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Commentary

Immune Factors and the Resistance against Bacterial Invasion in Dental Caries

Cuzalina Rasteau*

Department of Dentistry, the University of Sydney, Sydney, Australia

ABOUT THE STUDY

Dental caries, commonly known as tooth decay, is a major public health problem affecting individuals of all ages worldwide. Caries is a complex process initiated by a bacterial biofilm that forms on the tooth surface and progresses through the enamel and dentin layers, ultimately reaching the dental pulp. The dental pulp is a vital tissue that contains nerves, blood vessels, and connective tissue, and plays a crucial role in tooth function and maintenance. Therefore, the defence and repair mechanisms of the dental pulp against caries are essential for tooth survival.

The dental pulp defence system comprises of physical, chemical, and biological mechanisms that prevent or limit the penetration of bacteria and their toxins into the pulp tissue. The physical barrier is provided by the enamel and dentin layers that cover and protect the pulp. However, if the integrity of these protective layers is compromised by caries or trauma, the pulp becomes exposed to the oral environment, making it vulnerable to bacterial invasion. To counter this, the dental pulp contains an innate immune system that includes various cells, such as fibroblasts, macrophages, dendritic cells, and odontoblasts, that can detect and respond to bacterial invasion.

The odontoblasts are the primary cells responsible for the pulp's defence and repair mechanisms. They are specialized cells that form a layer at the pulp-dentin interface and secrete a unique extracellular matrix called dentin. Dentin is a mineralized tissue that acts as a barrier to bacterial invasion by blocking the dentinal tubules that communicate with the pulp. When the dentin is exposed to bacteria, the odontoblasts secrete a variety of immune factors, such as cytokines, chemokines, and antimicrobial peptides that recruit immune cells to the site of infection and activate them to destroy the invading bacteria.

In addition to the innate immune system, the dental pulp also has an adaptive immune system that can generate a specific immune response against the invading bacteria. This response involves the activation of B and T lymphocytes that produce antibodies and cytokines, respectively, to neutralize and eliminate the bacteria. The adaptive immune response is crucial for long-term protection against recurrent infections and the development of immunological memory.

Despite the dental pulp's defence mechanisms, caries can still progress to the point where the pulp tissue is irreversibly damaged, resulting in pulpitis or pulp necrosis. Pulpitis is an inflammatory response of the pulp tissue to bacterial invasion, characterized by pain, swelling, and increased blood flow. The pulp's response to pulpitis is to initiate reparative processes to repair the damaged tissue and protect the remaining pulp tissue. This process is called pulp healing and involves the formation of a reparative dentin matrix by the odontoblasts. The reparative dentin matrix is similar to the original dentin but has a different composition and structure, making it less resistant to caries and mechanical stress.

If the pulpitis is severe or left untreated, it can progress to pulp necrosis, which is the death of the pulp tissue. Pulp necrosis can result in the loss of tooth vitality, apical periodontitis, and even systemic infections. The dental pulp's defence and repair mechanisms against pulp necrosis involve the recruitment of immune cells to remove the dead tissue and initiate healing. The odontoblasts also produce a matrix that acts as a scaffold for the growth of new tissue, including nerve and blood vessels.

In conclusion, dental caries is a multifactorial disease that affects the oral health and quality of life of individuals worldwide. The dental pulp plays a crucial role in tooth function and maintenance, and its defence and repair mechanisms against caries are essential for tooth survival. The dental pulp's defence system comprises of physical, chemical, and biological mechanisms that prevent or limit bacterial.

Correspondence to: Cuzalina Rasteau, Department of Dentistry, the University of Sydney, Sydney, Australia, E-mail: c.rasteau@gmail.com

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