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Succinoglycan Production and Œ≤-lactamases Confers Resistance to External Stresses in Agrobacterium tumefaciens

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Peptidoglycan (PG) is a conserved feature that initiates an immune response in almost all plants and animals. Slowing PG synthesis during host invasion may be an effective strategy for bacteria to avoid detection by the host immune system. Remarkably, loss of an essential PG synthase, PBP1a, not only results in the loss of PG synthesis but also activates a transcriptional program that transitions *Agrobacterium tumefaciens* from a free-living motile bacterium, into a host-invading state. During this transition, production and secretion of the exopolysaccharide, succinoglycan is upregulated. To explore the link between PG synthesis and succinoglycan production, I dissected known components of the succinoglycan regulatory and biosynthesis pathways and tested the hypothesis that succinoglycan may be protective against environmental stressors. Here, we find that cells without succinoglycan are sensitive to acid stress, membrane disruptors, and cell-wall targeting antibiotics. These findings suggest a direct role for succinoglycan in providing protection to external stressors. Additionally, I show evidence that *A. tumefaciens* utilizes beta-lactamases as an additional layer of protection from beta-lactam antibiotics. Through the use of both genetic and chemical inhibition, I propose here a comprehensive characterization of the two β-lactamases encoded in the genome of *A. tumefaciens*. Together, these findings will lead to a greater understanding of bacterial antibiotic resistance, informing on a major global health crisis.