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**Excitable toxin-antitoxin modules coordinated through intracellular bottlenecks**[1]

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Chronic infections and pathogenic biofilms present a serious threat to the health of humans by decreasing life expectancy and quality. The resilience of these microbial communities has been attributed to the spontaneous formation of persister cells, which constitute a small fraction of the population capable of surviving a wide range of environmental stressors. Gating of bacterial persistence has recently been linked to toxin-antitoxin (TA) modules, which are operons with an evolutionarily conserved motif that includes a toxin that halts cell growth and a corresponding antitoxin that neutralizes the toxin. While many such modules have been identified and studied in a wide range of organisms, little consideration of the interactions between multiple modules within a single host has been made. Moreover, the multitude of different antitoxin species are degraded by a relatively small number of proteolytic pathways, strongly suggesting competition between antitoxins for degradation machinery, i.e. queueing coupling. Here we present a theoretical understanding of the dynamics of multiple TA modules that are coupled through either proteolytic queueing, a toxic effect on cell growth rate, or both. We conclude that indirect queueing coordination between multiple TA modules may be central to controlling bacterial persistence.

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