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Poster #13: Interbacterial competition triggers stress-induced CRISPR immunity against bacteriophages

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Bacteria–phage interactions are commonly studied in monoculture, yet bacteria in nature experience interspecific competition that may alter their antiviral defences. We investigated whether competition directly regulates CRISPR-Cas immunity in Pseudomonas aeruginosa PA14. Mathematical modelling predicted that competition-induced CRISPR regulation would cause rapid enrichment of CRISPR-immune cells, distinct from the delayed effects of ecological selection. We tested this prediction using transcriptional reporters and short-term spacer-acquisition assays following infection with phage DMS3vir in monoculture or co-culture with Acinetobacter baumannii, Burkholderia cenocepacia, or Staphylococcus aureus. Co-culture with A. baumannii, but not the other competitors, increased spacer acquisition approximately tenfold and strongly elevated CRISPR-Cas expression within three hours. CRISPR-Cas expression correlated positively with both short-term spacer acquisition and longer-term evolution of CRISPR-mediated phage resistance. Quorum sensing did not explain this response: interspecific competition suppressed Las signalling, and disruption of Las signalling further increased CRISPR-Cas expression during competition with A. baumannii. Instead, competition with A. baumannii induced a strong cellular SOS response, and experimentally imposed DNA damage with mitomycin C caused dose-dependent increases in CRISPR-Cas expression in the absence of competitors. Together, our results identify competition-induced stress as a physiological cue that primes bacterial adaptive immunity before selection acts. These findings connect interbacterial antagonism to phage defence and suggest that microbial community context can accelerate the emergence of CRISPR-mediated resistance during phage infection and therapy.

Reference #1

Reference #2

Reference #3

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