International Journal of
Molecular Sciences

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Molecular Mechanisms of Periodontal Disease 2.0

Guest Editor:

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Deadline for manuscript submissions:
closed (31 January 2022)

Message from the Guest Editor

Periodontitis is a chronic inflammatory disease characterized by lymphocytic infiltration and alveolar bone destruction, along with tooth loss. Such destructive inflammation is elicited by host innate and adaptive immune response to periodontal biofilm-associated multiple microorganisms. In addition, several inflammatory cytokines produced from lymphocytes, leukocytes, fibroblasts, and gingival epithelial cells in the context of host immune responses have been identified as key molecules inducing periodontal tissue destruction. More specifically, proinflammatory cytokines, including IL-6 and IL-17, facilitate the RNAKL expression level in fibroblasts or lymphocytes, which results in the induction of bone resorption. To that end, this Special Issue, which is a continuation of a previous successful issue on the molecular mechanisms of periodontal disease, focuses on novel immune reaction systems from the molecular level to the cellular level (Th1, Th2, Th17, Treg, and B cells activity, osteoclastogenesis, dendritic cells and monocytes immune response, the role of fibroblasts/epithelial cells in inflammation, etc.) in mouse periodontal disease models.

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Message from the Editor-in-Chief

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Journal Rank: JCR - Q1 (Biochemistry and Molecular Biology) / CiteScore - Q1 (Inorganic Chemistry)

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