Porphyromonas gingivalis (Pg) is a major contributor to periodontal disease. Outer membrane vesicles (OMVs) are derived from the bacterial outer membrane and serve as an export system for a variety of biomolecules. Pg's OMVs are predicted to play a significant role in gum disease pathogenesis by inducing an inflammatory response in the gums via the TLR4 receptor and regulating biofilm dispersal. The mechanism responsible for cargo selection and OMV production in Pg is unknown. Our lab discovered that lipid A structure influences OMV production, more specifically, we found that lipid A C4' phosphatase deletion mutant, *ΔlpxF*, hypovesiculates and forms an abnormally dense biofilm. This mutant is also sensitive to Polymyxin B (PMB), an antimicrobial peptide that binds to phosphate groups on lipid A. We hypothesize that other mutants previously observed to produce fewer OMVs than WT will display biofilm phenotypes, PMB sensitivity, and TLR4 stimulation similar to *ΔlpxF*, even if the deletion affects another gene. Reduced OMV production by mutant *ΔwbpB* was confirmed with transmission electron microscopy. I then tested PMB sensitivity and performed biofilm assays to determine relative biofilm density for each strain. TLR4 stimulation was measured via IL6 secretion with an ELISA. Results showed that consistent patterns in LPS structural modifications were not always associated with impaired vesiculation. These results are not indicative of convergence in the pathways driving OMV production. This information can be applied to efforts to develop inhibitors of OMV production by Pg, potentially reducing the burden of periodontal disease for millions.