Dental Caries: A Current Understanding and Implications

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Dental caries continues to be one of the most prevalent chronic diseases worldwide. A multi-factorial disease that involves tooth structure, oral microbiota, and dietary carbohydrates, “tooth decay” results in the dissolution of the mineral content of teeth and must be thought of as dependent on its key contributing factors. With ramifications ranging from societal to psychological to medical, dental caries can have serious consequences for patients. The current paradigm for management of dental caries is evidence-based and favors non-invasive therapies to prevent and/or arrest the progression of the disease, with traditional surgical intervention reserved for circumstances of irreversible tooth structure loss. Journal of Nature and Science, 1(1):e27, 2015.

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Introduction

Dental caries, or “tooth decay” as it is more colloquially known, is a microbiologic disease1 that has implications both local to the oral cavity and in extreme cases, systemically. While other conditions, such as cancer, diabetes, and cardiovascular disease garner much attention in the scientific community and public, dental caries continues to quietly manifest with alarming prevalence. Indeed, dental caries has been identified as the most common chronic childhood disease in the United States, more common than asthma and hay fever.2 Globally, 60-90% of children and nearly 100% of adults have teeth affected by dental caries3

Beside the implications for personal and public health, dental caries has a strong economic impact. Oral diseases are the fourth most expensive to treat in the industrialized world.4 An estimated 5-10% of public health expenditures in these countries are for oral health.2,5 It should be noted that not all patients who would benefit from dental treatment in industrialized countries seek it, likely influenced by economics as well.6 And in various developing nations treating dental caries is often cost prohibitive; treating caries in children would exceed the entire allocated national children’s health care budget.7

Clearly dental caries is a disease whose prevalence and resulting economic burden merit attention. Because dental caries implicates the wider public, this article aims to provide a concise overview of the current understanding of dental caries, with those outside the dental field as its primary audience. This will be accomplished by reviewing the etiology of dental caries and highlighting implications for its management, with special attention given to disease prevention and/or arrest.

Pathobiology of dental caries

Dental caries is a microbiologic disease that results in the dissolution of the mineral structure of teeth.1 Although there are other factors involved, it is generally accepted that three basic components must be simultaneously present over a period of time for caries to clinically manifest: a tooth substrate, acidogenic bacteria, and fermentable carbohydrates for the bacteria to metabolize. Each of these components will be briefly examined here.

To begin, human teeth are anchored in the alveolar bone by a ligamental sheath (the periodontal ligament) and are composed of three histologically distinct layers.8 The innermost layer, the pulp, consists of vascular and nerve connections to systemic counterparts; these give teeth “vitality.” The pulp is encased protectively by dentin, which has a mineral content of about 75%; blastic cells for dentin (odontoblasts) can be found at the pulp-dentin interface. Dentin of the crown (portion of tooth clinically visible in oral cavity) is covered by enamel, which is even more highly mineralized than dentin at over 95% — it is the hardest substance in the human body. Cementum covers the root dentin and by contrast is only about 50% mineralized.8

At least two things are of note regarding the relationship between tooth histology and dental caries. First, more highly mineralized layers of teeth are more resistant to acidic dissolution which occurs in dental caries — thus enamel at one end of the spectrum plays an important protective role, but cementum on the other end is more susceptible to mineral loss. Secondly, the presence of live odontoblasts in the pulp provides potential for repair to acid-damaged dentin. Enamel does not have this same ability for self-repair, although the literature strongly points to other means to non-invasively repair its mineral structure, which will be discussed later.

Acidic, acidogenic bacteria are thought to be the primary agents responsible for lowering pH in the oral environment, which in turn results in the dissolution/demineralization of teeth.9 Although the oral ecosystem is complex and there likely a potentiating/additive effect of a variety of bacterial species in dental caries,10 the chief initiator of caries on tooth enamel is Streptococcus mutans.9,11 Other species, such as Lactobacilli, have been associated with advancing dental caries lesions, as well as Actinomyces on root cementum.9,12 These various species reside in biofilm on tooth surfaces – dental plaque; left undisturbed and after metabolism of fermentable dietary carbohydrates like sucrose,13 acidic bacterial by-products lower oral pH. During extended periods of lowered pH (approximately 5.5 for enamel, 6.5 for dentin14-15), minerals such as phosphate and hydroxyl ions leech out of the tooth into the oral environment.16

The first clinical sign of this demineralization is a slight color change in the enamel surface – the initial caries lesion; if oral pH is restored to a more neutral level by the removal of bacterial plaque and/or carbohydrates, then available calcium, phosphate, and hydroxyl ions in the saliva and plaque participate in re-uptake into the enamel crystalline structure.16 Thus the dental caries is thought of a chronic and on-going disease, with the pendulum swinging between demineralization of tooth structure during periods of low pH and remineralization during periods of neutral/higher pH.16-17 When the teeth are allowed to demineralize for extended periods of low pH, the eventual total mineral loss is too much to overcome by remineralization and the enamel sub-structure collapses, forming a cavity.17 Until this point, however, arrest of a caries lesion is possible.

To review, acidogenic bacteria like Streptococcus mutans create acid by-products, which in turn demineralize tooth structure. These...
acidogenic bacteria primarily metabolize dietary fermentable carbohydrates, like sucrose, to produce the acidic by-products – this makes patient diet and oral hygiene an important controlable aspect of the disease process. Indeed, data suggests that when sugar intake is limited to < 10% energy, caries experience is reduced. Furthermore, time plays an important role – the longer the teeth and bacteria are exposed to carbohydrates, the more time the bacteria have to create acidic by-products and thus demineralize the teeth. By extension, if a limit is placed on how much time the carbohydrates spend in the oral environment, then the acid challenge is reduced. For this reason, snacking and/or sipping on foods and beverages is discouraged. So in one sense, from a dental perspective it matters not how much carbohydrates are ingested, but how much time is spent doing so. Regardless, because patient diet relates strongly to daily decisions and actions, a current understanding of dental caries categorizes it as a “behavioral disease with a bacterial component.” This nuanced, but significant, shift in perspective on dental caries has resulted in a paradigm change with regard to its management.

**Problem of dental caries**

Now that a foundational understanding of the caries disease process has been established, it will be beneficial to highlight the problems that the disease presents. At a policy level, the high prevalence and resulting economic impact have already been mentioned. What about the impact of dental caries at the individual patient level?

The tooth color and appearance changes that accompany demineralization are well known to present psychological/esteem challenges for patients, especially if the dental caries affects multiple teeth. Quality of life can be further negatively affected if multiple/all teeth are lost to oral disease – removable dentures and related prostheses tend to provide lesser masticatory and facial support than natural teeth. Furthermore, the concurrent prevalence of dental anxiety keeps many patients from seeking treatment, which allows their dental caries experience to progress and worsen.

From a medical perspective, localized progression of dental caries can have serious implications. As noted earlier, prolonged periods of bacteria-induced low pH in the oral environment encourages an unchecked constant demineralization of tooth enamel. If enough mineral content is lost, the enamel apatite matrix substructure collapses into a cavitation, or “hole,” and is unremineralizable. Furthermore, this cavity is a niche for cariogenic bacteria. Recalling that dentin has a lower mineral content than enamel, once the cavitation penetrates to the dentin level, progression of the local disease is more rapid. For this reason, the historic treatment for a cavitated caries lesion has been to place a synthetic filling of some sort to seal the hole. Continued demineralization of the dentin, accompanied by proliferation of cariogenic bacteria, eventually results in communication of the caries infection with the pulp – the nerve and blood supply for the tooth. Pulpitis, or inflammation of the pulp, can arise due to proximity of a caries lesion, and the resulting discomfort/pain can be intense.

Actual physical communication between cariogenic bacteria and the dental pulp usually results in an irreversible pulpitis or/and pulpal necrosis. In these cases, typically an abscess forms at the apex of the tooth root, near the small aperture that allows vascular and nervous connection of the tooth with systemic counterparts. The purulent exudate resulting from the infected pulp tissue is limited to the extremely narrow root space and must flow to the root tip, thus forming the abscess. Unless the abscess is drained physically through extraction of the tooth or pulpectomy (removal of pulp tissue, precursor to root canal therapy), the infection will persist – antibiotics are typically not effective alone for elimination of the disease.

Untreated dental caries at this advanced stage now has the potential to spread to fascial spaces, which are very difficult to treat. Systemic involvement of infections that have carious origins become very serious – spread to other structures such as the brain have been observed. One high profile recent death involved spread to the brain from a dental infection in a twelve-year old. Deaths have been document in a number of other cases as well, typically from sepsis or airway obstruction. Dental caries is clearly a disease that presents a number of serious potential problems.

**Prevention and management of dental caries**

Because the pathobiology and problems related to dental caries have a strong medical component, it will come as no surprise that the current state of the art in caries management follows a medical model. While previous approaches involved surgical intervention (ie. dental fillings) as the normal response to dental caries, with oral hygiene playing a secondary passive role, the current scientific approach brings prevention to the forefront, resorting to the surgical only when necessary. Indeed, placing a filling in a diseased tooth never treated dental caries – it only restored that which was damaged by the disease. In fact, the disease can manifest again at the margins of any filling that is placed; this secondary or recurrent dental caries will necessitate an even larger filling in the future. True prevention and management of dental caries will be linked by necessity to the two somewhat manipulable components of its etiology: the bacterial and the behavioral.

It has been established in the literature that a variety of factors affect a person’s overall risk for dental caries. Oral bacterial load, dietary habits, and patient oral hygiene are examples of factors that contribute to caries risk level. Although some risk factors, such as salivary flow (saliva acts as a pH buffer), may not be under the control of the patient and/or dentist, many of the other factors are. Much like a primary care physician, who regularly and over the lifetime of a patient monitors and adjusts control of chronic diseases, dentists now are to be pro-active in managing dental caries over the lifetime of the patient. Depending on the patient risk level (which will be re-assessed regularly over time), the dentist will combine in-office therapeutics, patient behavior modification, and surgical interventions to treat the patient. This patient-centered, medical approach to dental caries is known as caries management by risk assessment.

While patient behavior modification, such as encouraging more regular brushing/flossing (to reduce plaque and bacterial load) and diet modification (to minimize carbohydrate exposure) will have the strongest benefit, it is very difficult for patients to make significant changes immediately. Additionally, a purely microbiological approach has also yielded little success; an antibiotic or vaccine for dental caries has not been developed in part because the disease is propagated by a wide diversity of oral flora. Despite these discouraging clinical realities, there are two modalities that in the scientific literature show strong preventive and disease halting properties – fluoride and pit-and-fissure sealants.

The efficacy of fluoride and pit-and-fissure sealants for managing dental caries relate very strongly to the concept that the demineralization and remineralization process, in which caries plays a key role, is a pendulum that constantly swings back and forth between the extremes. As long as a carious lesion on a tooth has not resulted in the cavitation of the surface enamel, that lesion can be arrested non-invasively. Fluorides and sealants act to do just this.

Fluoride, which is the ionic form of the element fluorine, has been well established to prevent and arrest dental caries in its various forms, whether in-office or take-home. Fluoride has three modes of action with regard to dental caries. First, fluoride ions in the oral environment act to accompany the re-uptake of calcium and phosphate into the enamel under acidic conditions, thus enhancing remineralization. Secondly, fluoride ions chemically insert themselves into the crystalline apatite structure of outer enamel – the...
negatively charged fluoride ion replaces hydroxyl groups in the apatite. This new fluorapatite (or fluor-hydroxyapatite) is actually more acid resistant than the original hydroxyapatite.46 While the threshold for demineralization of hydroxyapatite is approximately pH 5.5, with fluoride incorporated into the crystal structure the new threshold is closer to pH 4.5. And finally, fluoride appears to act therapeutically against cariogenic bacteria. Fluoride enters through the cell membrane of bacteria, interrupting metabolism of carbohydrates and the production of adhesive polysaccharides (which allow bacterial adherence to teeth/plate).46 Thus, the primary anticaries mechanism of fluoride is realized through the topical route, with direct contact to teeth. All patients will benefit from daily oral exposure to small quantities of fluoride, such as found in fluoridated drinking water and over-the-counter fluoride toothpaste.48 Moderate to high caries risk patients will additionally benefit from regular in-office high concentration fluoride varnish/gel49 and prescription fluoride toothpaste.50 Fluoride is able to remineralize and arrest current caries lesions, and prevent future lesions.

Pit-and-fissure sealants address dental caries manifesting in a very specific region of tooth anatomy. The large teeth in the posterior region of the mouth present with a number of depressed grooves, pits, and crevices – this is the pit-and-fissure system. These areas are difficult to clean and can easily harbor cariogenic bacteria and plaque; pit-and-fissures are the most common areas to find the manifestation of dental caries. Dental sealant is a plastic-resin that is flowed into the pit-and-fissure system, effectively blocking out the physical niche for bacteria. Once cured solid, typically through a wavelength-specific photoinitiator and curing light, the risk for caries in the pit-and-fissure system is greatly reduced.48-49 Sealants can be placed on the pit-and-fissure system of any tooth, or the teeth of any patient, considered at risk for dental caries.50 Even more amazingly, but quite logically, sealants can be placed over active caries lesions in pit-and-fissures that have not progressed to the point of cavitation.50 Cariogenic bacteria in these circumstances are effectively trapped and isolated from dietary carbohydrates; they thus are greatly reduced in number and viability,51-55 and the lesion halts progression.58 Sealants, like fluoride, are well established to prevent and arrest dental caries.

In summary, prevention and management of dental caries today will depend heavily upon accurate/regular caries risk assessment, appropriate behavior modification, and judicious use of non-invasive evidence-based modalities like fluoride and sealants to prevent and arrest acute caries lesions. For lesions that progress to cavitation, limited surgical intervention with a dental filling is recommended.20 Although this medical philosophy of caries management is quite logical and may even seem common sense to the reader, this represents a major shift in thinking within the dental specialty. There has been a hesitancy to accept treatments like placing sealants over early caries lesions in the practicing community; it will take time for trends to change.

Conclusion
Dental caries is a prevalent disease with bacterial and behavioral components. The disease has serious potential ramifications, both in a global sense and for the individual patient. Current advances in understanding of the caries disease process, as well as treatment of that process, allow for a shift toward the medical management of the disease.


