Periodontitis is a poly-microbial disease characterized by chronic inflammation of the gums and the eventual destruction of alveolar bone. The progression of gum disease into periodontitis is partially driven by a shift in the composition of the oral microbiota from symbiotic to a dysbiotic, more complex community that can evade killing while promoting inflammation. Neutrophils are white blood cells that act as the innate immune system’s first responders to infection. They play an important role in periodontitis because they are the main phagocytic cells in the periodontal pocket. During homeostasis, they can eliminate bacteria through the generation of reactive oxygen species and the microbicidal contents of their granules, among other mechanisms. However, oral pathogens have evolved defenses against neutrophil killing and further promote inflammation to harvest nutrients for bacterial growth. The newly appreciated bacteria *Filifactor alocis*, has been found in lesions of periodontitis patients, but not in the oral microbiota of healthy individuals. *F. alocis* is strongly resistant to killing and can manipulate neutrophil (PMN) effector functions. Suppressor of Cytokine Signaling 1 (SOCS1), is a protein that acts as a negative regulator of signaling. In PMNs subject to an *F. alocis* challenge, SOCS 1 mRNA is increased dramatically. RNA-sequencing results found the SOCS 1 mRNA goes up by twelve-fold after six hours in comparison to untreated cells. The RNA-seq was verified by qPCR, providing similar results. Confocal imaging and SOCS1 immunostaining was used to find intensity of fluorescence of the SOCS1 protein in *F.a* challenged cells. F.a was tested at 1hr, 3hr, and 6hr time points, along with a negative control of untreated cells and a positive control of cells stained with LPS or FSL-1. The results indicate SOCS 1 is increased in *F.a* challenged PMNs. This shows *F.a* likely can manipulate neutrophil function by affecting the SOCS1 protein.