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Bacterial multicellular behaviour in antiviral defense

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Phage defense is commonly studied at the level of individual cells and intracellular immune systems. However, bacterial populations often display multicellular organization, developmental differentiation, and extensive chemical interactions that can influence the outcome of phage infection.

Using *Streptomyces* as a model, we examine antiviral mechanisms that operate at the population level. Developmental transitions can generate transiently phage-resistant cell states and restrict phage propagation within structured populations (1,2). Secreted secondary metabolites provide an additional layer of defense. In the first funding period, we have demonstrated that aminoglycoside antibiotics inhibit early stages of phage infection and that modifications through resistance mechanisms retain the antiviral properties of the molecule (2,3). DNA-intercalating antiphage molecules further illustrate how chemical defense can alter infection trajectories. For daunorubicin-sensitive phages, inhibition after first-step genome transfer of T5-like phages permits continued pre-early gene expression but prevents productive infection, resulting in abortive infection through "mutual destruction"of both phage and host (5). Related phenotypes occur across taxonomically distinct phages and chemically different DNA-intercalating compounds. Moreover, the DNA-intercalating anthracycline daunorubicin can synergize with downstream nucleic acid-targeting defense systems, demonstrating that chemical and canonical immunity can act cooperatively in a context-dependent manner.

Together, these findings show how developmental state, spatial organization, and extracellular metabolites contribute to multilayered antiviral defense in bacteria.

Reference #1

Luthe T, Kever L, Hänsch S, et al. Streptomyces development is involved in the efficient containment of vi

Reference #2

Kever L, et al. Aminoglycoside antibiotics inhibit phage infection by blocking an early step of the infect

Reference #3

3) Ernst L, Gätgens C, Rackow B, et al. DNA-intercalating antiphage molecules trigger abortive infection

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