Short communication

Labial gland disease in the genus *Formica* (Formicidae, Hymenoptera)

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Summary

Labial gland disease causes swelling of the labial glands in the pupal stage in *Formica* species. The resulting adults have enlarged thoraces and are called secretergates (Wasmann's pseudogynes). They infect the younger larvae, probably during feeding. The agent of the disease is unknown. It soon becomes non-infectious outside the labial glands. Diseased females were not seen to feed larvae. Yet their offspring contained secretergates. The discrepancy remains unexplained.

Symptoms of labial gland disease are an enlarged and malformed thorax, making worker ants somewhat resemble queens. Wasmann (1915, and numerous other papers) regarded them as worker-queen intercastes, and called them "pseudogynes".

Novák (1948), however, discovered that the enlargement of the thorax was caused by swelling of the reservoirs of the labial glands in the pupal stage. Therefore they were not intercastes, and he suggested the terms "secretergates", "secretogynes" and "secretaners" respectively for workers, females and males suffering from this disorder, and the general term "secretoforms".

Secretoforms have been found in six of the eight European species of the Formica rufa group, and may also occur in F. uralensis Ruzsky and F. nigricans Emergy. They do occur in other species of the genus viz. in F. sanguinea Latreille (Wasmann, 1915), F. fusca Linnaeus (Wasmann, 1915), F. lemani Boudroit (Collingwood, 1956), F. rufibarbis Fabricius (Wasmann, 1915), and in some North American Formica species. They are a major mortality factor in F. sanguinea (Wasmann, 1915: 272–281) and could be so in other species.

The disorder is infectious. Experiments have revealed that secretergates infect the larvae. No symphyles or other ant guests are required for transmission of the disease within the nest. This was established for *Formica rufa* Linnaeus (polygynous form) and *F. polyctena* Foerster ¹, and goes almost certainly for all other species susceptible

A more detailed account of these and subsequent investigations have appeared in Elton 1989.

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to the disease. Infected colonies usually continue to produce secretoforms for prolonged periods, and perhaps indefinitely.

The labial glands of the larvae are the silk glands. They are unaffected at first. The larvae spin normal cocoons. It is not until the pupal stage, when the integument is still distensible, that the reservoirs swell. There are various degrees of swelling. In extreme cases the secretergates resemble small queens. But they never have wings. Intermediate forms between secretergates and normal workers are extremely rare, and were only found in an odd colony. Intermediate forms between workers and queens are common in real intercastes, but have never been found in the field, only in experiments on caste differentiation (Gösswald and Bier, 1954). Occasional reports on secretergates or "pseudogynes" in species other than *Formica*, need checking to ensure that they are not really intercastes.

Diseased females, secretogynes, do have wings. The enlargement of the thorax is not so conspicuous, and intermediate forms between secretogynes and normal females are more common than intermediate forms between secretegates and normal workers.

The cause of the disease is unknown. Ponsen (1965) could not find any bacteria, protozoa or fungi in the diseased glands. But he found globular particles, about 20 nm in diameter, absent in normal glands. Later, Peters found virus-like particles of 25 nm diameter in partly purified extracts of secretergates and, surprisingly, similar particles, though in smaller quantities, in normal workers (personal communication). No further progress has been made in identifying the disease agent.

Laboratory experiments using sugar water colored with magenta (basic fuchsine) showed that secretergates feed all other members of the colony, including larvae. Infection probably takes place during feeding, although it cannot be ascertained whether secretions from the labial glands were transferred, because only the contents of the intestinal tract are colored by magenta, and none of the glands or their secretions.

All members of an ant colony, including secretergates, constantly exchange food with one another (Gösswald and Kloft, 1960; Rosengren, 1979). Therefore, a certain percentage of the food circulating in a diseased colony must have passed through the bodies of secretoforms. Normal workers and normal queens in diseased colonies could, therefore, be infectious. Yet normal individuals could not be shown to carry over the disease from secretergates to larvae. Presumably the agent – whatever it is – becomes non-infectious when outside the labial glands. The period during which normal individuals could possibly be infectious was shown to be four days at most, but could also be a few minutes or seconds.

Brief infectious periods in normal workers might explain that no brood is ever infected in its entirety, whether in the field or in the laboratory. Rarely more than 50% of the larvae developed into secretergates. Some of them would then have been infected directly by secretergates and a minority indirectly by normal workers. Never do all the workers become secretergates, so the disease in itself can seldom be the sole cause of a colony's extinction.

Adult normal workers and queens cannot become permanent carriers of the disease and cannot, therefore, become diseased themselves.

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Diseased females, secretogynes, may reproduce like normal queens. However, they may also be completely sterile (though inseminated) and others may produce brood, most of which is inviable. They were seen to feed normal workers, but not larvae. Yet secretergates developed in laboratory colonies in which secretogynes were the only diseased individuals. Indirect infection by temporarily infectious workers might be the explanation. However, the number of induced secretergates, especially by two *F. rufa* secretogynes (27.6 and 26.1%; N = 116 and 107) seems rather high for indirect infection. The number of secretergates induced by two virgin secretogynes in the brood of a normal queen: 2.7% (N = 148), seems a more likely figure. The discrepancy between the two figures is as yet unexplained.

Brood mortality was excessive in some laboratory colonies. This was due to a reproductive deficiency of some normal, inseminated queens from diseased field colonies. It is also very likely that the normal workers from some – though not all – diseased field colonies were deficient in their brood-rearing capacity. These factors may have contributed to the dying out of certain diseased colonies, despite protection against woodpeckers and human interference.

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