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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Communication doesn't automatically prevent cheating

> *There are enough examples of ''cheating'' in bacteria ... that mindless obedience to such [quorum-sensing] chemical signals cannot be assumed. Mindlessness can be assumed, but not obedience. -- Denison et al. (2003) Ecology 84:838-845*

Millions of cooperating cells can do things far beyond the ability of an individual cell. This is most obvious in multicellular organisms, whose cells cooperate because they are all genetically identical, or nearly so. Genetically diverse populations of cells could often benefit from cooperating, but do they? For example, the mixed bacteria populations associated with plant roots might benefit from keeping the plant healthy, so that it can continue to feed them with its root exudates. But for this to happen, they need some method of coordinating their plant-benefiting activities. Furthermore, cells whose genes lead to this form of cooperation must, on average, survive and reproduce more than "cheaters" who don't invest in cooperative activities. Otherwise, cooperative traits will disappear.

Quorum sensing, an exchange of chemical signals among bacteria, can solve the coordination problem. But this week's paper Cooperation and conflict in quorum-sensing bacterial populations shows that quorum sensing doesn't automatically solve the problem of cheaters. The paper is by Stephen Diggle, Ashleigh Griffin, Genevieve Campbell, and Stuart West and published in *Nature*.

There are two key elements to quorum sensing: 1) release of signal molecules, which build up when many signal-producing bacteria are nearby, and 2) responding to high concentrations of signal molecules by doing something that is only beneficial if many cells do it at once. For example, the pathogen *Pseudomonas aeruginosa* typically only produces protein-attacking enzymes when they can produce enough to be effective in digesting the lung tissue of whoever they're infecting. Quorum sensing is used to determine whether enough cells are present to do this. Mutants exist that fail to produce the signal ("signal-negative") or fail to respond to the signal ("signal-blind"). Are there any conditions under which either mutant would tend to become more common?

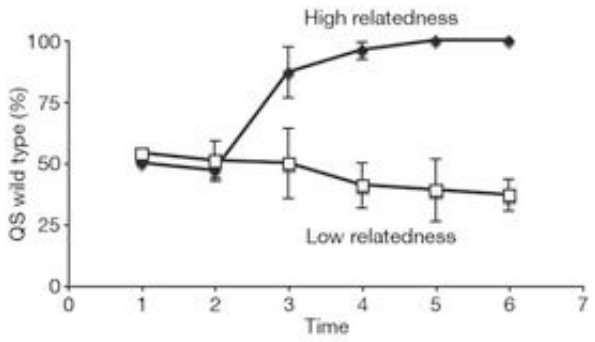
To find out, the authors started with genetically uniform cultures of normal (quorum-sensing "wild-type") and mutant bacteria. The only way to get energy in their "quorum-sensing medium" was by breaking down a protein, using the same quorum-sensing-controlled protease enzymes that these pathogens normally use to digest their host's proteins. Under these conditions, a pure culture of either mutant grew slower than the wild-type bacteria. Adding signal molecules increased the growth of signal-negative but not signal-blind cells. So if everyone cheats -- can you still call it cheating then? -- everyone loses.

On the other hand, when they mixed a few cheating mutants into a population of wild-type bacteria, the signal-blind cheaters increased from 1% of the population at the beginning to 45% after 48 hours. These cheats got to use the protein digested by the normal strain, without paying the metabolic cost of producing and releasing the

protein-degrading enzyme. Similarly, the signal-negative mutant saved the cost of producing signal, and increased from 3% to 66% of the population.

Cheating mutants must arise all the time by random mutation, so why are they fairly rare in nature, rather than 45-66% of the population? Kin selection, maybe. If bacteria usually interact with others of the same genotype (high Hamilton's "relatedness"), then cheaters mostly cheat other cheaters and wild-type quorum-sensing cooperators get to cooperate with each other.

To test this hypothesis, the authors manipulated "relatedness" and let quorum-sensing evolve over six rounds of growth. For high "relatedness" they started each round with a single colony (Hamilton's r=1), while the low-relatedness treatment got the mix of genotypes that evolved in the previous round. Starting in round 1 with a 50:50 mix of quorum-sensing wild-type and cheaters, the frequency of quorum-sensing cells went to nearly 100% when relatedness was set to 100% at the beginning of each cycle, while it fell to about 35% when relatedness was low, as shown in this graph from their paper.



What levels of relatedness are found in actual *P. aeruginosa* infections? An infection starting from a single cell might be expected to have high relatedness, due to very high genetic similarity among cells reproducing by cell division. But relatedness is measured relative to the population with which individuals compete. In a Grafen diagram, an isolated population has B close to A, but P close to B, for a low value of Hamilton's r. So the authors suggest that *P. aeruginosa* relatedness may actually be fairly low in long-term lung infections, as in patients with cystic fibrosis. Quorum-sensing cheaters have been found in these patients. When the bacteria cycle back and forth between cooperating (or cheating) within a host, and then competing for hosts, quorum sensing should be favored.

Additional commentary and pictures at Not Exactly Rocket Science.

Also this week:

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Posted by R. Ford Denison on November 16, 2007 11:08 PM | Permalink

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