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| **Name:** |
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| 2A-1 |
| **Pathology Question:** |
| How does accumulation of plaque affect dental caries progression? |
| **Report:** |
| Dental caries is a disease which involves the progressive destruction of tooth structure as a result of continued exposure to acid produced by cariogenic bacteria in the oral microbiome. In order to progress, a susceptible host, a carbohydrate rich environment (sucrose), and a buildup of cariogenic bacteria must be present. In health, cariogenic bacteria exist in low numbers compared to other species of oral bacteria due to the presence of the many components that form the acquired pellicle to protect the teeth against acid producing bacteria. These components include proteins, chemical compounds, fluoride and calcium ions, immunoglobulins and water. Many of these proteins, including statherin and proline-rich proteins promote remineralization of the enamel following the exposure to acid. The saliva also contains high levels of bicarbonate, which helps to neutralize acid, as well as water to dilute acid and flush out bacteria. Many antibacterial proteins also exist in saliva including lysozyme, lactoferrins, peroxidases, histatins and cystatins. In addition to these proteins, saliva also contains IgM and IgG which bind to the surface of microbes and prevent bacterial metabolism.  Bacteria tend to accumulate in areas that are more difficult to clean, including interproximal areas below the contact area as well as in deep pits and fissures on the occlusal surfaces of posterior teeth. Early in the caries disease process, intial bacterial colonizers (such as S. mutans) begin to bind to the pellicle. It is well documented that S. mutans is one of the most important organisms for early colonization and caries initiation. The presence of S. mutans allows for the subsequent adhereance of acidogenic bacteria such as Lactobacillus. As plaque accumulates of time, this biofilm continues to grow and mature, becoming extremely diverse and efficient at producing acid and avoiding the body’s defense mechanisms. These biofilms are diverse, containing hundreds of species of bacteria. Once mature enough, bacteria can undergo quorum sensing to allow for very coordinated gene expression and responses to host defense mechanisms. In early carious lesions, enamel destruction is purely chemical, as bacteria do not have the ability to invade intact tooth structure. These lesions can be remineralized and arrested, aided by the presence of excess fluoride ions in saliva. However, with sustained poor oral hygiene and a carbohydrate rich diet, bacteria and acid begin to accumulate, pathological factors begin to dominate over the protective factors of the oral cavity.  As demineralization continues to occur throughout enamel, first degree reactions take place and odontoblasts in the pulp stimulate calcification of the distal aspect of odontoblastic processes to prevent the invasion of bacteria and progression of the lesion. The lesion then tends to spread along the DEJ, leading to second degree reactions that cause calcification of the proximal end of the odontoblastic process and subsequent reparative dentin formation. With a larger and larger lesion, third degree reactions take place and more reparative dentin is formed. With continued production of reparative dentin, odontoblasts begin to die and pulpal irritation greatly increases.  Due to the progressive nature of this disease, it is imperative that cariogenic bacteria are kept at low levels. In patients with reduced salivary flow (often due to medication), those with poor oral hygiene, or those that consume especially sugary diets are at a much greater risk for caries simply due to the fact that bacteria are allowed to accumulate, mature into a protective biofilm, and efficiently produce acid, leading to the progressive demineralization and destruction of tooth structure. |
| **References:** |
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