

SHANIA DAVIS & JOSEPH HORZEMPA, Department of Biological Sciences, West Liberty University, West Liberty, WV USA. The *Pseudomonas aeruginosa* 1244 pilin glycan enhances twitching motility and renders bacteria more susceptible to human beta defensin 2.

*Pseudomonas aeruginosa* is an ESKAPE (highly drug resistant) pathogen that is commonly acquired during hospital stays. This bacterium produces type IV pili which are adhesins and motor appendages that mediate a surface motility referred to as “twitching.” These pili are polymers of protein subunits referred to as pilin. The pilin subunit of *P. aeruginosa* 1244 is glycosylated. Previous studies have shown that the pilin glycan affects the efficiency of twitching motility under certain conditions, fiber surface polarity, and overall bacterial surface polarity. We also confirmed and extended these previous findings by showing pilin glycosylation substantially enhances twitching motility on positively charged surfaces (poly L-lysine coated plastic). Because modulation of the bacterial surface charge has been shown to mediate defensin resistance in other organisms, we tested whether pilin glycosylation affected sensitivity to human beta defensin 2 (HBD-2). Interestingly, the isogenic mutant strain lacking the pilin glycan (1244G7) showed increased resistance to HBD-2 compared to wild type bacteria and those completely lacking a type IV pilus (1244.47). This increased resistance to HBD-2 could explain data from a recent study in which various *P. aeruginosa* clinical strains were isolated that had pilin glycosylation defects. We therefore sought to determine if pilin glycosylation modulates the interaction between pili and HBD-2. Here, an immunoprecipitation assay was carried out using HBD-2 as bait and either purified glycosylated pili or non-glycosylated pili as bait. These studies are ongoing and could provide insight into an important host-pathogen interaction. [This work was supported by the National Institutes of Health, National Heart Lung and Blood Institute (1R15HL147135) and an Institutional Development Award (IDeA) from the National Institute of General Medical Sciences (P20GM103434) which funds the WV-INBRE program].