

Feverish honeybees

The therapeutic benefits of fever have been recorded since the time of Hippocrates. At the turn of the 20th century, syphilis was a dreaded disease whose neuro-degenerative effects were terrible and many mental institutions were occupied with patients suffering from it; there was no effective treatment available then. In 1905, Karl Landsteiner showed that fever was able to kill syphilis-causing spirochaetes. Twelve years later, in a discovery that was to earn him the Nobel Prize, Julius Wagner-Jauregg discovered that infecting syphilitic patients with a mild form of fever-causing malaria resulted in remission of the neuropsychiatric condition in 30–50% of patients; the malarial fever apparently arrested the growth of the spirochaetes (the rate of natural remission of the disease was only 1%). The malaria itself was treatable with quinine. Since then, the understanding of the role of fever in combating infection took a new turn. Fever is now considered to be an evolutionary adaptation to fighting infection and is a result of active thermoregulation (Nesse and Williams 1994). It continues to be accepted as a basic evolutionary response by many clinicians (Romanovsky and Szekely 1998; Marik 2000). Kluger (1979) has shown that even poikilothermic lizards actively seek out warm places when infected. Amazingly, it now turns out that honeybee colonies too are capable of mounting a systemic response to infection by developing fever.

Larvae of honeybees (*Apis mellifera*) are often infected by the heat-sensitive fungus *Ascosphaera apis* which causes “chalk brood disease”. The disease derives its name from the chalky mummified masses that the dead larvae resemble. The normal temperature within the brood portion of the hive (brood-comb) is 33–36°C and, within this range, is correlated closely with ambient temperature. Starks *et al* (2000) have found that combs infected with *Ascosphaera* display an elevated temperature; in effect, they get fever. In replicated experiments, they recorded temperatures of the brood-comb in pre-treatment, treatment and post-treatment periods. The pre-treatment period consisted of a pre-feed period of ten days in which colonies were unfed and a feed period of five days in which sugar-water was provided. In the treatment period, colonies were fed for two days on sugar-water containing macerated sporulating mummified larvae; these were sufficient to cause infection. In the post-treatment period, colonies were unfed. The experiment ended on the 30th day. Brood-comb temperatures were significantly higher in the treatment period compared to the pre-treatment period while there was no significant difference between brood-comb temperatures of the pre-feed period and the post-treatment period. Moreover, *A. apis* apparently needs only a slight chilling of the larvae to cause disease. It is clear then that honeybees elevate brood-comb temperatures, which they achieve by increased flight muscle activity, as a defensive response to infection. According to the authors, this prevents larvae from succumbing to the disease. The authors suggest that either honeybee workers detect infestation before symptoms are visible or that the larvae somehow communicate the ingestion of the fungus. In any event, the infection is detected fairly early and triggers the appropriate febrile response.

Here is evidence that the honeybee colony is responding just as a unitary organism would to infection. Starks *et al* (2000) refer to the phenomenon as “a striking example of convergent evolution between this ‘superorganism’ and other fever-producing animals”. In honeybee colonies a single queen does all the egg-laying and the hegemony of the queen is generally accepted, part of the reason being that the workers are often more closely related to each other than to the queen owing to the haplo-diploid system of sex determination. This can lead to an extremely close level of integration at the colony level, along with division of labour. In consequence, colonies of honeybees and other social insects have been described as superorganisms (one colony = one organism), and it appears that colony-level selection rather than individual-level selection is in operation (Seeley 1997; but see Keller and Reeve 1999). The ability of the colony to develop a fever is yet another indication of an organism-like physiological response. Honeybees exhibit other regulatory mechanisms to control elevated nest temperature such as fanning, tonguelashing (water-evaporation from extruded fluid droplets) and partial evacuation of the nest to prevent overheating (Seeley and Heinrich 1981). They also use temperature as a lethal defense against predatory wasps: the wasps are surrounded by workers and get virtually overheated to death (Ono *et al* 1995). And now fever!

References

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