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Simple models do not explain early dynamics of *H. influenzae* bacteremia XINXIAN SHAO, Department of Physics, Emory University, BRUCE LEVIN, Department of Biology, Emory University, ILYA NEMENMAN, Department of Physics, Emory University — There is an abundance of largely qualitative information about the physiological and molecular mechanisms of bacterial pathogenesis. However, little is known about population dynamic processes by which bacteria colonize hosts and invade cells and tissues and thereby cause disease. Classic experiment of Moxon and Murphy[1] observed that, when inoculated intranasally with a mixture of equally virulent strains of Haemophilus influenzae type b(Hib), neonatal rats develop a bacteremic infection that often is dominated by only one random competing strain. A common qualitative explanation for this phenomenon is that the bacteria must switch stochastically into a rapidly growing phenotype to start the full-fledged invasion. Then the first bacterium to switch activates the host immune response, which in turn shuts the door in front of the second strain. We implemented this model computationally and analytically, and we conclude that this model cannot explain the data, specifically, the observed dependence of the rate of infections on the inoculum size. New experiments are needed to identify mechanisms underlying the dependence.

[1]Moxon E R,Murphy P A (1978). Haemophilus influenzae bacteremia and meningitis resulting from survival of a single organism. PNAS, 75(3), 1534-1536.

Xinxian Shao
Department of Physics, Emory University

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