



The Role of the Immune Response in Chronic Marginal and Apical Periodontitis

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Abstract

The immune response is a complex, dynamic and strongly individual biologic network that plays an essential role in the pathogenesis of chronic apical and marginal periodontitis. Recent research in the field of periodontology has indicated that the major determinant of susceptibility to disease is the nature of the immunoinflammatory response as marginal periodontal tissue damage is thought to be primarily mediated by the host reaction. Whether the same rules apply for the development of apical periodontitis, however, remains largely unexplored. This review aims to draw parallels between the pathogenesis of chronic periodontitis of endodontic and marginal origin, outline the evidence for the destructive role of immune response in chronic marginal periodontitis and raise questions about its role in chronic apical periodontitis. It would be worthy to further explore the impact of the immune system on the characteristics and progress of these diseases and transfer some of the scientific models from the field of periodontology to the field of endodontics. Research in this area could lead to a more comprehensive understanding of the dynamics of apical and marginal periodontitis and lay the foundation of new personalized treatment strategies.

Keywords

asymptomatic apical periodontitis, chronic marginal periodontitis, pathogenesis, host susceptibility

INTRODUCTION

The immune response is an elaborate biologic network that integrates the complex and interconnected processes of pathogen recognition, innate immunity, and adaptive immunity.¹ This intricate constellation of processes is flexible, dynamic and strongly individual.

The immune system plays an essential role in periodontal homeostasis and the pathogenesis of inflammatory periodontal diseases.² The immune response is protective in its intent, but once it becomes dysregulated, inappropriate, persistent, or excessive in some way, its nature turns destructive.³

In the field of periodontology, the challenge presented by the polymicrobial plaque biofilm triggers immune and inflammatory processes mediated by a large number of pro-inflammatory and anti-inflammatory cytokines and

enzymes. In the field of endodontics the same process occurs as the result of the egress of bacteria and their by-products from the apical foramen to the periapical tissues. Marginal and apical periodontal diseases are considered local inflammatory reactions in response to bacterial challenge. They are multifactorial diseases, their dynamic influenced by the initiating factor. However, the disease also occurs in the context of other host and environmental factors. While the variability in periodontal pathogens undoubtedly conducts the periodontal disease progression, a less explored topic remains the role of the individual variances in the host immune system. Landmark publications and advances in cell biology and immunology have resulted in paradigm shifts in our understanding of periodontal diseases. Recent research in the field of periodontology has indicated that the major determinant of susceptibility to disease is the nature of the immunoinflammatory response.³ Marginal

periodontal tissue damage is thought to be primarily mediated by the host's reaction.^{4,5} Whether the same rules apply for the development of apical periodontitis, however, remains largely unexplored. This review aims to outline the similarities in the pathogenesis of chronic periodontitis of endodontic and marginal origin, synthesize the existing evidence for the destructive role of the immune response in chronic marginal periodontitis and raise questions about its involvement in chronic apical periodontitis.

Chronic apical and marginal periodontitis

Chronic marginal periodontitis (CMP) and asymptomatic apical periodontitis (AAP) are two of the most common chronic inflammatory diseases in the field of dental medicine. They both represent an immune response of bacterial aetiology that affect the periodontal structures and are manifested clinically by bone resorption.⁶ While CMP involves the tooth-supporting soft and hard tissues, AAP affects the periapical periodontal structures.⁶

The etiology, pathogenesis, and histopathology of AAP are similar to that of CMP. Infection plays a causative role in the development of the diseases. Bacteria from the endodontic or periodontal biofilm initiate and perpetuate the inflammatory responses. Bacterial colonization of the periodontal pocket or the endodontic space, as well as the egress of bacterial toxins into the periapical tissues, activate the innate and adaptive immune system thus inducing

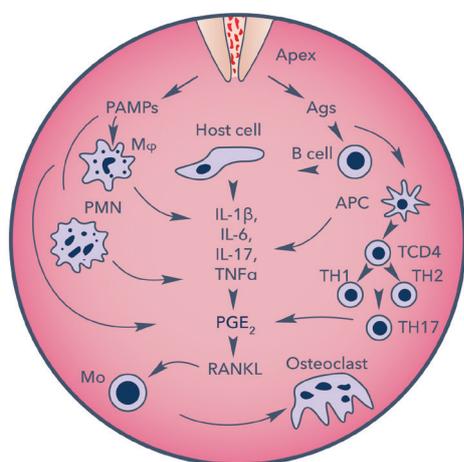


Figure 1. Pathogenic pathways of asymptomatic apical periodontitis.

a periodontal inflammatory reaction and consequent bone destruction.⁷ The immunologic response in the marginal and apical periodontium is mediated by the same cell types. These cells release identical inflammatory mediators and induce an alteration in the physiology and metabolism of the periodontal tissues. CMP and AAP are characterized by the prolonged release of proinflammatory mediators and consequent progressive bone resorption (Fig. 1).

Pathogenesis of CMP and AAP

The pathogenesis of chronic periodontitis involves both the innate and adaptive immune response, as the activation of the innate immunity is a prerequisite for the initiation of the adaptive immunity.⁸ The periodontal host response is highly complex, as it combines both protective and destructive elements.^{9,10} Different theories attribute this paradox to various aspects of the immune response.

Innate immunity in periodontal pathogenesis

The innate immunity comprises a broad range of cytokines, chemokines and cell surface receptors produced by effector cells (macrophages, dendritic cells, neutrophils, monocytes, epithelial cells and endothelial cells). Microbe-associated molecular patterns (MAMPs), like lipopolysaccharide (LPS), bacterial nucleic acids, fimbriae and proteases, activate resident and infiltrating host cells in the periodontium. The effector cells identify and respond to MAMPs via pattern recognition receptors. Once recognized by the host cells they activate diverse intracellular signalling pathways. The early activation of the innate immune response is in the form of elicitation of cytokine responses. The cytokines triggers changes in the periodontal cells, as well as turnover and degradation of components of the extracellular matrix (ECM).¹¹ The breakdown products of ECM components activate the macrophages and result in further, self-sustaining cytokine secretion.¹² The upregulated cytokine expression results in vascular changes, polymorphonuclear neutrophils (PMNs) activation and migration, ultimately leading to osteoclastogenesis and osteoclast activation.¹³

IL-1 β and TNF- α are some of the key pro-inflammatory cytokines in the innate immune response. They upregulate the expression of other inflammatory mediators to potentiate the inflammatory response. They activate leucocytes and endothelial cells, stimulate the production of chemokines, prostaglandins and matrix metalloproteinases (MMPs). IL-1 β has a major role in the pathogenesis of periodontal disease as a potent inducer of bone resorption and of connective tissue degradation via the induction of MMPs.^{14,15} TNF- α is a pro-inflammatory cytokine that also induces bone resorption and upregulates prostaglandin E₂ (PGE₂) and MMP secretion.

According to Offenbacher et al.¹⁶ bacterial products like LPS induce the expression of IL-1 β and TNF- α , upregulating the production of cytokines, chemokines and cyclooxygenase products which amplify the inflammation. When the levels of inflammatory mediators reach a certain threshold the pathways of bone resorption are activated.¹⁷ Soft and hard tissue degradation commences, with MMPs breaking down the connective tissue. IL-1 β and TNF- α stimulate PGE₂ release from fibroblasts and osteoblasts, which triggers bone resorption.¹⁸

Innate immune response and periodontal destruction

The balance and dynamics between pro- and anti-inflammatory activities in CMP has been a subject of intensive research. The nature of the inflammatory response varies between individuals, as well as over time, which is represented by the different degrees of tissue damage. Some people are therefore considered more susceptible to periodontal destruction than others. The innate immune response is initially protective up to a certain level of inflammatory mediators. That critical level is referred to as a disease threshold, as once crossed it initiates disease progression and the clinical signs and symptoms of periodontal disease.³

The innate immune-inflammatory response is determined by both the microbial challenge and the host defense system. The levels of mediators produced depend on the individual's monocyte responsiveness and the pathogenicity of the colonizing microflora. Microbial burden fluctuations impact the inflammatory response dynamics - low pathogenic bacteria elicit low inflammatory response, while high pathogenicity of the microflora upregulates the release of inflammatory mediators and amplifies the immune response.^{19,20} Once their levels cross the disease threshold periodontal tissue breakdown commences. Prolonged increased levels of inflammatory mediators also trigger the pathways of bone resorption.

These observations led to the development of the theory of host susceptibility in the field of periodontology. This theory holds that the inflammatory mediator levels are mainly influenced by the individual monocyte responsiveness. It has long been known that the quantitative and qualitative differences in immune responses between individuals are a feature of human development and evolution.²¹ The theory of host susceptibility suggests marked inter-individual differences in released mediator levels in response to the same bacterial challenge. According to this model some individuals present with a hyper-responsive phenotype that accounts for an elevated expression of inflammatory mediators at given bacterial challenge.²² Clinically, this is manifested by an increased inflammatory response and CMP disease progression at a lower microbial load threshold. In contrast, hyporesponsive individuals produce lower levels of inflammatory mediators in comparison to normal individuals and are therefore resistant to the progression of chronic inflammatory diseases as periodontitis.

The individual's ability to produce inflammatory mediators can be evaluated by measuring the levels of inflammatory mediators released from peripheral blood monocytes (PBMCs). PBMCs are precursors to macrophages, DCs and osteoclasts - cells which orchestrate the initial response to microbes. Initial studies focused on the release of PGE2 from circulating monocytes as it is a key mediator for the initiation and perpetuation of bone resorption.^{23,24} These findings were later confirmed by further investigation of the secretion of IL-1 and TNF- α by stimulated PB-

MCs.^{22,25,26} According to Champagne et al.²⁷ monocytes isolated from periodontitis patients produced higher levels of PGE2 than healthy controls. Monocytes isolated from patients with severe forms of periodontal diseases produced even higher levels of PGE2 than those isolated from patients with milder forms of the disease.²⁷ Remarkably, PGE2 levels produced in different periodontitis patient groups remained fairly constant at all doses of LPS.^{23,28} The authors proposed that the amount of inflammatory mediator produced by an individual's monocytes in response to increasing doses of bacterial challenge is a characteristic of that individual's host response. It is hypothesized that this is the underlying reason of variances in the periodontal response to subgingival plaque in individuals.

Other experimental models focused on PBMCs suggest that certain subsets of circulating monocytes may represent a hyper-inflammatory phenotype and exhibit a distinct cytokine secretion profile.²⁹ However, there is only limited data on monocyte subsets in periodontal disease.³⁰ Another study suggests that destructive periodontal disease may also be attributed to the dysregulation of inhibitors, rather than the overproduction of IL-1 and TNF- α .³¹

The constancy of the described immune response is also a subject of discussion. According to some studies the nature of the inflammatory response is governed by genetic factors, environmental and behavioral factors.^{27,32} They can affect the individual's dose-response curve, shifting the host response towards increased or decreased responsiveness.²⁷ Whether this shift will have a clinical impact is dependent on its severity and mediator levels produced.

Adaptive immune response in periodontal pathogenesis

Cytokines produced in the initial innate immune response activate the adaptive immune response pathways via T- and B-cell recognition of specific antigen structures. The cytokine milieu determines the differentiation of particular effector T-cell subsets. Antigen presenting cells (APCs) present specific antigens to naïve CD4+ T cells (Th0) cells. They differentiate into different subsets of CD4+ T cells like Th1, Th2, Th17, Treg cells. Each T-cell subset is associated with its unique cytokine secretion profile that regulates different aspects of the immune response and affects the dynamics of periodontal disease progression.

Th1 cells produce IFN- γ , a cytokine responsible for the activation of the cell-mediated immunity against pathogenic microorganisms that comprises of phagocytosing macrophages and cytotoxic cell like NK cells and CD8+ T cells. Th2 cells regulate humoral (antibody-mediated) immunity and mast cell activity through the secretion of cytokines (IL-4, IL-5, and IL-13), and coordinate the B-cell response. Periodontal tissue breakdown dynamics have long been attributed to the dynamic interaction between these two subsets.

Treg cells have an immunosuppressive action that is mediated by the secretion of transforming growth factor- β

(TGF- β) and IL-10. They have a regulatory function on other T-cell subsets. These cells are increased in periodontitis lesions of both CMP and AAP and may therefore have a role in disease pathogenesis.³³ IL-10 is a regulatory mediator with a dual role. It suppresses Th1 and Th2 responses, macrophage and DC functions and downregulates cytokine production from Th1 cells, Th2 cells, PMNs, and NK cells. However, it can also activate B cells. These different aspects of IL-10 biology likely depend on the local cytokine environment.

Th0 cells can also differentiate into Th17 cells. They are believed to amplify proinflammatory responses in CMP and AAP. They produce pro-inflammatory cytokines IL-17 and IL-22 which mediate the immune responses against extracellular bacteria.³⁴ IL-17 has a number of activities in common with pro-inflammatory cytokines, such as IL-1 β and TNF- α , has a synergistic activity with them and induces their expression.³⁵ IL-17 activates neutrophil infiltration in periodontal disease and upregulates MMPs. However, a protective role of IL-17 in bone homeostasis has also been suggested, possibly via effects on neutrophil function.³⁶

Adaptive immunity and periodontal destruction

The cellular and molecular aspects of the adaptive immunity interact in a diverse and complex way to determine the nature of CMP and AAP. The immune-inflammatory response consists of multiple pro- and anti-inflammatory pathways entangled in positive and negative feedback loops. This duality has been investigated in CMP and is attributed to the cellular aspect of the adaptive immunity - cell-mediated immunity (CMI). It orchestrates both the protective and destructive components of the disease.

Traditionally, the periodontal host-pathogen relationship was described by a linear model, in which the bacteria are initiators of the inflammatory process and disease progression is orchestrated by the adaptive host response. This theory undervalued the complex nature of the process.³⁷ Gaffen et al.³⁵ proposed a circular model for CMP pathogenesis according to which bacteria are necessary for both initiation and progression of the disease. They constantly shape the T-cell response through differential TLR-mediated activation of APC and the secreted cytokines. The dysregulation of the host response triggers a persistent inflammation that is ineffective in resolving the infection. It in turn provides the bacteria with nutrients and new niches for colonization through the generation of periodontal pockets. The circular model offers a more comprehensive and dynamic model of CMP pathogenesis.

The fate of the innate immune inflammatory process in CPM has long been attributed to the “protective Th1/destructive Th2” model underlying the dynamic interactions between T-cell subsets and their impact on periodontal disease. According to this model, Th1 cells provide a protective IL-12/IFN γ -stimulated cell-mediated immunity and

inhibit osteoclastogenesis.³⁸ They are associated with early and stable periodontal lesions and do not trigger periodontal tissue breakdown. Th2 cells activate B-cells and characterize non-protective antibody responses and progressive periodontal lesions possibly through enhanced B-cell-derived IL-1 β .⁹

However, the Th1/Th2 model is not sufficient to explain the role of T-cells in CMP pathogenesis.³⁵ There is no consistent evidence of the existence of distinct Th1 and Th2 cell populations, as infiltrating T-cells can simultaneously express mRNA for both Th1 and Th2 cytokines and regulatory cytokines.³⁵ The dichotomous Th1 vs. Th2 model is insufficient to accurately describe the host-pathogen interactions in the periodontium.

A clarifying insight into periodontal pathogenesis was given by the discovery of the Th17 subset and its role in the destructive phase of CMP disease. Th17, rather than Th1, is implicated as the specialized osteoclastogenic lymphocyte that links T-cell activation to bone resorption.³⁹ These reports give a more nuanced understanding of host-pathogen reactions in the periodontium and open the debate of the role of T-cells in CMP under a new, extended Th1/Th2/Th17 paradigm.^{35,40} However, innovative research models are needed for its investigation in the pathogenesis of AAP.

Marginal and apical periodontitis pathogenesis

The similarities in the pathogenesis of chronic marginal and apical periodontitis encourage us to draw parallels between the two conditions. Research focus on marginal periodontal pathogenesis has improved our understanding of the disease over the last few years, as research discoveries continue to lift the veil on this complex process.

CONCLUSION

It would be worthy to further explore the impact of the immune system on the characteristics and progress of AAP development and transfer some of the scientific models from the field of periodontology to the field of endodontics. To date, no studies regarding the correlation between the hyperinflammatory phenotype and AAP dynamics have been found in literature. Research in this area could lead to validation of a prognostic model for assessing AAP's dynamics as well as commencing the implementation of a personalized treatment approach and innovative therapeutic agents for the regulation of mediator secretion in hyperinflammatory patients.

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Роль иммунного ответа при хроническом маргинальном и апикальном периодонтите

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Абстракт

Иммунный ответ представляет собой индивидуальную, динамичную и строго индивидуальную биологическую сеть, которая играет чрезвычайно важную роль в патогенезе хронического апикального и маргинального периодонтита. Последние исследования в области пародонтологии показали, что основной детерминантой восприимчивости к заболеванию является характер иммуно-воспалительного ответа, так как считается, что маргинальное повреждение ткани пародонта опосредовано главным образом ответом хозяина. Однако остаётся вопрос, относятся ли те же правила к развитию апикального периодонтита. Этот обзор направлен на то, чтобы провести параллели между патогенезом хронического периодонтита эндодонтического и маргинального происхождения, наметить доказательства деструктивной роли иммунного ответа при хроническом маргинальном периодонтите и поставить вопросы о его роли в хроническом апикальном периодонтите. Также было бы целесообразно продолжить изучение влияния иммунной системы на характеристики и развитие этих заболеваний и перенести некоторые научные модели из области пародонтологии в область эндодонтии. Исследования в этой области также позволили бы глубже понять динамику апикального и маргинального периодонтита и заложить основы для новых персонализированных стратегий лечения.

Ключевые слова

бессимптомный апикальный периодонтит, хронический маргинальный периодонтит, патогенез, восприимчивость хозяина
