Disease and social evolution

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Parasites that cause diseases of varying intensities are ubiquitous in the natural world. However, the role of disease as a factor in shaping the ecology and evolution of their hosts has only recently begun to be sufficiently appreciated. Parasite load has, for example, been shown to be an important parameter that females use to assess the quality of their mates. In response, males are known to evolve elaborate secondary sexual characters to impress upon females their health in general and their ability to resist parasites in particular[1,2]. An even more profound role that parasites play in modulating social evolution has recently come to light.

In social insects such as ants, bees and wasps (belonging to the order Hymenoptera) males usually develop from unfertilized eggs while females develop from fertilized eggs. This *haplo-diploid* mode of genetics results in curious asymmetries in genetic relatedness. For example, two full-sisters are related to each other by 3/4 rather than by 1/2 as in diploid organisms. Such an asymmetry in genetic relatedness can potentially promote the evolution of social life in these insects. This is because, a female wasp for example, can gain more fitness by rearing her full-sisters (coefficient of genetic relatedness = 3/4) rather than an equal number of offspring (coefficient of genetic relatedness = 1/2)[3,4].

Queens in many social Hymenoptera mate with several males and simultaneously use sperm from different males to produce several patrilines of daughters (this phenomenon is known as *polyandry*)[5,6]. Daughters belonging to different patrilines would of course be half-sisters of each other with a coefficient of genetic relatedness of 1/4. Fitness gained by rearing half-sisters would obviously be considerably less than that gained by rearing offspring. This habit of multiple mating by the queens should therefore decrease the propensity of the queen's daughter to stay back in her nest and help her raise more daughters. The question therefore is why should queens mate multiply? Would they not be better off mating with a single male and thus ensuring the cooperation of their daughters? In search of a solution to this apparent paradox people have begun to focus on possible advantages of genetic variability (provided by the presence of multiple patrilines) within a colony. Several such factors have been postulated although their involvement in providing a selective advantage for multiple mating has not so far been conclusively demonstrated. For instance, to the extent that task performance in the colony has a genetic basis (and there is some evidence for this[7,8]) genetic variability provides for more efficient division of labour. A somewhat different kind of argument is that intra-colony genetic variability could provide effective resistance to diseases which might otherwise spread rapidly when all workers in a colony are highly related to each other and thus sensitive to the same parasites[9,10].

Recent studies of the bumble bee, *Bombus terrestris* and its intestinal trypanosome parasite, *Crithidia bombi* not only confirm such an advantage for intra-colony genetic variability but demonstrate that disease can have rather complex effects on the dynamics of social evolution[11-13]. *Crithidia bombi* spreads from one bumble bee to another through the ingestion by bumble bees of live parasite cells during direct physical contact or through contact with faeces of infected animals. *Bombus terrestris*, living in a temperate environment, suspends colonial life during winter when new queens hibernate while old queens and all workers die. In the following spring, queens emerge from their hibernacula and initiate new colonies. The queen first produce daughters who become workers and later, with the help of this labour force, they produce daughters who mate, hibernate and become queens in the next year. The parasite depends for its continued survival across years, on infecting new queens before they begin to hibernate. Infected queens are likely to pass the infection on to their daughter workers as well as daughter queens because of the possibility of contacting infection through physical contact as well as contact through faeces within their colonies. Laboratory experiments performed by Shykoff and Schmid-Hempel in Switzerland show that the spread of infection from one bumble bee to another depends significantly on the genetic relatedness between the source and recipient of infection[11,12]. This suggests a genetic basis for susceptibility and supports the idea that infection would spread rapidly in a relatively genetically homogenous colony compared to a colony with genetically more variable individuals. Given a reasonable chance of their being infected, queens who mate multiply and produce genetically variable daughters should therefore be at an advantage compared to queens who mate singly and produce genetically similar daughters. Disease is thus a potential factor that selects for multiple mating by the queen.

The *Bombus–Crithidia* story has other fascinating ramifications however. In normal uninfected colonies, workers begin by having poorly developed ovaries and spending all their time working for the colony to rear the queen's (their mother's) brood. With time however workers gradually develop their ovaries and towards the end of the colony cycle, virtually revolt against queen-control and begin to lay their own eggs. The success of queens therefore depends upon producing new daughter queens before workers begin to revolt. A queen that dies after producing only workers and no daughter queen gains little, if any, fitness. Curiously, queens seem to benefit from the infected status of their daughter workers. Infection retards the ovarian development of workers and thus keeps them working for longer periods of time and postpones the time of their revolt. Queens therefore have more time to complete the production of their new daughter queens[13]. In principle this should provide an opposing selective force. Since queens benefit from having infected workers and such infection spreads more effectively in genetically similar lines of workers, a queen would be better off mating singly and produce daughters who are all full-sisters of each other. Disease could thus in principle select for single mating instead of multiple mating.

However, there is an unresolved problem here. If workers in a colony are

infected, the queen is bound to be infected too. The parasite which has detrimental effects on workers has similar effects on the queens. Infected queens can start new nests but they lay eggs at a somewhat lower rate compared to uninfected queens. This leads to significantly smaller worker populations in infected laboratory colonies[13]. But, presumably because the laboratory colonies were supplied with unlimited food, this difference in worker populations did not translate into significant differences in the abilities of infected and uninfected queens to produce daughter queens[13]. In nature difference in worker populations between colonies is almost certainly expected to lead to different probabilities of new queen production. Obviously, the critical factor is the magnitude of the disadvantage to an infected queen in nature in terms of reduced probability of producing daughter queens relative to the advantage to her due to the ovarian impairment of her daughter workers which leads to extended cooperation on their part. If the disadvantage of her own infection is greater than the advantage of worker infection then we would expect selection in favour of multiple mating, leading to genetic variability and lower rates of infection. If the disadvantage of her own infection is less than the advantage of worker infection than we would expect selection in favour of single mating leading to genetically similar daughters and consequent high rates of infection of the daughters. All this reasoning is from the queen's point of view because selection on multiple versus single mating is expected to act on the queen—the daughter workers have little say in this matter.

The story does not end there. Multiple mating is only one way of increasing intra-colony genetic variability. The presence of multiple queens either simultaneously or in succession (termed *Polygyny*) is another way in which intra-colony genetic variability can increase substantially in other social insects[14,15]. Here it is entirely possible that workers have some say in the matter of the number of queens. In some ants for example, it is well known that workers not only decide how many queens may be reproductively active in their colony but they even decide which individuals may become reproductively active queens[16–19]. When selection leading to high or low genetic variability can act on the workers, the differential effects of disease on queens and workers can have effects that may be the opposite of what we have seen above. When both polyandry and polygyny are simultaneously present in colonies of a species[14,15,20], i.e. when selection can act on both queens and workers, one might expect a mind-bogglingly complex effect of disease on the dynamics of social evolution.

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