Evolutionary Ecology: Wasp Mother’s Little Helper

The medical application of antibiotics dramatically reduced human infant mortality in the previous century. A new study indicates that ground nesting wasps exploit Streptomyces strains that they rear in their antennae for the same purpose.

Jacobus J. Boomsma and Duur K. Aanen

Digger wasp females build nest burrows in the soil and usually specialize in hunting specific insects or spiders [1]. Some kill their prey immediately, but most have sting venoms that merely paralyze their victims. This adaptation makes good sense. After filling a brood cell with several prey specimens, the wasp mother lays an egg and seals the cell. This practice ensures every larva has sufficient food to complete development, but does not allow a mother to inspect and groom her grubs later on. Keeping prey alive until they are almost eaten is a clever way to reduce the risk that larvae have to compete with microorganisms that would quickly multiply in dead decaying prey [2]. But longer lasting protection against microbial assault is more difficult. This is particularly so when it takes many months for the young wasps to eclose from nest cells excavated in moist contagious soil, where there are often fragments of dead prey still lying around.

In this issue of Current Biology, Kaltenpoth et al. [3] document that one of the largest digger wasps, the European beewolf, Philanthus triangulum, has found a remarkably innovative solution to the problem of protecting its larvae against infections during their nine months diapause. It has domesticated a Streptomyces bacterium that apparently produces antibiotics to provide long-term antiseptic cradles for the developing wasp brood. The female wasps rear the Streptomyces in special cavities of their antennae. After having provisioned a nest cell with one to fiveparalysed honey bees, the mother smears the ceiling of the cell with a white bacterial mass that she presses out of these antennal cavity ‘glands’ (Figure 1A). The developing larva stays in close contact with this Streptomyces paste and somehow manages to get the bacteria incorporated in the velum of a silk cocoon that it produces after it has finished feeding. This bacterial gift appears crucial for survival. When deprived of these little helpers, almost none of the wasp brood completes development, whereas most of them do when the bacteria are present.

Fighting human infant mortality by antibiotic treatment is now routine practice in developed countries, but has a history of less than a century. It started in 1928 when Alexander Fleming [4] discovered that the fungus infecting his bacterial lab cultures excreted a substance, penicillin, that kills bacteria. Developments were rapid after this breakthrough: we now have an entire suite of antibiotics and use them in large quantities to treat and prevent infections of our livestock and ourselves [5]. Most of these are similarly derived from Streptomyces bacteria [6], but insects ‘discovered’ the essence of this medical practice millions of years earlier than we did and achieved effective reductions of grub mortality by the forces of natural selection alone.

The use of antibiotics in food production is also not a unique human invention. A few years ago, the fungus-growing ants were shown to have domesticated Pseudonocardia bacteria — relatives of Streptomyces — to control a specific fungal disease, Escovopsis, of their mutualistic fungus gardens [7,8]. This pest control symbiosis probably goes back to the very start of fungus-farming by attine ants around 50 million years ago [9,10]. The ants grow the bacteria on their cuticle and can actively regulate their abundance in response to the severity of infections [7,11].

Kaltenpoth et al. [3] provide partial evidence that the entire Philanthus genus may have antennal Streptomyces symbions, which indicates that the bacterial mutualists have co-evolved with these digger wasps, as suggested to be true of the symbiosis between attine ants and Pseudonocardia bacteria [12]. Remarkably, both the bacterial symbions of beewolves and those of fungus-growing ants have been staring biologist in the face without being recognized. The conspicuous white mass in beewolf nest cells has been known for a long time, but was interpreted only as a sign to direct the newly eclosed adults back to the central burrow and the outside world [13].

Similarly, ant researchers knew that fungus-growing ants could be completely covered in a white waxy bloom (Figure 1B) [14], but until the recent work by Currie and coworkers [7,8] an adaptive explanation was not even attempted. One of the major implications of the Kaltenpoth et al. [3] study is therefore that symbioses between insects and Actinomycetes — the group to which both Streptomyces and Pseudonocardia belong — have arisen multiple times and deserve to be more actively looked for in other insects as well.

Many details of how these mutualistic interactions work are waiting to be clarified. We do not know how genetically diverse these symbions are, or on what substrate the insects rear them. Evolutionary theory, however, is powerful enough to formulate testable hypotheses on the type of biological properties that would keep these protective mutualisms stable. One prediction is that there may be many genetically different
strains of symbiont in a single breeding population, but that each host female carries only a single strain that is vertically transmitted by a single parent (from mother to daughters). If this were not the case, the strains growing on the same insect body would compete, which would make them less effective as ‘helpers’ for the wasps. This implies that hosts are generally under selection to avoid the mixing of symbiont-lineages [15].

Vertical transmission by a single parent is a very effective way to achieve this goal, and the insect order Hymenoptera — to which both beewolves and fungus-growing ants belong — may be pre-adapted for this kind of uniparentally transmitted symbionts, because males do not contribute to nest provisioning or brood care. The principle of symbiont competition has recently been shown to explain why fungus-growing ants rear a single strain of fungus per nest [16,17]. Vertical transmission has indeed been convincingly inferred for the Actinomycete symbionts of fungus-growing ants [7,12,18] and has been implicated for the symbionts of beewolves by Kaltenpoth et al. [3], but final evidence based on DNA sequences is still lacking.

In human medication and husbandry, the evolution of resistant pathogens is a serious problem [5,19], so it is interesting to contemplate how insect-microbe symbioses might have tackled this kind of problem. The potential to evolve resistance depends on the working mechanism and operational spectrum of a particular antibiotic. The _Pseudonocardia_ symbiont of fungus-growing ants produces a ‘narrow spectrum’ antibiotic that specifically targets a single fungal disease [7]. The Actinomycete has therefore likely maintained its competitive edge in a direct coevolutionary arms race with its natural enemy [12].

Details about specificity and working mechanism for the _Streptomyces_ mutualist of beewolves remain to be clarified. It is possible that the antibiotic is merely a hygienic defense against a wide array of soil borne microbial saprophytes and non-specific insect pathogens, in which case resistance problems are unlikely to be common. Without detailed bioassays on specificity, however, it may be premature to entirely dismiss the possibility of a ‘narrow spectrum’ antibiotic function of this symbiont. Beewolves specialize on honey bee prey — social insects that have a number of unique brood diseases [20]. These are normally controlled by active grooming of adults and brood in the hive, but are also transmitted by the honeybee workers [20]. If one of these fungal or bacterial pathogens were able to multiply in beewolf pupae, the selection force responsible for the domestication of _Streptomyces_ might ultimately have been contagious prey rather than contagious soil.

Sophisticated forms of medication are counted among the triumphs of modern science and our increasing dependence on pharmaceuticals has become a distinct characteristic of human civilization. The best we may hope for is that our science can stay ahead of the brutal forces of mutation and natural selection that tend to favor our germs in an increasingly globalized and densely populated world. In the midst of these developments, we should realize that the insects that have managed to domesticate antibiotics-producing bacteria have probably been able to avoid or overcome resistance problems. Detailed studies of how this is achieved may thus provide important keys for our own long-term survival.

References

Protein Localization: Reach out and Touch the Forespore

Bacterial proteins are typically sorted to subcellular regions with distinct physical characteristics that serve as cellular ‘addresses’, but many proteins are evidently sorted to specific areas that lack any apparent unique identity. Recent work in Bacillus subtilis suggests that such proteins may be localized by interacting with extracellular domains of proteins in an adjacent cellular compartment.

Kumaran S. Ramamurthi and Richard M. Losick

The proper localization of a protein depends on two parameters. First, the protein itself must harbor a localization signal that specifies its ultimate destination. Second, this destination must harbor a chemical landmark that distinguishes it from other regions of the cell [1]. Some proteins, however, are targeted to specific subcellular locations with no obvious unique physical characteristic. In bacteria, these regions are often patches of membrane, either at the cell poles or at recently created cell division septa. To date, it is unclear how a cell distinguishes these subcellular sites from other sites in the cell. Two recent studies [2,3] have revealed a mechanism by which this protein localization occurs during the process of sporulation in the Gram-positive bacterium Bacillus subtilis.

A hallmark of sporulation is the formation of an asymmetrically positioned division septum (the polar septum) which divides the developing cell into two adjacent, but unequal-sized compartments called the forespore (the smaller cell) and the mother cell [4,5] (Figure 1). Surrounding the cells is the bacterium’s cell wall, which keeps the forespore and the mother cell adjoined. The polar septum that separates the two cells initially contains a layer of peptidoglycan, but this cell wall material is degraded shortly after its formation, leaving the septal membranes of the mother-cell and forespore in close proximity.

The mother cell is known to elaborate a large number of proteins that come to localize on the mother-cell face of the division septum [6–8]. Evidence indicates that these proteins are initially inserted indiscriminately into the membrane surrounding the mother cell and are rapidly recruited to the polar septum by a diffusion-and-capture mechanism [9].

In subsequent development, the septal membrane of the mother cell migrates around the forespore in a phagocytic-like process that eventually results in complete engulfment of the forespore within the cytoplasm of the mother cell. Proteins that have been deposited on the mother-cell face of the septum remain associated with the septal membrane during this encapsulation process such that when engulfment is complete the forespore is fully enveloped by membrane decorated with proteins that had originally been localized to the polar septum.

Prior to engulfment, the septal membrane is contiguous with the remainder of the plasma membrane and delineates the outer boundary of the mother cell. What then is special about the septal membrane that provides a unique chemical environment for the capture of specific sporulation proteins? Perhaps the septal membrane is embedded with certain proteins that provide a landmark for the recruitment of other proteins. But if such landmark proteins exist — and, as we shall see, at least one such landmark protein has been identified — this merely begs the question: how do septal landmark proteins come to localize specifically to one patch of membrane in the mother cell?

The reports by Blaylock et al. [2] and by Doan et al. [3] indicate that the answer lies in the fact that the septal membrane is adjacent to the forespore, whereas the remainder of the plasma membrane faces cell wall. Remarkably, the extracellular domain of an integral membrane protein called SpoIIQ (henceforth simply Q) produced in the forespore directly contacts, and thereby anchors, the extracellular domain of an integral membrane protein called SpoIIIAH (henceforth simply AH) from the mother cell. Indeed, the recognition that Q has an extracellular domain prompted the suggestion some years ago that it might be able to interact with proteins in the mother cell [10]. In other words, AH and Q reach out to each other across the two cells, thereby anchoring AH specifically in the patch of membrane that is within contact with its counterpart in the forespore.

Blaylock et al. [2] found that, in the absence of Q, AH does not localize to the septum, but is