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Dimorphic Development of *Amblyospora* sp. (Microspora: Amblyosporidae) in *Culex salinarius* Gynandromorphs[1,2]

The microsporidium *Amblyospora* sp. of the mosquito *Culex salinarius* exhibits dimorphic development. In vertically infected male hosts the parasite initially infects oenocytes and later invades fat body where it undergoes meiosis (E. I. Hazard, T. G. Andreadis, D. J. Joslyn, and E. A. Ellis, *J. Parasitol.* **65**, 117–122, 1979) and forms large numbers of haploid spores killing most of the larvae as fourth instars. In females, infection is restricted to larval oenocytes. The larval oenocytes are normally restricted to the abdomen where they form groups of five or six closely packed cells on each side of abdominal segments 1 to 8 lying in pockets of the lateral fat body (A. N. Clements, 1963, "The Physiology of Mosquitoes," Macmillan, New York). Uninfected larval oenocytes normally disappear during the pupal or early adult stages. Infected oenocytes persist into the adult stage at which time the parasites multiply rapidly and fill the oenocytes and ultimately form diplokaryotic spores after the female takes a blood meal. At this time the infected oenocytes begin to circulate freely throughout the hemocoel including the thorax and occasionally even the head (D. W. Hall, *J. Amer. Mosq. Control Assoc.* **1**, 514–515, 1985). The diplokaryotic spores are responsible for infection of the ovaries and vertical transmission of the parasite to progeny of infected females (T. G. Andreadis and D. W. Hall, *J. Protozool.* **26**, 444–454, 1979). The haploid spores have been shown to be infective for copepod intermediate hosts (A. W. Sweeny, E. I. Hazard, and M. F. Graham, *J. Invertebr. Pathol.* **46**, 98–102, 1985). Since vertical transmission of the parasite by females plays an important role in its survival and epizootiology (T. G. Andreadis and D. W. Hall, *J. Invertebr. Pathol.* **34**, 152–157, 1979), it is adaptive for it not to infect the fat body of females and kill them as it does males.

By analyzing development of the parasite in the appropriate gynandromorphic hosts, it should be possible to determine whether the difference in susceptibility of the fat body of males and females is due to the inherent difference in genotype of the fat body of the two sexes or to the effect of a humoral factor (e.g., a hormone). Mosquito gynandromorphs are sexual mosaics in which the same individual contains some tissues which are genotypically female and others which are genotypically male. A line of demarcation between male and female tissue is almost always present dividing the specimen in half. Theoretically this division could occur in any plane. Anterior–posterior and bilateral gynandromorphs occur with equal frequency, but the most common forms are oblique gynandromorphs in which the head and one side of the thorax are of one sex and the other side of the thorax and the abdomen are of the other sex. A review of the literature on mosquito gynandromorphs and a discussion of the mechanism by which they are produced have been published by the author (D. W. Hall, *J. Fla. Anti-Mosq. Assoc.* **58**, 25–28, 1987).

In the present study, 51,000 adult *C. salinarius* from an infected colony were examined for the presence of gynandromorphs. Only three gynandromorphs were found, two of which were lightly infected and the third was uninfected. A detailed description

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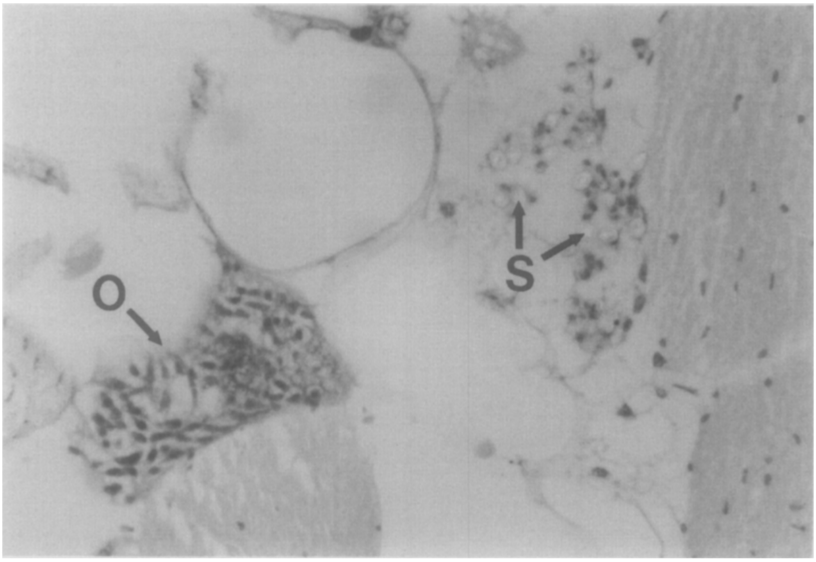

FIG. 1. Histological section of *Amblyospora*-infected *Culex salinarius* gynandromorph. O, oenocyte with diploid stages; S, haploid spores in fat body (Delafield's hematoxylin and eosin). 450×.

of the gynandromorphs is given elsewhere (D. W. Hall, *J. Amer. Mosq. Control Assoc.* 4, 196–197, 1988). Unfortunately, both of the infected individuals were essentially identical in the arrangement of the presumptive male and female tissues. In each specimen, the head and left side of the thorax were female and the right side of the thorax and the abdomen were male. Each individual had normal male external genitalia and well-developed testes. Examination of hematoxylin-stained sections of the infected gynandromorphs revealed the presence of haploid spores in fat body presumed to be genotypically male but none in fat body presumed to be genotypically female. There were also oenocytes with diplokaryotic stages (Fig. 1) in the same area which was presumed to be genotypically male since they originated in the abdomen. The absence of infection in female fat body suggests that the parasite does not develop there due to the inherent genotype of that tissue. Any humoral inhibitory effect would have to originate in female tissues in the head or thorax since all abdominal tissues in the gynandromorphs were presumptively male. It would be expected to also inhibit development in the male fat body in the thorax which was not the case. Firm conclusions cannot be drawn since the specimens were only lightly infected, and the lack of infections in female fat body may have been due to chance. Examination of more gynandromorphs is desirable, but the study was discontinued because of the time required for raising the large numbers of infected mosquitoes and searching for the gynandromorphs.

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