Role of Odontoblasts in the Dental Pulp's Defence against Caries

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Description

Dental caries is an infectious illness that affects the enamel and dentin over time and is brought on by mouth bacteria. The dental pulp then experiences inflammatory reactions as a result of microorganisms. If the infection is not too severe, these occurrences can result in pulp healing after the diseased enamel, and dentin tissues are removed and the tooth is clinically restored. However, despite treatment, chronic inflammation frequently remains in the pulp, resulting in irreversible loss of healthy tissue and a reduction in innate repair abilities. In order to keep infectious pathogens and restorative materials away from the pulp and ensure complete tooth healing, a reactionary or reparative dentin barrier must form. Data from clinical studies and *in vitro* experiments show explicitly that dentin barrier creation only takes place when pulp inflammation and infection are kept to a minimum, allowing for the restoration of tissue homeostasis and health. To ensure the longevity of dental treatments, it may be beneficial therapeutically to encourage the resolution of pulp inflammation.

Due to their precise localization at the pulp-dentin interface and the embedding of their lengthy cellular processes in dentin tubules, odontoblasts are the first pulpal cells encountered by dentin-invading pathogens and their released products. They play the same role that skin and mucosal epithelial cells play elsewhere in the body as the first physiologically active line of defence for the host in the mouth. In order to prevent bacterial invasion and activate the innate and adaptive components of dental pulp immunity, odontoblasts may be involved. These two actions can only be brought about by the pulp cells recognising the pathogen. The detection ("sensing") of molecular structures that are shared by infections and necessary for microbe survival is the general process by which such recognition takes place. These patterns are recognised by a small number of so-called pattern recognition receptors and are known as Pathogen-Associated Molecular Patterns (PAMPs).

One major consequence of TLR activation is the upregulation of innate immunity effectors, including antimicrobial agents and proinflammatory cytokines and chemokines that recruit and activate tissue-resident and blood-borne immune and inflammatory cells. Odontoblasts have been found to produce several antibacterial agents, among which beta-defensins and nitric oxide have received particular attention. Beta-Defensins (BDs) are cationic, broad-spectrum antimicrobial peptides that kill microorganisms by forming channel-like micropores that disrupt membrane integrity and induce leakage of the cell content. They are mainly produced by epithelial and immune cells to protect skin and internal mucosa from pathogen invasion.

The crowns of human teeth are covered in symbiotic microbial communities composed primarily of tooth-safe Gram-positive saprophytic bacteria. These communities form biofilms that stick to the highly mineralized enamel, forming a barrier that is impermeable to pathogens and guarding the loose connective tissue called the dental pulp located in the tooth's centre and the underlying mineralized dentin. However, certain bacterial populations from these communities emit acids that gradually demineralize enamel when exposed to a sugar-rich environment. This causes a "cariogenic" lesion, which is characterised by a cavity in which "cariogenic" bacteria multiply and produce more acids, gradually deepening the lesion. Gram-positive bacteria, which pre-dominate the dentin caries microflora and include streptococci, lactobacilli, and actinomyces, destroy dentin when the enamel barrier is compromised. These germs multiply and undergo metabolic activity, which releases bacterial components into dentinal tubules and causes them to diffuse toward the peripheral pulp. The release of bioactive molecules from the dentin matrix may also be made possible by dentin demineralization. At the dentin-pulp interface, host cells that recognise bacterial components start to produce antibacterial, immune, and inflammatory reactions. When accompanied by dentin formation at the pulp-dentin interface, these events may eliminate early bacterial infection and block the path of its progression.

After a protracted period of chronic inflammation, unchecked bacterial invasion most frequently causes irreparable chronic pulp inflammation. As a result, pulp necrosis, root canal infection, and periapical illness may develop. After the dental professional removes microorganisms and the pulp immune system neutralises intratubular diffusing components, both of which reduce the production of proinflammatory mediators, pulp inflammation, also known as "pulpitis," typically subsides. However, pulpal inflammation may persist after dental treatment and develop into a low-grade, chronic condition if the caries lesion is close to the dentin-pulp interface. Similar to other connective tissues, this chronic inflammation is to blame for the permanent loss of normal tissue function and the decline in the body's ability to defend itself against future injuries. The formation of a barrier of reactionary dentin by the original living odontoblasts and/or reparative dentin by newly differentiated odontoblast-like cells in animal models enables complete pulp healing in some cases.

Dentin neoformation lowers the risk of long-term discomfort from external bacterial or chemical agents by shielding the underlying pulp from dentin infection and the biomaterial used for crown fillings. It is plausible to assume that speedy reactionary or reparative dentin production starts, pulp healing happens quickly, and health is restored. Determining molecular and cellular agents that might reduce immune and

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inflammatory processes in the pulp and hasten the return of tissue homeostasis and health after the bacterial infection has been treated is significant from a clinical perspective. These substances ought to aid in preventing the development of pulp inflammation into a chronic condition. It's critical to develop a thorough understanding of the processes that launch and regulate the early stages of human pulp antibacterial defence and dentinogenesis-based reparative mechanisms in caries-affected human teeth in order to recognise these agents.

Conclusion

Numerous *in vitro* investigations have demonstrated that when exposed to PAMPs from Gram-positive bacteria, odontoblasts release inflammatory cytokines and chemokines. An acute-phase protein known as Lipopolysaccharide-Binding Protein (LBP) is known to reduce the generation of proinflammatory cytokines by activated macrophages. Several bacterial cell wall components, including lipopolysaccharides, lipoteichoic acids, lipopeptides, and peptidoglycan, have been shown to be resistant to LBP's ability to bind to host cells. In order to better understand the molecular effectors and regulators of human dental pulp immunity and assess their therapeutic potential to encourage the restoration of dental pulp homeostasis and health, more research is necessary.