Periodontitis

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Keywords: periodontitis; periodontal disease; short-chain fatty acids

1. Definition

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2. Introduction

Periodontitis is characterized by a complex inflammatory response triggered by the presence of a dental biofilm. This bacterial biofilm is able to elicit a dysbiosis in the subgingival microbiome leading to the destruction of the periodontal supporting tissues and eventually tooth loss. Metabolites released by periodontopathic bacteria are capable of provoking an immune response inducing the influx of neutrophils and macrophages to the gingival crevice and epithelium. An excessive migration of leukocytes facilitates the periodontal breakdown and is often associated with the initiation and progression of periodontal diseases. Therefore, it can be assumed that the release of secondary metabolites may play a crucial role in periodontal disease. However, the specific role of these metabolites remains unclear.

3. Latest Research

A systematic review [1] showed that short-chain fatty acids, a bacterial metabolite, may negatively affect the viability of oral epithelial cells by activating a series of cellular events that include apoptosis, autophagy, and pyroptosis. Short-chain fatty acids impair the integrity and presumably the transmigration of leucocytes through the epithelial layer by changing junctional and adhesion protein expression, respectively. Short-chain fatty acids also affect the expression of chemokines and cytokines in oral epithelial cells. Future research needs to identify the underlying signaling cascades and to translate these in vitro findings into preclinical models.

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