increases. Redox potentials change, and anaerobic bacteria start to grow, many of which produce proteolytic enzymes that breakdown host cells and soft tissue matrices and feed on the breakdown products. The gram-negative anaerobes such as *Prevotella, Porphyromonas, Fusobacterium*, and *Treponema* are found in the periodontal pockets where there is attachment loss (Moore and Moore 1994). It is suspected that the pathogens for both caries and periodontal disease can be transmitted from person to person, but disease emerges because quiescent pathogens that have always been present in small numbers in health oral flora are allowed to proliferate and dominate the plaque as a result of certain stresses and stimuli. Clearly, preventive strategies would seek to eliminate the stresses (Marsh 2003). Changes in redox potentials and pH in the plaque can favor the growth of periodontal pathogens. *P. Gingivalis,* for example, grows better when the pH is alkaline and haem-containing proteins (blood proteins such as haemaglobin) are available as a substrate (McDermid *et al.* 1988).

Periodontal disease etiology

'Periodontal disease' is an all-encompassing term that refers to a number of diseases of the periodontium. These include gingivitis, chronic periodontitis and aggressive periodontitis. The bacteria involved in these periodontal disease states were introduced in Table 1-3. How these bacteria come to dominate the sulcus is based, in part, on the *in vitro* experiments by Marsh and others (2003) in the previous section.

The pathogenesis of gingivitis

Gingivitis occurs when the gingival margin becomes red and edematous, and bleeds easily on palpation or probing. There are changes to the anatomy (usually puffiness or swelling), loss of adaptation to the tooth, and increased gingival crevicular fluid. Histologically, the tissue responds to local plaque bacteria in three ways (Payne *et al.* 1975). First, there is an acute inflammatory response with infiltration of neutrophils. Second, a chronic inflammatory infiltrate dominated by T and B lymphocytes is accompanied by collagen breakdown and proliferation of junctional epithelium. Third, progression through the acute phase of inflammation is followed by a chronic inflammation and progressive destruction of gingival tissue. Many systemic conditions predispose the gingiva to this inflammatory response. These include the conditions that affect vascular changes (leukemia, hemophilia, diabetes, Addison's disease), immunodeficiency conditions (HIV), hormonal changes (puberty, pregnancy, steroid therapy), and abnormal responses to drugs (seizure therapy, anti-rejection drugs) (Research Science and Therapy Committee of the America Academy of Periodontology 1999).

The pathogenesis of periodontal disease

Periodontal disease is characterized by attachment loss where the periodontium (gingival, periodontal ligament, and bone) fail to remain attached to the tooth and its root surfaces. Most sites of periodontal attachment loss start with inflammation, or gingivitis, but this is not always the case. The factors that lead to the initiation of periodontitis are not well known. It has been observed that periodontitis with attachment loss can be sporadic, acute, or chronic (Jeffcoat and Reddy 1991). In young adults loss of attachment can start in the proximal sites of the posterior molars (Thompson *et al.* 2006) where one expects poor oral hygiene. In susceptible patients, however, the disease can be quite aggressive and rapid and not be associated with gingivitis. The invasion and proliferation of virulent pathogens in the crevicular sulcus can lead to destruction of periodontal tissues because some of the periodontal pathogens produce enzymes especially dangerous to the integrity of the periodontium. Organisms such as *P. Gingivalis* can produce proteolytic enzymes (proteases, collagenase, fibrolysin) that degrade collagen and noncollagenous proteins. Metabolic byproducts such as hydrogen sulfide and ammonia can be toxic to mammalian cells, and lipopolysaccarrhides (LPS) can induce bone resorption (Hausmann 1970).

Once established in a periodontal pocket that has progressed (e.g., >6 mm), host-mediated destructive processes are initiated after the barrier of an intact periodontium is breached. Under normal circumstances, polymorphonuclear leucocytes are usually effective in staving off invading bacteria, but in the periodontium, they are overwhelmed trying to phagocytose the invading bacteria and the LPSs, releasing destructive enzymes in the extracellular environment. More collagen and basement membrane destruction ensues. The host response mechanism is believed to involve prostaglandin E_2 (PGE_2) and arachadonic acid, which promote the local release of matrix metalloproteinases, enzymes that lead to further destruction of the host tissues. Inflammatory mediators (also include the interleukins (interleukin 1 or IL-1; interleukin-6 or IL-6; interleukin 8 or IL-8) and tumour necrosis factor (TNF-α).

The biofilm in deep pockets responds to changes in oxygen tension, and anaerobic bacteria begin to dominate away from the influence of the saliva and in the

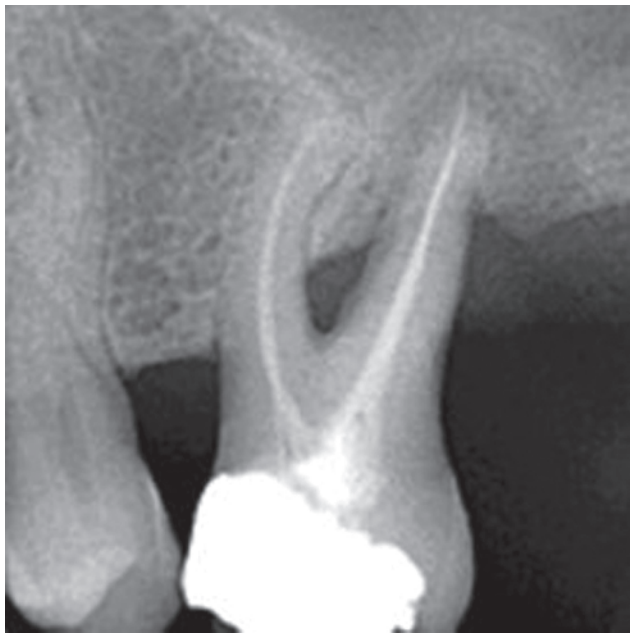

Figure 1-21 Radiograph of a tooth with periodontal bone loss. This periapical view of the maxillary right left first molar indicates significant bone loss and furcation involvement. Notice the large amalgam filling and the root canal treatment. Despite efforts to save this tooth through conventional restorative procedures from previous extensive caries, this tooth will be lost to periodontal disease.

deeper depths of the pockets. Advanced periodontitis is now an established anaerobic, gram-negative infection. Obvious treatment strategies include oxidizing agents, but at this point the disease has already been established. Our interest lies in preventing attachment loss, not treating the infection after the fact or intervening surgically—that is a topic for other textbooks. When the tooth has progressed to the point of major bone loss (Figure 1-21), it is obviously too late.

Oral cancer

Squamous cell carcinoma arising from the oral mucosa is the most common malignant tumor of the oral cavity, constituting more than 90% of all oral malignancies (Neville and Day 2002). In this book, the term 'oral cancer' will be used synonymously with oral squamous cell carcinoma. The oral mucosa is structurally similar, although not identical, to the mucosa of the oropharynx, hypopharynx, and larynx, and all of these mucosal surfaces are subject to the carcinogenic effects of smoking and alcohol. Many epidemiologic studies have been reported for 'head and neck cancer' and primarily refer to carcinoma of the oral cavity, oropharynx, hypopharynx, and larynx (Curado and Hashibe 2009). Data on the oral cavity will be presented where these have been reported separately; otherwise the discussion will be based on data on these sites studied as a group.

Oral cancer causes destruction of local tissues resulting in pain, inability to chew, swallow, and speak, and altered facial appearance. Metastasis to lymph nodes of the neck occurs frequently, and the metastatic malignant cells can invade vital tissues of the neck such as major nerves and blood vessels. The two major treatment modalities for oral cancer are surgical resection with neck dissection and radiation therapy to a field that includes the primary site and regional lymph nodes with evidence of metastases. The need to eliminate the entire malignant tumor often leads to extensive loss or damage to orofacial tissues. Despite surgery and/or radiation therapy, oral cancer can recur at the primary site or in the neck. The 5-year survival rate is only about 55% overall, but survival is much better for small localized lesions (stage I disease) where the 5-year survival is above 80% (Neville and Day 2002; Jemal *et al.* 2010).

The morbidity and mortality from oral cancer can be reduced through prevention. Primary prevention is achieved by reduction or elimination of risk factors due to lifestyle or habits, thus avoiding the development of disease. Oral cancer is strongly associated with tobacco use and alcohol drinking. In a global analysis of cancer mortality that can be attributed to behavioural and environmental risk factors, 52% of deaths from oral and oropharyngeal cancer (163,000 deaths/year) are attributable to smoking and alcohol use. When high-income countries and low- and middle-income countries are separately analyzed, 80% of oral and oropharyngeal cancer deaths (32,000 deaths/year) in high-income countries are attributable to these risk factors (Danaei *et al.* 2005). Better education of health care professionals and the public about the risk of oral cancer from tobacco and alcohol can curtail these habits and reduce the incidence of oral cancer and the burden of treatment.

Secondary prevention is achieved by treatment of incipient or early lesions to stop progression of disease and to promote a return to health. The oral mucosa is accessible to regular examination with simple equipment, so monitoring, detection, and treatment of early disease are highly feasible. Oral cancer may be preceded by a clinically identifiable premalignant lesion, which is typically a white or red patch, called leukoplakia and erythroplakia, respectively. Premalignant oral lesions and early oral cancer are often subtle and asymptomatic, but they can be detected by clinical oral examination by a health care professional who is familiar with the clinical features of these lesions. Secondary prevention also requires effective treatment of premalignant lesions and early cancer (Neville and Day 2002).

Secondary prevention complements primary prevention by intercepting disease that develops in the absence of known or controllable risk factors or after the exposure to carcinogenic agents has ceased. Early

intervention increases the chance of success of treatment with minimal side effects and complications.

Why do people get dental decay, periodontal disease, and cancer?

It would be important for the dental practitioner to identify those patients who are at high risk for dental disease. Some people just seem prone to disease. Some, despite all effects of optimal dental hygiene and healthy diets struggle to avoid dental decay and periodontal disease, and are worried about oral cancer. We discuss more in detail throughout the book what the risk factors are that increase a person's risk to dental disease. It seems that genetic susceptibility, poor oral hygiene, poor diets, and lifestyle choices all combine to increase the risk for poor oral health outcomes.

Poor oral hygiene

Poor oral hygiene is an obvious risk factor for dental diseases, primarily dental caries and periodontal disease. Although some people manage to remain dental disease free with minimal daily hygiene, mouth care remains one of the best preventive measures for controlling the onset of dental disease. It is the mainstay of primary prevention.

Lifestyle choices

A smoker who indulges in frequent snacks containing sugar will undoubtedly struggle with not only coronal caries but with root caries as he develops deep periodontal pockets. The outcome is early tooth loss, and a compromise in his ability to eat healthy foods. This creates a spiral of deteriorating poor oral health. Changing one's lifestyle is difficult, no question. However, when a dentist or hygienist is successful in influencing a patient's lifestyle choices, and that person changes for the better with obvious improved oral health, there is a sense of satisfaction for both patient and dental practitioner.

Diet and preventing oral cancer

Oral cancer rates can also be influenced by the diet. Although counter-intuitive, some researchers have found that a diet rich in animal protein may be protective (Carley *et al.* 1994; Morse *et al.* 2000), while others found higher intake of fruits protective (Winn 1995; Horn-Ross *et al.* 1997).

Smoking: an addiction that increases the risk for all three dental diseases

Caries

The evidence that smoking is an independent risk factor for caries in children is accumulating. For example, a study by Leroy *et al.* (2007) indicated that passive smoke was a risk factor for caries in children even after other known risk factors were taken into account. One study *in vitro* may have uncovered a mechanism to explain this increased risk for caries (Baboni *et al.* 2010).

Periodontal disease

The more adults smoke, the greater their risk for periodontal disease (Haber *et al.* 1993; Martinez-Canut *et al.* 1995). Smoking affects periodontal tissue vasculature, promotes proliferation of periodontal pathogens, and reduces the immune response to invading periodontal bacteria. Smoking cessation benefits patients with periodontal disease (Dietrich *et al.* 2007). Thus, long-term smoking makes it difficult to treat periodontitis. Indeed, a subset of refractory periodontitis patients is smokers (Schenkein *et al.* 1995).

Oral cancer

The link between tobacco use and oral cancer has been established (US Department of Health and Human Services 2004). It is a global problem, and the effects of tobacco consumption can be worse in some countries than others according to Professor Newell Johnson (2001):

> "Taken together, the effects of tobacco use, heavy alcohol consumption, and poor diet probably explain over 90 percent of cases of head and neck cancer."

Getting patients to change their habits and reduce excessive tobacco use and alcohol consumption would obviously go a long way to prevent oral cancer. One approach would be education since very few people know the risks for oral cancer (Horowitz, *et al.* 1995).

Based on Gelskey (1999) and a general knowledge of the caries and oral cancer literature, a summary table of causation could be developed (Table 1-4). There are still unanswered questions as to the role of cigarette smoke contributing to the etiology of caries, periodontal disease, and oral cancer. However, it can be concluded from this table that smoking is a major risk factor in the development of all three major oral diseases.

Can dental professionals prevent oral diseases before they occur?

A common thread of prevention attempts to tie these three main oral diseases together. Clearly their etiologies are different. Caries results from opportunistic bacteria that produce acids from dietary sugars. Periodontitis results from the growth of proteolytic bacteria deep in the gingival crevice in conditions of low oxygen tension and protein nutrient supply. Oral cancers result from the uncontrolled growth of dysplastic host cells as a result of carcinogenic stimuli. Each dental disease requires separate preventive strategies. Preventing these diseases on a population basis will require public health strategies

Table 1-4 Smoking—a risk factor for all three dental diseases

Criteria of causation	What is needed	Caries	Periodontal Disease	Oral cancer
strength of association	Does the association produce high odds ratios (after regression analysis)?	yes	yes	yes
consistency	Do other studies looking at the same association find similar results?	yes	yes	yes
specificity	Does the disease increase when the cause is introduced (or decrease when taken away)?	Some evidence	yes	yes
temporality	Does the cause precede the disease?	?	yes	yes
biological gradient	Is there a dose response?	ND	yes	yes
biological plausibility	Does the biological mechanism make sense?	mechanism unknown	yes	yes
coherence	Is the cause not in conflict with the natural history of the disease?	yes	yes	?
analogy	Is the cause associated with other diseases of similar etiology?	yes	yes	yes
experimental evidence	Do clinical trials prove causality?	None available, may be unethical to test	None available, may be unethical to test	None available, may be unethical to test

that mostly involve increasing the standard of living, better education, and improved access to professional care. These are lofty goals for some third world countries with limited resources. The more developed countries have managed to do this by making oral health an important part of overall general health and spending a higher proportion of their health care budgets on oral diseases. Their populations therefore experience relatively low oral disease prevalence despite engaging in high risk activities, such as the consumption of sugar-rich processed foods, excess alcohol ingestion, regular tobacco use, and neglect of the oral cavity. The WHO is attempting to reduce the risk of oral diseases by promoting healthy lifestyles, encouraging public health programs, improving education, and encouraging the control of chronic diseases such as diabetes (Petersen and Ogawa 2005; Petersen 2009).

This textbook is not meant to be comprehensive in terms of public health solutions to these diseases. Nor is it comprehensive in terms of providing advice on how to manage diseases once they have taken root. The reader of this text will find strategies to prevent dental diseases in clinical practice. The reader might be a dentist, dental hygienist, a dental student, a dental hygiene student, a dental therapist or dental assistant, or any person working, or seeking to work, in an auxiliary position in a dental or dental hygiene clinic. Some dental public health programs now provide direct professional care in government-sponsored dental clinics or subsidized nongovernmental organizations. Such an organization is the Head Start program in the US that partners with private practitioners (From: http://www.aapd.org/headstart/information.asp).

Final remarks

In this text, with a heavy focus on caries, the reader will be able to gain some insight on how to identify patients at risk for dental disease, how to introduce therapies that are known to reduce the risk, how to prevent caries or reverse caries, how to diagnose and prevent periodontal disease, and how to help those patients at risk for oral cancers. Education and guidance is paramount in a clinical practice setting in an attempt to achieve better oral health for the patient. Restorative dentistry can only be successful when the disease is under control. To quote the founder of "Your Teeth for a Lifetime," Dr. William Hettenhausen, who dedicated his career to preventive dentistry and nutrition, "You don't call the carpenter when your house is on fire."

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