Opinion
Volume 8:2, 2022

Oral Health Case Reports

ISSN: 2471-8726 Open Access

Periodontal Ecology and Human Neutrophils

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Introduction

We are continually exposed to bacterial threats as humans through the air we breathe, our skin, and the food we eat. Our innate immune system, on the other hand, has developed to defend us from the persistent threat of infection posed by constant exposure to alien germs at each of our body's accessible entry zones, allowing us to live a healthy life. Not all of our experiences with these foreign 'animalcules' end in a threat to human health. Furthermore, the symbiotic relationship between the host and commensal microbiota has received increasing attention in recent years and has been shown to be advantageous to our health. The host and its related, extremely complex environment are in a fragile equilibrium.

About the Study

Despite the fact that the mouth cavity is a strongly populated part of human body with a diversified microbial population, its interaction with the host immune system is poorly known when compared to studies on the gut microbiome. Neutrophils are the key innate immune cell that is drawn to the mouth cavity in large numbers and is crucial for maintaining healthy periodontal tissue. Due to its intimate interaction with the oral biofilm population, the junctional epithelium, a specialised tissue that surrounds the tooth, creates a chemotactic interleukin gradient, which results in continual recruitment of neutrophils to the gingival sulcus.

Because prenatal illnesses including leukocyte adhesion deficit, Chediak-Higashi syndrome, Papillon-Lefèvre syndrome, and chronic/cyclic neutropenia, all of which impede neutrophil recruitment, higher periodontitis severity has been found. Pathogens have evolved mechanisms to evade neutrophil clearance in chronic inflammatory diseases like periodontitis, resulting in neutrophil accumulation in periodontal tissue and the potential release of the neutrophil arsenal into the extracellular space, which can cause tissue damage and, in severe cases, bone loss.

While neutrophils' oxidative burst response is an effective technique for clearing anaerobic bacteria infection, other periodontal infections, such as Porphyromonas gingivalis, are resistant to oxidative death. Hyperactive/primed neutrophils can also predispose people to periodontitis. The production of reactive oxygen intermediates, numerous cationic peptides, and enzymes such as matrix metalloproteinases characterises the heightened response of these neutrophils, resulting in greater tissue damage and positioning the neutrophil as a periodontitis culprit.

To sustain periodontal health, a careful balance between neutrophil activity and bacterial assault must be maintained. The significance of human neutrophils in maintaining a healthy oral mucosa is discussed in this review, as well as evidence for how cell interactions with periopathogens can alter the homeostatic balance into a dysbiotic environment, favouring a chronic inflammatory situation that leads to periodontitis. The amicable cooperation created with the indigenous symbiotic oral population enhances the sentinel movement of neutrophils via the junctional epithelium into the gingival space in the oral cavity. The number of neutrophils patrolling the junctional epithelium is dramatically increased by colonisation with commensal bacteria, according to animal experiments utilising germ-free mice.

The great porosity of the junctional epithelium, as well as the IL-8 chemotactic gradient created there, aid neutrophil migration to this location. The oral population populating the tooth and the junctional epithelium will be separated by neutrophils, who will establish a protective barrier. However, other elements of neutrophil activity can be influenced by the microbiome. Because studies in GF mice show a considerable drop in neutrophil counts, it is now established that the presence of the resident microbiota contributes to the steady state of neutrophil production. Neutrophils' interactions with metabolites or byproducts produced by the surrounding microbial population, such as short-chain fatty acids, are crucial for sustaining both health and function.

Furthermore, the microbiota contributes to the active phenotype of old neutrophils in circulation and aids in their clearance, offering new insight into why these cells have such a limited life span in circulation. The relationship between neutrophils and the indigenous, or commensal, symbiotic bacteria population in periodontal health is strictly regulated to avoid tissue injury. The lack of the oral commensal microbiota has little effect on the structure of gingival tissue, according to studies comparing GF mice with specific-pathogen free animals; this is in striking contrast to the gut commensal microbiota's substantial function in the structural creation of intestinal tissue.

The chemotactic receptor CXCR2 enhances neutrophil recruitment to periodontal tissue, as revealed in knockout mice. Because neutrophils are missing from the junctional epithelium of CXCR2 knockout mice, yet they are present in the blood arteries, this was discovered. Both CXCL1 and CXCL2, ligands of the CXCR2 receptor, are expressed in the junctional epithelium of GF mice; however, only CXCL2 expression levels, not CXCL1, are significantly higher in the epithelium of SPF mice as compared to GF animals. By specifically boosting the production of the powerful neutrophil chemokine CXCL2, the presence of the indigenous oral bacterial population increased neutrophil recruitment to periodontal tissue [1-5].

Conclusion

CXCL2 expression and neutrophil distribution in distinct locations of the junctional epithelium across the tooth were compared in GF, SPF, and GF mice that were gavaged with single commensal oral bacteria such Streptococcus sp. or Lactobacillus sp. In the periodontium of SPF mice and bacteria-gavaged GF animals, neutrophils have a similar location pattern, but it differs from the pattern found in GF mice. Furthermore, in bacteria-colonized mice, there is a positive association between neutrophil location and CXCL2 expression levels in the junctional epithelium.

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Received: 17 February, 2022, Manuscript No. ohcr-22-65828; **Editor Assigned:** 20 February, 2022, PreQC No. P-65828; **Reviewed:** 25 February, 2022, QC No. Q-65828; **Revised:** 04 March, 2022; Manuscript No R-65828; **Published:** 08 March, 2022; DOI: 10.37421/2471-8726.2022.8.50

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How to cite this article: Green, Oliver Lee. "Periodontal Ecology and Human Neutrophils" Oral Heath Case Rep 8 (2022): 50.