**Quorum sensing in bacteria.**

Miller MB, Bassler BL.

**Abstract**

Quorum sensing is the regulation of gene expression in response to fluctuations in cell-population density. Quorum sensing bacteria produce and release chemical signal molecules called autoinducers that increase in concentration as a function of cell density. The detection of a minimal threshold stimulatory concentration of an autoinducer leads to an alteration in gene expression. Gram-positive and Gram-negative bacteria use quorum sensing communication circuits to regulate a diverse array of physiological activities. These processes include symbiosis, virulence, competence, conjugation, antibiotic production, motility, sporulation, and biofilm formation. In general, Gram-negative bacteria use acylated homoserine lactones as autoinducers, and Gram-positive bacteria use processed oligo-peptides to communicate. Recent advances in the field indicate that cell-cell communication via autoinducers occurs both within and between bacterial species. Furthermore, there is mounting data suggesting that bacterial autoinducers elicit specific responses from host organisms. Although the nature of the chemical signals, the signal relay mechanisms, and the target genes controlled by bacterial quorum sensing systems differ, in every case the ability to communicate with one another allows bacteria to coordinate the gene expression, and therefore the behavior, of the entire community. Presumably, this process bestows upon bacteria some of the qualities of higher organisms. The evolution of quorum sensing systems in bacteria could, therefore, have been one of the early steps in the development of multicellularity.

**The Evolution of Quorum Sensing in Bacterial Biofilms**

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## Abstract

Bacteria have fascinating and diverse social lives. They display coordinated group behaviors regulated by quorum-sensing systems that detect the density of other bacteria around them. A key example of such group behavior is biofilm formation, in which communities of cells attach to a surface and envelope themselves in secreted polymers. Curiously, after reaching high cell density, some bacterial species activate polymer secretion, whereas others terminate polymer secretion. Here, we investigate this striking variation in the first evolutionary model of quorum sensing in biofilms. We use detailed individual-based simulations to investigate evolutionary competitions between strains that differ in their polymer production and quorum-sensing phenotypes. The benefit of activating polymer secretion at high cell density is relatively straightforward: secretion starts upon biofilm formation, allowing strains to push their lineages into nutrient-rich areas and suffocate neighboring cells. But why use quorum sensing to terminate polymer secretion at high cell density? We find that deactivating polymer production in biofilms can yield an advantage by redirecting resources into growth, but that this advantage occurs only in a limited time window. We predict, therefore, that down-regulation of polymer secretion at high cell density will evolve when it can coincide with dispersal events, but it will be disfavored in long-lived (chronic) biofilms with sustained competition among strains. Our model suggests that the observed variation in quorum-sensing behavior can be linked to the differing requirements of bacteria in chronic versus acute biofilm infections. This is well illustrated by the case of Vibrio cholerae, which competes within biofilms by polymer secretion, terminates polymer secretion at high cell density, and induces an acute disease course that ends with mass dispersal from the host. More generally, this work shows that the balance of competition within and among biofilms can be pivotal in the evolution of quorum sensing.