

This Week in Evolution

Each week I discuss one of the hundreds of papers with new data on evolution, published in the past month.

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Cooperation and cheating in microbes: quorum sensing and persisters

Two papers on cooperation this week. If you were trying to help someone, but end up causing problems for them, were you being cooperative? I have no idea, so I like to study cooperation in microbes. Microbes don't have brains, so "intent" isn't a factor. And the only definition of "benefit" that makes sense is an increase in Darwinian fitness or reproductive success, which is often easy to measure in microbes; just count them.

I like these [definitions](#):

Cooperation: a behaviour which provides a benefit to another individual (recipient), and which is selected for because of its beneficial effect on the recipient. [Exhaling CO₂ isn't cooperation; it evolved as a side-effect of breathing oxygen, not to benefit plants.]

Cheaters: individuals who do not cooperate (or cooperate less than their fair share), but are *potentially* able to gain the benefit of others cooperating. ["Equal share" might be less ambiguous.]

The evolution of cooperation and cheating in microbes is a hot topic these days, for various reasons. Species without brains simplify the analysis of cooperation and allow interesting comparisons with humans and other animals. (Do humans mostly cooperate with relatives, as bacteria and some animals do, or do our brains lead to cooperation with nonrelatives?) Some kinds of cooperation among microbes may shed light on the origin of multicellular life. Cooperation among microbes (all [investing in nitrogen fixation](#), for example) is critical to the benefits they provide to plants, while other kinds of cooperation may make pathogenic microbes more deadly.

Cooperation can be pointless if the number of cooperators is below some threshold. That's why listener-supported radio stations in small towns are rare. I have suggested a solution to this problem [elsewhere](#), but my solution only works for reasonably intelligent life-forms.

How do bacteria solve the threshold problem? For example, a single bacterial cell can't provide its plant host with enough nitrogen to make much difference; only billions of bacteria, working together, can do that. Similarly, only a large number of harmful bacteria, working together, can produce enough of a "virulence factor" to overcome host defenses.

Bacteria often exchange "quorum-sensing" chemical signals that control activities requiring a large number of cells. Each cell produces a little homoserine lactone or whatever. If there are enough cells in a small enough space, the homoserine lactone concentration gets high enough to trigger cooperative activities.

This week's first paper is "Social cheating in *Pseudomonas aeruginosa* quorum sensing", by Kelsi Sandoz, Shelby Mitzimberg, and Martin Schuster of Oregon State University, just published [online](#) in the Proceedings of the National Academy of Science. (I will also discuss another recent paper on the evolution of "persister" cells.)

Pseudomonas aeruginosa is a pathogen of humans that typically uses quorum sensing to control production of virulence factors. However, mutants that fail to respond to quorum sensing signals are commonly found in patients. The authors of this week's paper suggest that these mutants are "cheaters" (see above) because they benefit from the virulence factors produced by others, without paying the metabolic cost of making these factors themselves. (Just as some humans listen to listener-supported radio without contributing.)

It's hard to measure costs and benefits to individual bacteria inside sick people, so they developed an experimental system to study cooperation and cheating related to quorum sensing in culture flasks. The main energy source they provided to the bacteria was the protein, casein, which has to be broken down outside of the cells, by excreted enzymes, before the bacteria can use it. These excreted enzymes are normally involved in disease and are controlled by quorum sensing. The bacteria benefit, collectively, by producing these enzymes, but there's an individual cost to make them.

Therefore, it is not surprising that the percentage of the bacterial population not making a key extracellular enzyme increased to 20% in about two weeks. Most of these were "defective" in production of several quorum-sensing-controlled factors. When they checked the DNA sequence of a key quorum-sensing gene, all were found to be mutants. The more of these cheaters there were, the slower the overall population growth. When the cheater and cooperator were grown together, the cheater had a generation time of 206 minutes, while the cooperators grew more slowly, with a generation time of 335 minutes.

Given their faster reproduction, what prevents these cheaters from taking over? If there are too many of them, the overall population growth would be slowed by lack of extracellular enzymes. However, I wouldn't expect this to hurt the cheaters as much as it hurts the cooperators, with their higher costs. They say that "compensatory mutations appeared to emerge before the cheater load became detrimental to the entire population, essentially converting cheaters into cooperators." I don't understand this. Mutations are usually random, with respect to their effects on fitness, and I wouldn't usually expect a mutation that helps others to spread. This aspect of the research needs to be explored further.

Thanks to my brother, Glenn, for suggesting this paper.

The second [paper](#) is a theoretical analysis of bacterial "persisters." These cells are alive, but so dormant that they are resistant to many antibiotics that would kill them if they were more active.

Andy Gardner, Stuart West, and Ashleigh Griffin, of the University of Edinburgh, point out that becoming a persister can benefit bacteria in two different ways. The direct benefit is resistance to catastrophes, such as antibiotics.

But there is also an indirect benefit. Persisters don't consume resources, so they free up resources for other bacteria nearby. If those other bacteria are close relatives (especially clone-mates), then a gene that increases the chance of a bacterium becoming a persister may increase the survival and reproduction of other copies of itself, consistent with the selfish-gene and kin-selection hypotheses. On the negative side, as long as a cell remains in the persister state, it is not reproducing.

So, what percent of a given bacterial clone should become persisters? It depends on:
1) the frequency of catastrophes (make more persisters if catastrophes are frequent)

- 2) the availability of resources (if resources are scarce, a cell is giving up less potential reproduction in becoming a persister, and relatives will benefit more from the reduction in demand for resources), and
- 3) how related the bacteria are (freeing up resources for use by nonrelatives is a losing strategy).

Testing these predictions should be fun.

Posted by R. Ford Denison on September 27, 2007 05:27 PM | [Permalink](#)

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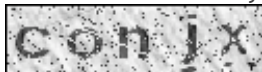
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