Dispatches

Cooperation: Bridging Ecology and Sociobiology

Ecology is considered central to the evolution of cooperation, but there is little direct evidence for this. New support for the idea has come from a study which shifted the path of evolution from cooperation to cheating in flasks of bacteria, simply by altering their disturbance regime.

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Cooperation does not always pay, at least from an evolutionary standpoint. Natural selection will only favor social actions in certain ecological contexts. This is easier said than shown, however, and the most stringent test would require one to manipulate the ecology of a social species and then assess the effect on its evolution. But is this even possible? The last few years have seen a suite of studies that demonstrate the power of microorganisms to answer difficult questions in sociobiology [1,2]. In this issue of *Current Biology*, Brockhurst *et al.* [3] add another arrow to the microbes' quiver. Using aquatic cultures of the bacterium *Pseudomonas fluorescens*, they show that the frequency of ecological disturbance is key to the evolution of cooperation.

The importance of ecology in the evolution of cooperation has long been recognized. In his epic wanderings through nineteenth-century Siberia, Prince Kropotkin (Figure 1A) was struck by how the bitter conditions and "terrible snow-storms" must force cooperation within species — so much so that he wrote a book, somewhat neglected by biologists, called Mutual Aid [4,5]. A passionate anarchist, Kropotkin's discussion is laden with moral interpretation, and it lacks a consistent account of the evolutionary processes behind cooperation. Nevertheless, his central message has a surprisingly contemporary air: cooperation is everywhere, particularly when the conditions are right.

Jump now to 1960s London, where Bill Hamilton is busily scribbling down what will form the theoretical foundations for modern sociobiology, now known as inclusive fitness, or kin selection, theory. Hamilton's insight was simple but extremely elegant. A cooperative act that carries a personal cost, c, can be favored when it provides a benefit, b, to a related individual, because it does not matter to natural selection whether you or another individual pass on copies of your genes, just so long as they get passed on [6]. He captured this logic in his often cited rule $br > c$, where r is the genetic relatedness among actor and recipient [1,2]. Indeed, one can argue this rule is too-often cited in place of alternative and equally-valid approaches, like multi-level selection theory [7]. We do so here to make a specific point. Although most work on Hamilton's theory focuses on genetic relatedness, this 'r' is only one term of three in his equation. The other two are all about ecology.

The ecological nature of Hamilton's rule is missed by some, who mistake the contemporary focus on relatedness for an error in Hamilton's reasoning [8,9]. Even so, sociobiology *does* tend to neglect ecology [10], particularly now that relatedness is routinely quantified using genetic markers. The neglect is not total, however. There has been excellent work on vertebrates that emphasizes the role of ecology in decisions to stay and the help in social groups [11,12]. Meanwhile, ingenious insect studies have shown that nesting conditions both do [10] and do not [13] affect behavioral proclivities for social life. And the interest in ecology is set to increase, with a book-length treatment due out later this year

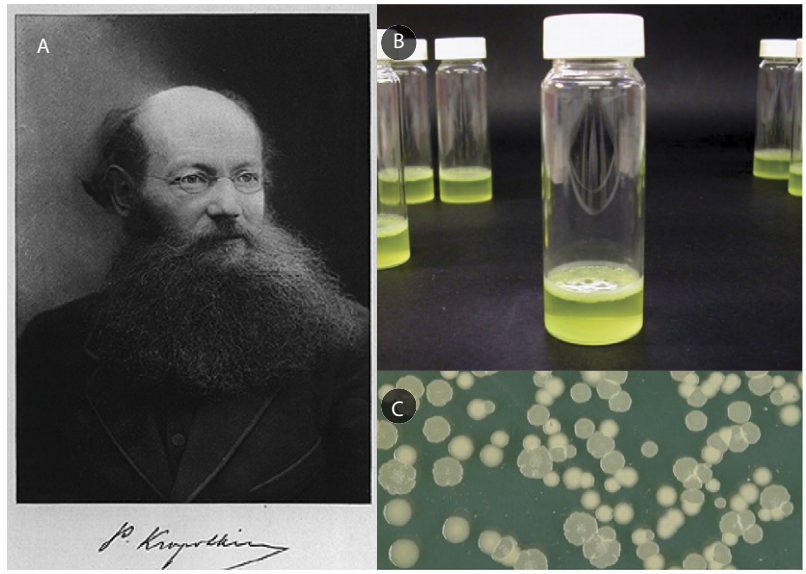

Figure 1. Ecology and social evolution.

(A) The anarchist Prince Kropotkin emphasized the importance of ecology in social evolution. He recognized that cooperation occurred in all walks of life, and hypothesized that one day it might even be found in microbes. (B) An aquatic microcosm containing the wrinkly strain of the bacterium *Pseudomonas fluorescens* showing the floating biofilm. (C) The two strain types as they appear on agar. The larger more defined colonies are the wrinkly spreader. (Photos B and C used with kind permission of Andy Spiers.)

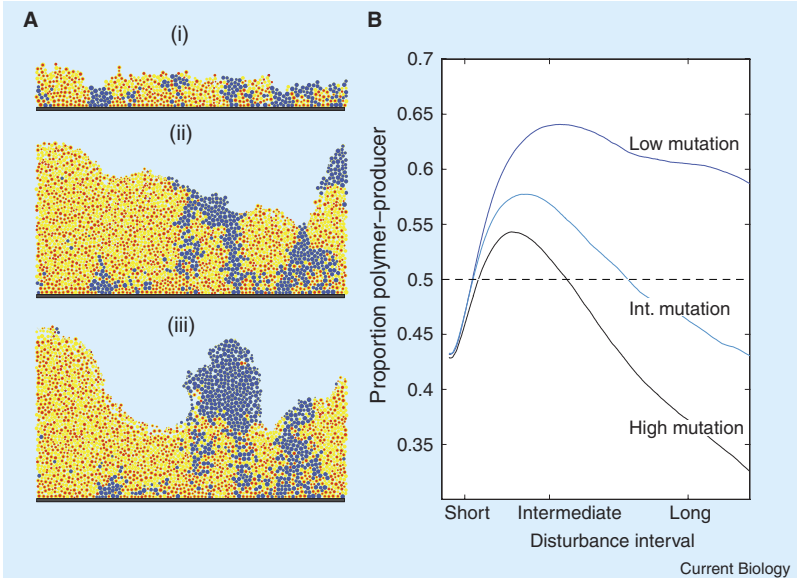

Figure 2. Ecological disturbance can have comparable evolutionary effects in floating and surface-attached biofilms.

(A) An individual-based simulation of a surface-attached biofilm [18] for comparison with floating biofilms [3]. The simulations start with a 1:1 mix of two strains: red are polymer producers, blue are non-polymer producers, and the yellow is the secreted polymer. (i) Short interval: polymer producers lose as polymer is costly to make. (ii) Intermediate interval: polymer producers win by suffocating non-producers [18]. (iii) Long interval: non-polymer producers win because when they arise in the oxygen-rich region, they divide rapidly and release many cells. This last effect did not occur in our original simulation [18] and requires two additional assumptions: first, that cells detach from the biofilm surface throughout development; and second, that there is mutation between the two cell types. (B) Frequency of polymer-producers as a function of biofilm age. Frequency is the number of cells produced during the time interval (number in biofilm + number detached). Probabilities of mutation between strains: 0.003, 0.01 and 0.02 per cell per division.

[14]. Nevertheless, ecological studies are challenging and remain the exception.

This is where microbes enter the fray. Brockhurst *et al.* [3] asked how ecological disturbance affects competition between two strains of *P. fluorescens* (Figure 1B,C). One, the smooth strain, behaves in the mode of a bug of classical microbiology, dividing rapidly in liquid culture and displaying little or no higher-level community structure. The other, the charismatically named wrinkly spreader, grows more slowly but produces a polymer that sticks cells together in a biofilm. Previous work revealed that these strains display a fascinating evolutionary dynamic in liquid culture [15–17]. If one starts with a pure smooth culture, this will be invaded by wrinklies which make a biofilm at the air–water interface (Figure 1B) that partially suffocates the smooth cells below. But new smooth mutants can then invade the wrinkly biofilm by exploiting its buoyancy without paying the cost of polymer production. This later invasion is highly successful, so successful in fact that in time these 'cheater' cells can sink the boat, sending the whole biofilm into the anoxic depths of the beaker [15].

Onto this system, Brockhurst *et al.* [3] superimposed a simple manipulation. They seeded liquid cultures with the wrinkly strain, and then, at varying intervals, took everything and put it in the laboratory equivalent of a blender. The bacteria emerged unscathed and a fraction of the resulting goo was used to reseed another tube, and so on, for sixteen days. The outcome was dramatic. Under the extremes of daily disturbance or no disturbance at all, the wrinkly spreaders (cooperators) could barely survive, and the smooth cells (cheaters) won out. By contrast, at intermediate levels of disturbance the wrinklies were the majority and cooperation dominated.

What caused these effects? The full mechanisms are not yet clear but the Brockhurst *et al.* [3] study opens with a simple mathematical model that offers an answer. At one extreme, too-frequent disturbance prevents the wrinkly polymer-producers from attaining the densities that allow them to monopolize the air–water interface. At the other, too-infrequent disturbance gives smooth mutants time to arise in the floating biofilm and reap the benefits of being at the air–water interface. It is suggested that such processes may be widespread. In support of this, we found comparable processes in a mechanistic model of surface-attached biofilms (Figure 2), although polymer-production is less cooperative in our case. With surface attachment, polymer is less needed for biofilm integrity and may primarily function to suffocate unrelated cells [18].

But what does this all mean for general theory, like Hamilton's $br > c$? Consistent with our opening logic, the high disturbance treatment is easily interpreted in terms of changing costs and benefits. With frequent disturbances, there is never the time to reap the benefit of polymer-production that would outweigh production costs. The undisturbed treatment is somewhat more complicated, and in many ways more interesting, because it is not clear that the benefit b of cooperation is changed relative to intermediate disturbance. Instead, it is thought to be the appearance of smooth cells in the biofilm that causes the wrinklies to lose. And this functions by driving down r: the genetic relatedness between each wrinkly polymer-producer and the cells in the biofilm that it benefits [1,2]. It turns out that no term in Hamilton's rule can escape ecology.

There are also medical implications. *P. fluorescens* is relatively harmless but the degree of cooperation in more pathogenic species is thought to affect their virulence, antibiotic resistance and overall persistence [19,20]. Whether cooperation is good or

bad from a medical perspective will depend on each pathogen's mode of action. But either way, antimicrobial treatment represents an ecological disturbance which can strongly affect a pathogen's cooperation and, therefore, the harm it causes. In the end though, we return to the Anarchist Prince, Kropotkin, who would probably have very much approved of this latest research development. Not only does it cement the link between ecology and sociality that he found so striking, it does so in among the smallest and simplest of cooperators. So simple, one might even call them pond-life.

> "…*we must be prepared to learn some day, from the students of microscopical pond-life, facts of unconscious mutual support, even from the life of micro-organisms*."
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> Kropotkin, *Mutual Aid*, 1902

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DOI: 10.1016/j.cub.2007.03.005

Plant Meristems: Cytokinins — The Alpha and Omega of the Meristem

Recent studies have revealed important new details of how cytokinin-dependent mechanisms control plant growth. Intriguingly, cytokinins are involved in both maintaining meristems and promoting differentiation.

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Cytokinins were discovered 50 years ago as small adenine-derived molecules essential for plant growth and cell division [1]. The last decade saw the discovery of genes required for the synthesis, degradation and perception of cytokinins, and for their immediate downstream transcriptional effectors [2,3]. Despite these advances, how cytokinins cause changes to plant growth behavior is still poorly understood. Four new studies [4–7] have revealed exciting new mechanistic details of how cytokinins are required for meristem maintenance, symbiotic nodule formation and for differentiation at the apical end of the root meristem.

The committing step of cytokinin synthesis is catalyzed by iso-pentenyl transferases, optionally followed by a cytochrome P450-dependent hydroxylation reaction, after which the sugar-phosphate moiety is cleaved off to yield the active cytokinin [3]. Cytokinins are perceived by transmembrane histidine kinase receptors (AHKs) and the ensuing phosphorelay is then transmitted to the nucleus via AHP proteins to regulate the activity of genes encoding response regulators (ARRs) [2]. ARRs come in two flavors: the B-type, which transcriptionally regulate many genes, including the A-type, which interfere with AHP function to dampen the level of responsivity to cytokinin. Thus, the proximal cytokinin effector network is constructed with a negative feedback loop to control the magnitude of subsequent responses [2], and this provides an elegant mechanism for other inputs to control cellular responses to cytokinin [8].

Shoot apical meristem activity requires high cytokinin levels, and at least two components of the meristem gene regulatory network responsible for maintenance of stem cells and their indeterminate progeny are known: KNOX transcription factors, which are expressed in undifferentiated cells of the meristem, activate the expression of at least two genes for iso-pentenyl transferases, while WUSCHEL (WUS), which is required to maintain the stem cell niche of the shoot apical meristem, negatively regulates the expression of some genes for