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Saryu Sanghani
Washington University in St. Louis

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Mentor: Barbara Kunkel

Pseudomonas syringae is a bacterial pathogen that infects *Arabidopsis thaliana,* tomato, and many other plants. We know little about how *P. syringae* strain DC3000 survives and grows within its hosts. During pathogenesis, *P. syringae* populates the apoplast of plant tissue, where it must tolerate the stress from defense mechanisms and also import and metabolize the available nutrients to survive and grow. Auxin is a plant hormone that has been shown to play a role in increasing disease symptoms caused by *P. syringae.* Our research seeks to clarify the metabolic pathways that *P. syringae* uses to grow in its hosts and to see whether auxin regulates these pathways.

In many contexts, auxin is a growth and development hormone that contributes to disease by increasing plant susceptibility to infection or by making nutrients available for pathogen growth. Auxin, however, inhibits growth of *P. syringae* on various carbon sources in culture and inhibits expression of specific *P. syringae* virulence genes. To decipher these seemingly paradoxical results, we want to elucidate the metabolic pathways and carbon sources used by *P. syringae.* We hypothesize that *P. syringae* uses auxin as a switch or a signaling molecule that turns off early virulence genes and turns on late virulence genes. These latter genes could be involved in metabolic shifts since different organic compounds could be made available at later stages of infection.

Having fed *P. syringae* various different carbon sources in culture, we saw that the levels of growth were very similar for all carbon sources (minus the negative control) except fructose—which cannot be metabolized as well due to a lacking enzyme. Regardless of carbon source, we did see the inhibitory auxin effect in each media. There was variation in the magnitude of inhibition, but not enough to make significant conclusions.