INFLUENCE OF BACTERIAL DEFENSE SYSTEM COMPONENTS ON CELL VIABILITY

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The ongoing arms race between bacteria and bacteriophages has led to the development of various defense strategies in bacteria. Prokaryotes have evolved mechanisms to terminate viral infection at multiple stages of the phage life cycle. Modification or masking of cell surface receptors prevents phage recognition, while superinfection exclusion systems (Sie) block the injection of bacteriophage nucleic acids if recognition occurs. Restriction-modification and the adaptive CRISPR-Cas systems target foreign nucleic acids that have entered the cell, and secondary metabolites can inhibit viral replication or transcription. Finally, abortive infection (Abi) systems halt bacteriophage propagation by inducing cell dormancy or programmed cell death [1].

It is now known that genes encoding these antiviral defense systems tend to cluster in regions called defense islands within the bacterial genome [2]. This observation has facilitated the discovery of previously unknown defense systems around already characterized defense genes. A four-gene operon, QatABCD, is one of the novel defense systems identified this way [3]. However, its precise mechanism of action remains unclear.

In this study, we investigate the effects of QatABCD components on *Escherichia coli* cell viability to determine a potential effector of the system. The growth of bacterial cells carrying plasmids with different deletion variants of the operon was monitored by measuring optical density in a plate reader under uninfected conditions. Additionally, phage infection dynamics were evaluated at varied multiplicities of infection (MOIs) using cells expressing the full QatABCD system. We find that specific QatABCD constructs differentially affect cell viability, pointing to candidate effector components of the system.

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