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Review

Acquired pellicle as a modulator for dental erosion

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ABSTRACT

Dental erosion is a multifactorial condition that can result in the loss of tooth structure and function, potentially increasing tooth sensitivity. The exposure of enamel to acids from non-bacterial sources is responsible for the progression of erosion. These erosive challenges are counteracted by the anti-erosive properties of the acquired pellicle (AP), an integument formed *in vivo* as a result of selective adsorption of salivary proteins on the tooth surface, containing also lipids and glycoproteins. This review provides an in-depth discussion regarding how the physical structure of the AP, along with its composition, contributes to AP anti-erosive properties. The physical properties that contribute to AP protective nature include pellicle thickness, maturation time, and site of development. The pellicle contains salivary proteins embedded within its structure that demonstrate anti-erosive properties; however, rather than individual proteins, protein–protein interactions play a fundamental role in the protective nature of the AP. In addition, dietary and synthetic proteins can modify the pellicle, enhancing its protective efficiency against dental erosion. The salivary composition of the AP and its corresponding protein-profile may be employed as a diagnostic tool, since it likely contains salivary biomarkers for oral diseases that initiate at the enamel surface, including dental erosion. Finally, by modifying the composition and structure of the AP, this protein integument has the potential to be used as a target-specific treatment option for oral diseases related to tooth demineralization.

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1. Introduction

Dental erosion is described as the loss of dental hard tissue due to dissolution and chemical etching by acids of non-bacterial source.[1] The dissolution process in dentine is more complex than that which occurs in enamel, due to the presence of the demineralized organic matrix that hampers ionic diffusion.[2,3] In recent years, the prevalence of dental erosion has significantly increased, particularly in developing countries, affecting anywhere between 4–82% of adults and 6–50% of children.[4] The pathophysiology of dental erosion is modulated by multiple factors including host behaviour, salivary flow rate, and the microenvironment surrounding the tooth. As a result of the multifactorial dependence, high prevalence, and potentially rapid and destructive behaviour of dental erosion, the development of effective management and preventive approaches to avoid the dissolution of the tooth is becoming increasingly important.[5] To date, there is no standard approach to treat and prevent erosive wear, but the use of fluoride-containing dental products is the most common preventative measure. While the constant presence of low levels of fluoride in the oral fluids is effective for caries control,[6] the anti-erosive effect of conventional fluorides requires a very intensive fluoridation regime.[7]

Alternatively, human saliva possesses several natural biological properties that protect tooth surfaces against demineralization.[8] For instance, the bicarbonate content in saliva supplies a constant source of ions that interact with the tooth surface, acting as a buffer that effectively resists changes in pH, thus neutralizing acids that are responsible for erosion.[9] Along with buffering capacity, salivary clearance of erosive agents and its remineralizing capacity also contribute to the anti-erosive properties of saliva.[9–11] The protective function of saliva can also be attributed to the formation of the acquired pellicle (AP), an integument formed *in vivo* as a result of selective adsorption of salivary proteins to the tooth surface, containing also lipids and glycoproteins.[12] Within seconds of enamel exposure to saliva, the initial phase of pellicle formation occurs,[13] during which precursor proteins (*i.e.*, statherin, histatins, acidic proline-rich proteins) selectively adhere to the surface, forming a protein layer 10–20 nm thick.[14] The rapid increase in pellicle thickness during the second stage of pellicle formation (100–1000 nm) and the presence of adsorbed knotted, globular-like structures *in vivo* suggests that protein aggregates, rather than individual proteins, are responsible for subsequent pellicle development.[15]

In this review, we discuss the role of AP physical properties, and the influence of salivary, exogenous, and synthetic proteins on the protective nature of the AP (summarized in Fig. 1). We also consider the pellicle to be a strong candidate for a future proteomics-based diagnostic tool, along with its potential as a target-specific therapeutic treatment option.

2. Anti-erosive properties of the pellicle

The AP protects the tooth from enamel demineralization by acting as a natural diffusion barrier inhibiting the direct contact between the tooth surfaces and dietary acids.[16] As a result, there is a decrease in diffusion rates of phosphate and calcium ions into the surrounding fluid following exposure to acidic conditions, thus protecting against tooth demineralization.[13,16] More specifically, the AP significantly inhibits the surface microhardness loss and surface roughness increase on bovine enamel that occurs as a result of exposure to organic acids (*i.e.*, citric acid; Ref. 17).

The protective efficiency of AP against erosion is dependent on its physical properties, including pellicle thickness and maturation time. The thickness of the AP varies widely throughout the oral cavity. The AP is thickest on the lingual surfaces of the lower teeth, since this region is constantly bathed in saliva excreted from submandibular and sublingual glands.[18] It has been reported that a thicker pellicle exhibits stronger protective effects against erosion.[19] The palatal surfaces of upper teeth are exposed to shear forces from the rubbing action of the tongue and these areas are also poorly bathed in saliva, resulting in a thin pellicle layer.[19] This could be related to the fact that palatal surfaces are frequent sites of erosion in children[20] and adults.[21] However, when buccal and palatal pellicles formed at the maxillary arch are compared, there is no significant difference in their protective effects.

It has been suggested that only mature, several day-old pellicles are capable of preventing enamel demineralization. However, when 24-h and 7-day-old pellicles were compared, there was no significant difference in the protective ability of the pellicles.[13] Studies have demonstrated that pellicles developed over 1 h offer maximum protection against demineralization, with no subsequent decrease in erosion when using longer maturation times.[19,22–24] Hannig et al.[25,26] studies found no difference in the protective effect of a pellicle formed after 3 min compared to a pellicle formed after 2 h. This can be attributed to the fact that pellicle formation, in terms of protein adsorption, begins within seconds of exposure of salivary proteins to the oral cavity,[25,26] producing an electron dense basal pellicle layer after 1 min.[27] Since subsequent pellicle layers are much less electron dense and are much more loosely arranged compared to the initial basal pellicle layer, they offer little additional protection against acidic attack.[25,26] This is consistent to the observation that after consumption of acidic beverages, the globular outer layers of the AP are removed to a different extent according to the localization of the specimens in the oral cavity, whereas the basal pellicle is not affected.[28]

Due to the distinct compositions of enamel and dentine, it can be assumed that the AP formed on these dental tissues might differ. Thus, it could be expected that the protective role

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