Dr. Matthew Cabeen is an associate professor and researcher in Oklahoma State University’s Department of Microbiology and Molecular Genetics. I interviewed him about his work as a researcher. He became interested in microbes while working in microbiology labs over the summers as an undergraduate. This work led to an interest in ribonucleic acid, or RNA. In graduate school, he began doing work with riboswitches related to RNA. Then, he met a new professor, Christine Jacobs-Wagner, who was studying bacterial development and the proteins responsible for a curved shape in bacteria. Here, Dr. Cabeen began studying the two bacteria on which he now focuses at OSU, *Bacillus subtilus* and *Psuedomonas aeruginosa.*

 Dr. Cabeen’s most recent publication on *P. aeruginosa* focused on the bacteria’s ability to produce biofilms. He described biofilms as ‘communities of cells held together by gluey secretions.’ These secretions are made up of sugars, deoxyribonucleic acid (DNA), and proteins (Cabeen, *et al.,* 2016). Biofilms also help bacteria resist antibiotic treatment. Since *P. aeruginosa* is a human pathogen, antibiotic resistance makes treating an infection of the bacteria harder. Dr. Cabeen’s research looked into a new approach to analyze biofilm formation. This technique had already been useful in a previous study focusing on the other organism Dr. Cabeen studies, *B. subtilus* (McLoon, *et al.,* 2011). By looking at the physical appearance of the bacteria, the colony morphology, the research team was able to find biofilm-related genes that were not focused on in previous research (Cabeen, *et al.,* 2016). They began by looking at colony morphology on agar plates. Using a mutant strain known to cause wrinkled colonies and increased biofilm production, they were able to look further into genes that play a role in the biofilm production (Cabeen, *et al.,* 2016). Three genes were found to be active in forming a biofilm that were previously thought to be uninvolved: *PA14\_16550, PA14\_69700,* and *ptsP* (Cabeen, *et al.,* 2016). Further experiments were carried out to confirm that these genes had an effect on biofilm formation or colony morphology or both. Mutations in genes *16550* and *ptsP* caused decreased biofilm formation in wild-type, or nonmutated, bacteria, and deleting the genes in hyper producers of biofilm also caused decreased production (Cabeen, *et al.,* 2016). Mutations and deletions to gene *69700* enhanced biofilm production (Cabeen, *et al.,* 2016). The study provided new insight into the mechanisms by which *P. aeruginosa* creates and maintains biofilms.

 When asked about the human implications for his research, Dr. Cabeen said, “The more we understand about how bacteria work the better equipped we are to utilize them in both ways.” Since bacteria can be both helpful and harmful, we need to know understand properties of both to benefit people. In the case of *P. aeruginosa,* its harmful effects to humans need to be understood to prevent negative health impacts. *As* for his ultimate goal in researching *P. aeruginosa,* Dr. Cabeen explained that a better understanding of biofilm formation gives healthcare providers insight in how to keep them from occurring. Since they are hard to stop once they start, the ultimate goal for biofilms is prevention.