Objective: Increased serum levels of inflammatory biomarkers such as CRP and IL-6 have been reported in periodontitis patients. We hypothesize that infection with the periodontal pathogen Porphyromonas gingivalis (P. gingivalis) induces cytokinemia. The objective of this study was to perform a proteomic survey of cytokinemic responses to evaluate the diversity, magnitude and kinetics of serum inflammatory biomarkers in mice infected with P. gingivalis. Methods: Eleven heterozygous ApoE+/- mice were used (six males and 5 females). A murine subcutaneous chamber model of P. gingivalis infection was used with intra-chamber injection of heat-killed P. gingivalis A7436 (10^9 CFU) at week 10 and live P. gingivalis A7436 (10^8 CFU) at week 13. Serum samples were collected at days -2, +5, and +12 before and after live P. gingivalis challenge. Serum levels of a panel of 18 cytokines were measured using the murine Bio-Plex cytokine assays on a Luminex-100. Mean±SD for each cytokine were calculated for each time point. Values at day 5 and day 12 were compared individually to values at day -2 by paired t test. Results: The pattern of biomarker response show a significant activation of the innate immune response (P<0.05) with a 2-4-fold increase in the levels of IL-1, TNF, and IL-6. Cytokines characteristic of a Th2 response were also significantly increased 2-fold (IL-4, IL-5, and IL-10). Interestingly, cytokines characteristic of a Th1 response remained unchanged (IL-2 and IFN-γ), as did IL-12. Remarkably, G-CSF and KC (murine equivalent to IL-8) were significantly increased 20-fold, indicating a major neutrophil recruitment and activation response. Together with unchanged levels of GM-CSF, MIP-1α, and RANTES, the pattern and kinetics of the response are consistent with a serum acute phase cytokine response. Conclusion: P. gingivalis infection in the murine subcutaneous chamber model induced a strong innate immune and Th2-dominated serum inflammatory response. Work supported by NIDCR-DE14459 and RR-00046.