Stripe formation in bacterial systems with density-suppressed motility

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***Short Abstract* — Engineered E. coli bacteria in which motility is coupled to cell density generate periodic stripes of high and low densities on agar plates. We theoretically study the origin and mechanism of this pattern formation process in a one-dimensional kinetic model that includes growth and density dependent motility of the cells. The calculated front profile of the growing population reveals an intriguing aggregation mechanism. We derive an analytical criterion for the onset of pattern formation and calculate the phase diagram for the transition from the stripe to the no-stripe phase.**

***Keywords* — Pattern formation, cell density, molitlity.**

Pattern formation is ubiquitously seen in nature. Already colonies of bacteria or simple eukaryotic cells show cooperative behavior that generates complex shapes and patterns. Examples include the emergence of aggregation patterns of *Dictyostelium amoebae* [1], convection patterns in suspensions of swimming microorganisms [2] and rippling patterns in fruiting bodies of *Myxococcus xanthus* [3]. One of the best studied systems is that of *E. coli* colonies grown on agar plates that form complex patterns of spots, stripes, and rings [4, 5]. These findings have triggered extensive mathematical modeling typically performed in the framework of Keller-Segal models [6–8]. However, despite considerable efforts the detailed physical mechanisms of organization still remain unclear (for an overview see, e.g., [9]).

Synthetic biology provides novel experimental approaches to probe the mechanisms of pattern formation. In a recent study we have demonstrated that engineered *E. coli* bacteria with a cell-density dependent motility form periodic spatial patterns [10]. The density dependent motility was introduced into *E. coli* by adopting the quorum-sensing module of a different bacterium (*Vibrio fischeri*). This system synthesizes and excretes a small molecular acyl-homoserine lactone (AHL). At high extracellular levels (caused, e.g., by high cellular densities) AHL accumulates intracellularly leading (via engineered regulation of the *cheZ* gene) to non-motile cells.

On plates these bacteria form highly regular and stable stripe patterns consisting of periodically alternating regions of high and low cellular densities. A thorough characterization of these spatial patterns gave rise to the following key experimental observations [10]: (i) Regulation of cell motility by AHL is essential for pattern formation. A mutant in which AHL did not regulate *cheZ* did not show any pattern formation. (ii) Cells in the low density stripes are motile, while cells in the high density stripes are non-motile. (iii) Bacteria form pattern in one- and two-dimensional geometries. (iv) Pattern formation requires a sufficiently large initial density perturbation. (v) The geometry of the pattern depends on the cellular motility. There is a transition with decreasing cellular motility from a phase with periodic patterns to a phase with finite number of stripes.

To understand the experimental system, a quantitative model with three key ingredients is represented. Computer simulations directly help the experiment to study the underling mechanism of the experiments. Here, a theoretical analysis of a simplified model gives an insight to the origin and mechanism of this pattern formation process. The traveling wave front profile is calculated. With the analytical criterion for the onset of pattern formation, the phase diagram for the transition from stripe to the no-stripe phase is determined [11].

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