Neutrophil recruitment in periodontal disease

Inflammation is our body´s immediate response to microbe invasion or tissue injury. Consisting of an intricate network of inflammatory cells, tissues and biological processes, inflammation is vital for elimination of unwanted intruders and initiation of tissue repair. Neutrophils are the first inflammatory cells to arrive at the infected or injured site, where they engulf microbes and cell debris or capture intruders by release of web-like DNA structures. For these purposes neutrophils leave the blood stream at the vicinity of the affected tissues and migrate further towards their prey, guided by a series of chemotactic signals. The critical role of neutrophil recruitment from blood to tissue can be illustrated by the fact that genetic defects disrupting this process result in recurrent severe infections. The symptoms of such disorders typically also include destructive inflammation of the tooth-supporting structures, i.e., periodontitis. Periodontitis is a microbe initiated inflammatory disease leading to deepening of gingival pockets and degradation of alveolar bone; which may eventually result in tooth loss. Neutrophils seem to play an important role in the maintenance of periodontal health as insufficient neutrophil numbers or defect neutrophil functionality, as seen in patients with rare genetic defects, often lead to rapidly progressing periodontitis. Moreover, although representing a minor fraction of the leukocytes in the periodontal lesion, neutrophils are the dominating cell type in the inflammatory exudate of the periodontal pocket.

This PhD thesis describes the neutrophil journey from circulation to the periodontal pocket, with the intention of adding new insights regarding the nature of bacteria derived chemotactic signals and neutrophil subsets operating at this site.

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