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Poster #33: Gasdermin-mediated phage defense as a model for intercellular immune signaling in Streptomyces

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Gasdermins are pore-forming proteins that mediate programmed cell death in both eukaryotes and bacteria. In animals, pathogen-induced activation of gasdermins not only eliminates infected cells but also promotes the release of inflammatory signaling molecules that coordinate immune responses. In bacteria, gasdermins have recently been identified as anti-phage defense systems that abort infection through membrane pore formation, yet whether they also enable intercellular signaling remains unknown. We identified functional gasdermin homologs in *Streptomyces* species that provide defense against bacteriophage infection. As filamentous, multicellular bacteria that rely on extracellular signaling to coordinate development and stress responses, *Streptomyces* provide a unique model to investigate whether gasdermin activation is coupled to intercellular signaling during phage infection. We hypothesize that gasdermin pore formation facilitates the release of extracellular signaling molecules from infected filaments, allowing uninfected regions of the colony to detect phage attack and mount coordinated antiviral responses before infection spreads. Our work aims to determine whether bacterial gasdermins function not only as autonomous cell-death effectors but also as mediators of colony-wide immune coordination. This will provide insight into the evolution of innate immunity and reveal whether coordinated intercellular communication is a conserved feature of gasdermin-mediated defense across domains of life.

Reference #1

Reference #2

Reference #3

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