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Porphyromonas gingivalis-Induced Periodontitis and its Contribution to Rheumatoid Arthritis Autoimmunity

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DESCRIPTION

Periodontitis is a chronic inflammatory disease of the gum and bone surrounding the teeth caused by bacterial infections. One of the bacteria frequently found in periodontitis is *Porphyromonas gingivalis* (*P. gingivalis*). On the other hand, Rheumatoid Arthritis (RA) is a chronic autoimmune disorder characterized by inflammation and joint destruction. Studies have shown that there is a correlation between RA and periodontitis, particularly *P. gingivalis* periodontitis. The correlation between periodontitis and RA is thought to be due to molecular mimicry, where bacterial proteins from *P. gingivalis* share similar epitopes with human proteins, particularly citrullinated peptides. Citrullination is a post-translational modification where the amino acid arginine is converted to citrulline. This modification can lead to the generation of citrullinated peptides that can be recognized as non-self by the immune system and trigger an autoimmune response. *P. gingivalis* has been shown to possess enzymes capable of citrullinating peptides, leading to the generation of Citrullinated Bacterial Peptides (CBPs) that can be recognized by RA-specific autoantibodies.

Studies have shown that individuals with RA have higher levels of antibodies against *P. gingivalis* compared to healthy individuals. This suggests that *P. gingivalis* may be involved in the development of RA. Furthermore, *P. gingivalis* has been shown to be present in the synovial fluid and tissues of individuals with RA, suggesting that the bacterium may be involved in the pathogenesis of the disease. Studies have also shown that individuals with periodontitis have a higher risk of developing RA. One study found that individuals with severe periodontitis were nearly three times more likely to develop RA compared to those without periodontitis.

Another study found that individuals with *P. gingivalis* periodontitis were at a higher risk of developing RA compared to those without the bacterium. In addition to molecular mimicry, *P. gingivalis* has been shown to directly contribute to the development of RA. The bacterium can activate dendritic cells and macrophages, leading to the production of pro-inflammatory cytokines such as Interleukin-1 (IL-1) and Tumor Necrosis Factor-Alpha (TNF-alpha). These cytokines are known to be involved in the pathogenesis of RA. P. gingivalis has also been shown to induce the differentiation of osteoclasts, which are involved in bone destruction in RA.

Moreover, *P. gingivalis* can also affect the gut microbiota, leading to dysbiosis. Dysbiosis refers to an imbalance in the microbial community in the gut, which can lead to inflammation and autoimmune disorders. *P. gingivalis* has been shown to increase the abundance of opportunistic pathogens in the gut, leading to dysbiosis.

The association between periodontitis and RA is not limited to *P. gingivalis*, as other periodontal pathogens such as *Aggregatibacter actinomycetemcomitans* and *Tannerella forsythia* have also been implicated in the pathogenesis of RA. Furthermore, periodontitis has been shown to be associated with other systemic diseases such as cardiovascular disease and diabetes. The treatment of periodontitis may also have an impact on the management of RA. One study found that treating periodontitis in individuals with RA resulted in a reduction in the number of swollen and tender joints, as well as a decrease in the levels of C- Reactive Protein (CRP), which is a marker of inflammation.

The *P. gingivalis* periodontitis is a risk factor for the development of RA. The molecular mimicry between *P. gingivalis* and human proteins, as well as the direct activation of immune cells and the induction of dysbiosis, suggest that the bacterium may play a role in the pathogenesis of RA. Therefore, periodontal health should be considered as an important factor in the management of RA. Periodontitis can be treated by mechanical removal of the bacterial biofilm through scaling and root planing, as well as by the use of antibiotics or antimicrobial agents. However, the use of antibiotics in the treatment of periodontitis has been controversial due to the development of antibiotic-resistant bacteria. Therefore, alternative treatments such as photodynamic therapy and probiotics have been investigated.

Photodynamic therapy involves the use of a photosensitizing agent and a light source to generate reactive oxygen species that can kill bacteria. This therapy has been shown to be effective in reducing the number of periodontal pathogens, including *P. gingivalis*, and improving periodontal health. Probiotics, on the other hand, are live microorganisms that can provide health benefits when consumed in adequate amounts. Probiotics have been shown to improve gut health and modulate the immune system. Studies have shown that certain strains of probiotics can reduce the levels of *P. gingivalis* and other periodontal pathogens, as well as improve periodontal health.

In addition to treating periodontitis, the use of Disease-Modifying Anti-Rheumatic Drugs (DMARDs) and biologic agents has been effective in managing RA. DMARDs such as methotrexate and sulfasalazine can slow down the progression of joint damage in RA. Biologic agents such as Tumor Necrosis Factor (TNF) inhibitors and Interleukin-6 (IL-6) inhibitors can target specific cytokines involved in the pathogenesis of RA. It is important to note that the use of DMARDs and biologic agents may increase the risk of infections, including periodontitis. Therefore, individuals with RA who are receiving these medications should be closely monitored for periodontal health.

In conclusion, the correlation between *P. gingivalis* periodontitis and RA suggests that periodontal health should be considered as an important factor in the management of RA. Treating periodontitis may not only improve periodontal health but also have a positive impact on the management of RA. Alternative treatments such as photodynamic therapy and probiotics may also be considered in the treatment of periodontitis. However, the use of DMARDs and biologic agents in the management of RA may increase the risk of infections, including periodontitis, and should be closely monitored.