Periodontal disease initiation and progression occurs as a consequence of the host immune inflammatory response to oral pathogens. Tristetraprolin (TTP) is a zinc finger protein that binds to the distinct elements of multiple cytokine mRNAs and enhances degradation of specific target cytokine mRNAs. TTP is phosphorylated by the intracellular signaling pathways p38-MK2 mitogen-activating protein kinases (MAPKs) and may serve as a general mechanism of cytokine mRNA regulation. The objective of this presentation is to provide experimental evidence on how TTP and the p38-MK2 pathways can modify periodontal disease initiation and progression providing novel therapeutic targets for adjunctive management of chronic periodontitis.

In addition, other therapeutic targets including MAPK phosphatases (MKPs) that target the regulatory sites of MAPK kinases capable of negatively regulating MAPK activity to attenuate inflammatory cytokine response will be discussed. Both preclinical and in vitro data focused on how these pathways are specific for inflammatory bone loss and osteoclast formation will be discussed. Following this presentation, new foundation and translational significance of mRNA stability will be provided to the audience showing how insight of the functional nature of proteins to be targeted for future human studies that will modify innate immune cytokine expression for therapeutic benefit in the management of chronic periodontitis.