Modulation of predatory phenotypes in the bacterial predator Bdellovibrio bacteriovorus

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Résumé

A key question in community ecology is how food webs, which are the basis of sustainable life, are maintained. A large body of animal studies shows the importance of predator–prey interactions for trophic web stability and species coexistence. Yet, the experimental flexibility and the diversity of microbial systems can provide unique perspectives on predatory interactions that in turn, can affect large ecosystems. *Bdellovibrio bacteriovorus*, is an obligate bacterial predator. It is thus unable to replicate without prey and consequently cannot form biofilms, which are surface-associated cells embedded in secreted polymers. However, spontaneous host-independent (H-I) variants grow axenically and can form robust biofilms. We screened 350 H-I mutants and found that single mutations in flagellar stator (motor) genes were sufficient to generate H-I strains, deficient in motility and able to adhere to surfaces but unable to develop biofilms. Genes associated with flagellar, prey-invasion, and phenotype-shift secondary signal cyclic-di-GMP (CdG) functions were deregulated, and CdG cellular concentration changed significantly. Genetic complementation resulted in full reversion to the wildtype, host-dependent (H-D) phenotype, but functional complementation by paralogues was not observed. Moreover, specific mutations in the background of invasion pole-associated pilus mutants were necessary for biofilm formation and the introduction of a mutation in stator genes strongly reduced biofilm development. All H-I variants grew similarly without prey, showed strain-specific reduction in predatory ability in prey suspensions, but similar high efficiency in prey biofilms. Targeted sequencing suggested additional routes to host-independence. We further reveal that the CdG network includes a massively expressed untranslated RNA (*merRNA*) that acts as a standalone CdG riboswitch that controls motility. It also strongly affects general gene expression and is itself differentially expressed in an H-I background, compared to the H-D strain. Thus, stator and invasion pole-dependent signaling control the H-D and the H-I biofilm-forming phenotypes, through CdG signaling with single mutations overriding prey requirements, enabling shifts from obligate to facultative predation, with potential consequences on community dynamics. Our findings on the variety of changes leading to facultative predation also challenge the concept of *Bdellovibrio* and like organisms being obligate predators.

Mots-Clés: Predation, Bdellovibrio, biofilm, phenotype shift

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