Interview with Marianna Patrauchan

I sat down with Marianna Patrauchan an associate professor at Oklahoma State University in the microbiology division who was kind enough to grant me an interview and talk to me about the research that her and her colleges have been performing on *Pseudomonas aeruginosa*. I asked professor Patrauchan what this organism was and why it was important. She told me that it is a multidrug resistant pathogen, and that it causes severe and chronic infections in patients with cystic fibrosis, as well as patients with compromised immune systems, burn wounds, intensive care patients and patients with indwelling medical devices, catheter and shunts. When asked what her research had found about this bacterium she told me that “higher calcium (Ca2+) levels make this bacterium 5 – 10 times more resistant to antibiotics like tobramycin and this made it much harder to treat patients with this type of infection”.

I asked her to explain how calcium (Ca2+) brought about such a significant increase in *P. aeruginosa*. She told me that it was due to the calcium effecting the amount of efflux pumps. When asked how this happened professor Patrauchan explained that *Pseudomonas aeruginosa* has pumps spanning its dual membrane called RND efflux pumps. They are pumps that span the dual membrane layer of the bacteria. These pumps provide antibiotic resistance to this organism by pumping out substances that are identified as toxic to it, and this includes the antibiotics that makes it into the bacteria. When *P. aeruginosa* is exposed to high levels of calcium (Ca2+) more RND efflux pumps are made and this causes the bacteria to pump the antibiotics out at faster rates then they previously were capable. This is what causes the resistance to the antibiotics to be amplified so significantly in this bacterium.

I then asked the professor; how did you determine that it was the efflux pumps reacting to the calcium and not some other factor? She told me that in her team’s research they had taken samples of *P. aeruginosa* that were normal and samples that had mutations in the genes that coded for the RND efflux pumps that inhibited their functionality. They then exposed both to high levels of Ca2+ and then exposed them to the antibiotic tobramycin. Their results showed that the *P. aeruginosa* with the mutated RND efflux pumps did not show the same signs of increased resistance as their non-mutated counterparts.

I asked her what the ultimate goal for this research is and professor Patrauchan stated “it is to understand how this bacterium works, so that we can help those suffering from its effects”. If her research provides insights into how this bacterium works then it could lead to new ways to help patients who suffer from the chronic infections brought on by this bacterium.

Khanam, Sharmily, et al. “Calcium Induces Tobramycin Resistance in Pseudomonas Aeruginosa by Regulating RND Efflux Pumps.” Cell Calcium, vol. 61, 2017, pp. 32–43., doi:10.1016/j.ceca.2016.11.004.