4.1 Caries – Ancient Plague of Humankind

Peter Caselitz

Introduction

Humans are all too often haunted by their decayed teeth. Caries is not the largest plague of humankind, but certainly a daily nuisance. The aim of this chapter is to review the current theories of etiology, methods of scientific analysis, and particularly the evolution of caries in a historical dimension. Caries, or caries dentium, is the common name for tooth decay. It is a local disease characterized by an irreversible and progressive destruction of the hard dental tissue. Caries starts at the enamel surface of the tooth or – in case of recessed gingiva – at exposed parts of the neck. It works its way progressively through the dentine into the pulp cavity. The crown can be totally destroyed and other periodontal difficulties such as abscess may follow (for pulpoalveolar and periodontal diseases see Strohm and Alt, this volume). Finally – in more favourable cases – the affected tooth will fall out and the alveolus will be closed naturally. Alternatively, inflammation will spread into the surrounding bone or – specifically in the case of the upper teeth – the maxillary sinus will suppurate, leading to further, sometimes even deadly complications (for periapical diseases see Alt et al., this volume).

Etiology

Different theories of the etiology of caries have been discussed (for a historical review: Greve 1949; Cootjans 1955; Nikiforuk 1985). According to the most commonly accepted theory, the destruction of the hard dental tissue results from acids produced by micro-organisms of the oral flora, especially in an adherent gelatinous film on teeth and gingiva, called plaque. Plaque consists of food debris, salivary proteins, micro-organisms and polysaccharides of bacterial origin built up on the teeth in the absence of efficient oral hygiene (Moore and Corbett 1983, 140). Plaque is found mostly in fissures, small pits, and on the sides of the crown, and especially in the small area where the gingiva and tooth are in contact. It sometimes
forms a supragingival calculus called dental tartar which is visible in archeological finds as a band of hard material surrounding the crown like a ribbon. The occlusal surfaces are normally not affected because of the abrasive effect of rough diet components.

The conditions for the development of caries depend upon the chemical balance between acid and alkaline produced by plaque bacteria’s metabolism of different food components left over in the mouth as micro-debris. The metabolization of protein and carbohydrates produces alkaline waste products and lactic acid. The chemical balance switches from acid to alkaline several times a day. Particularly high acidity occurs when the diet contains large quantities of sugar. Sugars are metabolized much more rapidly than are other carbohydrates and as a consequence more lactic acid is produced more quickly (Hillson 1979).

Lactic acid carries out the initial attack on a tooth, resulting in demineralization of the enamel of the crown or of the cementum of an exposed part of the neck. The effect can be promoted by bacteria, such as *Streptococcus mutans*, *Streptococcus sanguis*, or *Staphylococcus albus* (Clarke 1924; Menzel 1925; Clement 1961; Shklair 1973; Hite 1975; review in Keene 1981). In case of caries in the neck area of a tooth, *Actinomyces viscosus* and *Actinomyces naeslundi* seem to be involved in the process. However, the acid theory has never reached total acceptance. Caries may also depend on genetic factors (Stewart and Spence 1976 versus Grundgeir 1987) as well as on socio-cultural influences. Even environmental factors may have an influence as well as may pathological conditions of saliva formation or nutritional disturbances during tooth formation manifested as enamel hypoplasias. It can be summarized that “caries is a complex multifactorial disease caused by the interaction between a susceptible tooth, the presence of certain cariogenic micro-organisms, and a suitable oral environment” (Patterson 1984, 62). Yet caries seems to depend largely on the sort and manner of eaten food (Horster 1939; Somogyi 1964; Bibby 1966; Hartles and Leach 1975). Another theory claims a possible transmission of caries-promoting bacteria from parents to children when they are fed with a licked spoon or baby’s bottle (Köhler and Bratthall 1978; Berkowitz and Jones 1985).

**Caries in Past Populations**

Caries is a very ancient and most widely spread disease. It already existed in the Australopithecines of South Africa (Robinson 1952; Clement 1956) as well as in the *Homo erectus* species from Java/Indonesia (Brodrick 1948). In the Neanderthals of Mount Carmel/Palestine caries is as common as in French upper paleolithic humans (e.g. in Aurignacian and Solutrean; see Praeger 1925; Vallois 1936; Krogman 1938; Brothwell 1963). Unfortunately, with the exception of two Neanderthal series the number of observations is too small for a statistical analysis. Caries is not limited to humans. Modern apes are affected (Colyer 1936; Schultz 1935;1967) as well as Pliocene animals and Pleistocene mammals (Clement 1958). Caries is also common in domesticated animals, especially when infected with *Streptococcus mutans* (Berger 1937; Zuhrt 1967; Baker and Brothwell 1980 versus Shklair 1981).