Biofilm formation is a key factor in periodontal disease progression, and bacterial interactions mediated by secreted extracellular products, including proteins, metabolites, signaling molecules, and -membrane vesicles (MVs), can influence biofilm development and community structure. However, the effects of extracellular products derived from mono- and multispecies cultures on Porphyromonas *gingivalis* (Pg) biofilm formation remain poorly understood. We hypothesized that extracellular products present in mono- and multispecies culture supernatants would alter Pg biofilm formation, providing insight into how bacterial interactions may contribute to periodontal disease. Culture supernatants were collected from mono-, dual-, and tri-species cultures of Pg, Streptococcus *gordonii* (Sg), and Bacteroides *thetaiotaomicron* (Bt). Pg was exposed to each supernatant condition (Pg, Sg, Bt, PgBt, PgSg, SgBt, and PgSgBt), and biofilm formation was quantified using crystal violet staining and absorbance measurements. Additional experiments included OMV isolation and characterization to further investigate extracellular products produced during polymicrobial growth. Exposure to bacterial supernatants resulted in differences in Pg biofilm formation relative to Pg controls. Supernatants from the Pg+Bt, Pg+Sg, and Sg+Bt cultures showed greater Pg biofilm biomass than the Pg control, suggesting that extracellular products generated through polymicrobial interactions can influence Pg biofilm development. These findings indicate that secreted factors from neighboring bacterial species may contribute to changes in Pg behavior associated with biofilm establishment. Future studies will repeat the biofilm assays to validate these preliminary findings and use size fractionation of culture supernatants, along with further MV characterization, to identify the extracellular components responsible for altered Pg biofilm formation. Testing different molecular weight fractions will help determine whether biofilm-promoting activity is associated with small molecules, proteins, or larger extracellular structures such as membrane vesicles, providing insight into mechanisms that may contribute to periodontal disease progression.