Pulp Inflammation Diagnosis from Clinical to Inflammatory Mediators: A Systematic Review

Marjorie Zanini, DDS, MSc,[*†] *Elisabeth Meyer, DDS,*[*†] *and Stéphane Simon, DDS, PhD, HDR*[*†‡]

Abstract

Introduction: Similar to other tissues, the dental pulp mounts an inflammatory reaction as a way to eliminate pathogens and stimulate repair. Pulp inflammation is prerequisite for dentin pulp complex repair and regeneration; otherwise, chronic disease or pulp necrosis occurs. Evaluation of pulp inflammation severity is necessary to predict the clinical success of maintaining pulp vitality. Clinical limitations to evaluating *in situ* inflammatory status are well-described. A molecular approach that aids clinical distinction between reversible and irreversible pulpitis could improve the success rate of vital pulp therapy. The aim of this article is to review inflammatory mediator expression in the context of clinical diagnosis. **Methods:** We searched PubMed and Cochrane databases for articles published between 1970 and December 2016. Only published studies of inflammatory mediator expression related to clinical diagnosis were eligible for inclusion and analysis. **Results:** Thirty-two articles were analyzed. Two molecular approaches were described by study methods, protein expression analysis and gene expression analysis. Our review indicates that interleukin-8, matrix metalloproteinase 9, tumor necrosis factor-α, and receptor for advanced glycation end products expression increase at both the gene and protein levels during inflammation. **Conclusions:** Clinical irreversible pulpitis is related to specific levels of inflammatory mediator expression. The difference in expression between reversible and irreversible disease is both quantitative and qualitative. On the basis of our analysis, *in situ* quantification of inflammatory mediators may aid in the clinical distinction between reversible and irreversible pulpitis. *(J Endod 2017;43:1033–1051)*

Key Words

Dental pulp, diagnosis, gene expression, inflammation, molecular pattern

Significance

Because of the limitations of accuracy of clinical diagnosis tools, outcomes after vital pulp therapy are unpredictable. Molecular-based diagnostic strategies are promising and may be relevant to determine proper clinical indications for vital pulp therapies and optimize prognosis.

The goal of any restorative or endodontic procedure should be to maintain dental pulp vitality and functionality without patient discomfort. Pulp tissue is necessary for tooth nutrition, innervation, and immunocompetency. Maintaining dental pulp vitality increases tooth mechanical resistance and long-term survival (1, 2). Therefore, pulp vital therapies such as indirect pulp capping, stepwise caries excavation, direct pulp capping, and pulp chamber pulpotomy may be preferable to root canal treatment in some situations. Unfortunately, the success of vital pulp therapy varies greatly, especially for direct pulp capping after carious excavation (31.8%–91.3%) (3, 4). Clinical early failures (within days or weeks) are multifactorial but certainly may be related to improper diagnosis of pulp disease. Indeed, poor pulp status evaluations may result in underestimation of pulp inflammation severity. This oversight can lead to irreversible pulp inflammation and pulp tissue necrosis, which results in spontaneous and persistent pain after therapy.

Proper evaluation of pulp inflammation and choice of appropriate materials before therapy are the keys to improving pulp vital therapy success. Tricalcium silicate–based cements have excellent sealing properties and bioactive properties because they induce good-quality hard tissue barriers (5–7). Accurate diagnosis requires knowledge of pulp inflammation severity and the likelihood of response to endodontic procedures. Intense or long-term pulpal inflammation that is due to unresolved infection or inflammation precludes regeneration (8). Because inflammation aids in pathogen elimination and repair stimulation, control of pulp inflammation severity is necessary for vital pulp therapy success (9, 10).

Currently, pain quality and history and responses to pulp sensitivity tests are the only clinical tools available to evaluate pulp inflammation severity (11, 12). Pulpal diseases are often clinically classified by using the criteria of the American Association of Endodontists (AAE) (13). The terms *reversible or irreversible pulpitis* simply reflect the intention of the practitioner to keep or remove vital pulp; therapy could be attempted on a tooth with reversible pulpitis, as defined by the absence of spontaneous pain or absence of pain after stimulation. Root canal treatment remains the therapy of choice for irreversible pulpitis because the pulp is too deeply inflamed to expect recovery.

Unfortunately, the AAE clinical categorization has limitations. Histologic observations show no correlation between clinical diagnosis and *in situ* pulp status (14, 15). Histologic evaluation of tissues clinically diagnosed as irreversible symptomatic pulpitis

From the *UFR d'odontologie, Université Paris Diderot, Paris, France; †Groupe Hospitalier Pitié Salpêtrière-Charles Foix, Paris, France; and ‡UMRS INSERM 1138 Team 5, Centre de recherche des Cordeliers, Paris, France.

Address requests for reprints to Dr Stéphane Simon, Oral Biology and Endodontixs, University of Paris Diderot (Paris 7), 75006 Paris, France. E-mail address: simendo@gmail.com

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do not show deep inflammation (16), whereas pulp necrosis may occur in asymptomatic or low-grade symptomatic patients whose disease could be wrongly diagnosed as reversibly inflamed. Therefore, pain characterization that is based on clinical tests seems inadequate to reliably differentiate between reversible and irreversible pulp inflammation. Because clinical diagnosis is unreliable, the outcome of vital pulp therapy is unpredictable, and practitioners may completely remove the pulp more often than is necessary to limit postoperative pain and infection.

Pulp inflammation involves several biological processes evaluable at the macroscopic, microscopic, and molecular levels. Macroscopic changes are mainly noticeable at the vascular level (eg, vasodilatation) (17, 18). Increased immune cell numbers are remarkable on microscopic examination (19). Release of multiple inflammatory biomolecules is also observed. The molecular immune response may precede the cellular immune response, suggesting that cytokines and other signaling molecules are synthesized and secreted by dentin pulp complex host cells before immune cell recruitment and activation (20).

The quality and quantity of these mediators are key to inflammatory evolution, especially with respect to the type of tissue immune response generated. Some mediators guide and amplify the inflammatory process. Type 1 cytokines (eg, interferon-γ, interleukin [IL]-2, IL-12, tumor necrosis factor [TNF]-α) orchestrate strong cellular immune responses, particularly intense phagocytic activity. Other mediators are responsible for tissue repair. Type 2 cytokines (eg, IL-10, IL-4) suppress macrophage activation and phagocytosis and stimulate B-cell proliferation and differentiation into plasma cells after resolution of cell-mediated inflammation (21, 22). Furthermore, the carious lesion model describes deeper carious lesions as having greater quantities of inflammatory mediators and more pulp inflammation (20, 23).

Inflammatory mediators orchestrate the inflammatory process inside the pulp tissue. Consequently, the molecular phase precedes the macroscopic and microscopic inflammatory changes; thus, it would be instructive to study their expression inside pulpal tissue both qualitatively and quantitatively relative to the severity of the pulpal disease. The aim of this study was to review articles concerning inflammatory molecule expression related to clinical diagnosis to identify key inflammatory mediators and describe their expression profile in inflammatory pulp disease.

Methods

We performed a literature review of expression of any inflammatory mediators related to clinical diagnosis to create a list of potential expression patterns. This systematic review was conducted by using Preferred Reporting Items for Systematic reviews and Meta Analyses

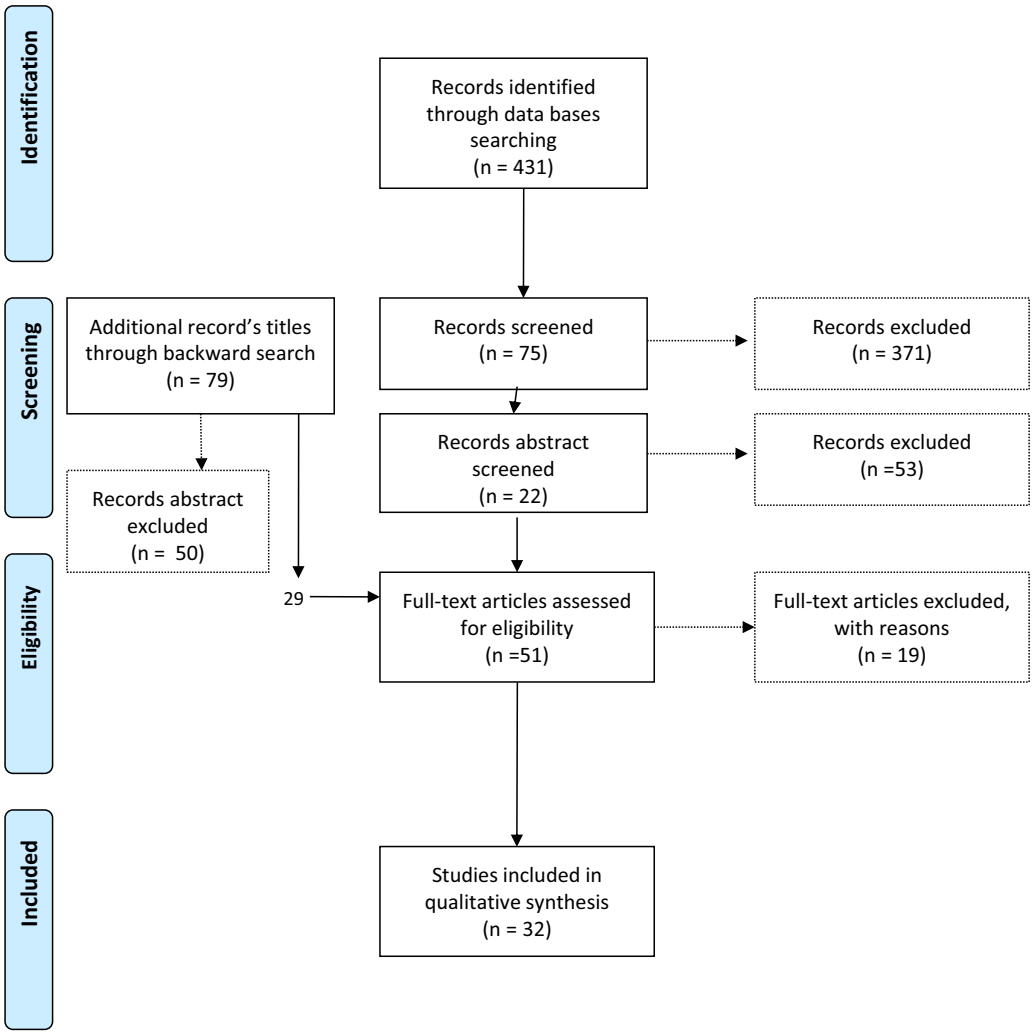

Figure 1. Flow diagram of records.

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