Evolution of Bacterial Suicide

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We propose a theoretical model that predicts that bacterial autolysis is evolutionarily advantageous to an individual and would fixate in physically structured environments for stationary phase colonies. We perform spatially resolved agent-based simulations of the model, which predict that lower mixing in the environment results in fixation of a higher autolysis rate from a single mutated cell, regardless of the colony's genetic diversity. We argue that quorum sensing will fixate as well, even if initially rare, if it is coupled to controlling the autolysis rate.

***Keywords* — Evolutionary dynamics, bacterial autolysis**

I. Introduction

In multicellular organisms, controlled cell death could be beneficial to the organism as a whole; for example, it could prevent the spread of a pathogen. However, while active, controlled cellular suicide (autolysis) in bacteria is commonly observed [1-3], it has been hard to argue that autolysis can be beneficial to an individual who commits it. The evolutionary stability of bacterial death requires genotypic assortment and phenotypic cooperation, therefore implying a structured environment [4]. Further, the cost of autolysis needs to be offset by the presence of a public good, such as the release of enzymes, toxins, or for protection of the colony from pathogenic invasions [4]. Since simultaneous evolution of autolysis and public goods is highly unlikely, it begs the question which feature evolved first. We propose that the evolution and fixation of autolysis can happen independently of any explicit public good.

II. Model

We model a resource-limited world consisting of N single-occupancy sites. The cells that inhabit these sites undergo a modified Fisher-Wright dynamics. Division is modeled as a random process that can only occur if there is an unoccupied site in the immediate neighborhood of the dividing cell. Cells age chronologically, and hence their fertility decreases with time even if cells don't divide. Autolysis is given by an unregulated, memoryless random process. The population is genetically heterogeneous in just one trait: characteristic life span or the autolysis rate. During division, this trait is multiplicatively modified, modeling mutation. Finally, cells move throughout the environment; high motility rates correspond to a well-mixed world, while low motility represents a highly structured one.

III. Results

Our simulations predict that lower mixing in the environment results in fixation of a higher autolysis rate from a single mutated cell, regardless of the colony's initial genetic diversity. Further, cells that exhibit the optimal autolysis rate for a specific environment fixate within a population of suboptimal cells. Lastly, our simulations demonstrate that cells that are capable of detecting the diffusivity of their environment and can adjust their autolysis rates accordingly on physiological scales, have a significant fitness advantage. Thus quorum sensing, interpreted as diffusion sensing [5] can be advantageous and will fixate even if rare if it is coupled to controlling the autolysis rate [6].

Experimental observations support our predictions. First, in *B. subtilis* biofilm, mutants that modify matrix composition and increase mixing are less prone to exhibit autolysis [2]. Second, quorum sensing *S. pneumoniae* mutants that are incapable of sensing concentration of the messenger molecule similarly show reduced propensity for autolysis [3].

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