Breakthroughs In The Cabeen Lab, And Future Applications That Could Come From It

Recently, I had the pleasure of speaking with Dr. Matthew Cabeen, an Assistant Professor at Oklahoma State University in the Department of Microbiology and Molecular Genetics. He researches bacteria cell stress responses, like how a person might respond to heat or spicy food, and biofilm formation, a protective layer that forms around a bacterium. Dr. Cabeen is a credible member of the science community; he has been doing research for 21 years and was a postdoctoral fellow at Harvard from 2010-2017. Dr. Cabeen has been with OSU for six years and has worked with a bacterial species called *Pseudomonas aeruginosa*, a bacterium that can and does infect humans. This bacterium will compete with other strains for resources just like larger animals do in the wilderness to reproduce. The bacteria do this by generating pyocins when their DNA is damaged. Pyocins are protein complexes that function like a spring-loaded spike that will stab through the wall of other bacterial cells, which can cause them to die. Antibiotic resistance is the correlation between this research and treating people infected by Pseudomonas and other bacteria. Antibiotic resistance is when a bacterial infection is less susceptible to treatment after continual antibiotic exposure. It is a growing problem within the scientific and medical community that requires much time and effort to combat. The breakthrough Cabeen's lab has made is that the removal of a specific gene called *xerC* would cause an increase in pyocin production independent of the primary production while simultaneously making cells hypersensitive to fluoroquinolone antibiotics. This antibiotic family includes drug brands such as Cipro, Levaquin, and Avelox. The generic names are ciprofloxacin, levofloxacin, and moxifloxacin, respectively.
Along with the fluoroquinolones, *Pseudomonas* is resistant to beta-lactams and aminoglycosides, and considering that there are roughly six groups of antibiotics that's quite a high resistance rate. Due to the discoveries in Dr. Cabeen's lab, he theorizes that a plan to circumvent antibiotic resistance for treating *Pseudomonas* infections that aren't as affected by the fluroquinolones would be to develop a drug that could mimic the *xerC* deletion. You could then weaponize a *Pseudomonas* bacterium against a resistant *Pseudomonas,* which would destroy the resistant strain through the pyocin production and competition and then conversely be destroyed by the fluroquinolone treatment. Dr. Cabeen, at this time, has published two papers on the specific topic of *xerC,* which go much more in-depth into the mechanics of how the pyocins and *xerC* function; they will be linked below for your reading*. I want to thank Dr. Cabeen for his time and work in the field.

*The information in this paper is taken directly from an interview with Dr. Cabeen, leading to the lack of citations.

References

1. Interview with Dr. Cabeen conducted on 4/6/2023
2. Adam S. Bronson, Nina S. Baggett, Matthew T. Cabeen. DNA Damage-Inducible Pyocin Expression Is Independent of RecA in *xerC*-Deleted *Pseudomonas aeruginosa* https://doi.org/10.1128/spectrum.01167-22 (2022)
3. Nina S. Baggett, Adam S. Bronson, Matthew T. Cabeen. SOS-Independent Pyocin Production in *P. aeruginosa* Is Induced by XerC Recombinase Deficiency https://doi.org/10.1128/mBio.02893-21 (2021)