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Periodontitis is a biofilm-induced chronic inflammatory disease leading to destruction of tooth supporting tissues and alveolar bone loss. The etiology of periodontitis depends on a complex interaction of host immune cells with periodontal pathogens that have outnumbered the non-pathogenic microflora in subgingival biofilm. Porphyromonas gingivalis is a late colonizer and modulates host immune responses to favor growth and survival of the entire oral biofilm. P. gingivalis can strategically invade gingival epithelial cells and infect underlying tissues. The initial interaction of P. gingivalis with the epithelium integrates both innate and adaptive immune responses to clear the infection. Several inflammatory mediators, such as cytokines and chemokines are secreted to attract immune cells to the site of infection. An exaggerated immuno-inflammatory condition at the gingival crevice leads to tissue degradation. In this thesis, we focus on developing and elucidating the role of novel tools for detection, prevention and treatment of P. gingivalis infection and periodontitis.