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Susceptibility of sixteen species of Diptera to the fungal pathogen *Entomophthora muscae* (Zygomycetes: Entomophthoraceae)

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Abstract

Sixteen species of Diptera from eight families were inoculated with conidia of *Entomophthora muscae* (Zygomycetes: Entomophthorales). The following species were susceptible and became infected at the rates indicated: *Musca domestica* (100%), *Sarcophaga haemorrhoidalis* (86%), *Phaenicia sericata* (44%), *Scatophaga stercoraria* (30%), *Drosophila melanogaster* (11%), *Aedes aegypti* (3%), and *Stomoxys calcitrans* (2%). The following species were not susceptible under the conditions of this study: *Phormia regina, Calliphora vicina, Rhagoletis pomonella, Eristalis arbustorum, Eristalis tenax, Toxomerus geminatus, Sphaerophoria scripta, Syrphus sp.* and *Allograpta oblique.* Differences in susceptibility were not related to the taxonomic affinities of the taxa tested; however, susceptibility may be related to interspecific morphological differences of hosts, such as, scales and hairs on the host or the degree of sclerotization of host integument.

Each host-pathogen interaction was examined. Characteristics of the post-mortem growth of the fungus were similar within a host species but differed between host species. These differences in post-mortem growth of the fungus were related to the suitability of host species as a substrate for the pathogen. The incubation period of the mycosis was related to host species and not to host size, it varied from seven days for the relatively large *S. haemorrhoidalis* to 17 days for the relatively small *A. aegypti.*

Introduction

Entomophthora muscae (Entomophthoraceae) is a pathogen of adult Diptera that causes epizootics in populations of economically important pest flies such as the house fly, *Musca domestica* [10, 19], the onion maggot, *Delia antiqua* [6], and the seed corn maggot, *Delia platura* [5]. The reputed host range for *E. muscae* is large including species in diverse families such as: Muscidae [14], Drosophilidae [9], Syrphidae [22], and Calliphoridae [10]. MacLeod et al. [16] presented an excellent overview of the literature concerning *E. muscae.*

Even though *E. muscae* has been reported as a pathogen of many different species and families of Diptera, it cannot be assumed that the pathogen was identical in all these cases. Only when a known isolate is used in controlled infection trials can accurate conclusions be drawn regarding its host range. Laboratory studies of the host range and characteristics of the taxon *E. muscae* are particularly important since the taxon is now thought to represent a poorly understood species complex [4, 12].

In spite of the many publications reporting discoveries of natural hosts of *E. muscae* there are few reports of isolates of *E. muscae* being used to infect different fly species in the laboratory. Baird [1] used an isolate of *E. muscae* from *Kellymia kellyi* to in-

fect *Pseudosarcophaga affinis* and *M. domestica.* Wilding [23] was able to transmit *E. muscae* from 'infected Wheat bulb flies, scatophagid flies and syrphid flies to healthy individuals of the same host or host family, but never under identical conditions, from one of these hosts, or host families, to another.' Wilding [24] was unable to infect *Leptohylemya coarctata* with *E. muscae* from cabbage root flies, *Delia brassicae.* Carruthers and Haynes [7] used isolates of *E. muscae* from *D. antiqua* to infect *D. platura* and vice versa. Kramer & Steinkraus [14] published the most complete study on host range of *E. muscae* to date. They found that an isolate of *E. muscae* from *Pollenia rudis* was able to infect *M. domestica, M. autumnalis, D. antiqua* and *D. platura* but not *Stomoxys calcitrans, Phormia regina* or *Muscina stabulans.*

The present study examined the host range of *E. muscae* in greater detail than has been done hitherto. This study adds to our knowledge of insect susceptibility to fungal infection, helps determine potential biocontrol targets for *E. muscae,* helps to clarify problems in the taxonomy of the *E. muscae* species complex, and leads to a greater understanding of the ecology of *E. muscae.*

Materials and methods

Source of experimental insects

The following fly species were obtained from disease-free laboratory cultures: *M. domestica, S. calcitrans, Sarcophaga haemorrhoidalis, P. regina, Phaenicia sericata, Eristalis tenax, Calliphora vicina, Aedes aegypti, Rhagoletis pomonella* and *Drosophila melanogaster.*

The other fly species tested: *Scatophaga stercoraria, Toxomerus geminatus, Eristalis arbustorum Sphaerophoria scripta, Syrphus sp.*, and *Allograpta obliqua*, were collected in the field and brought back to the laboratory for inoculation.

E. muscae *inoculum*

For over six years our laboratory has maintained an isolate of *E. muscae in vivo* by house fly to house fly transmission. This isolate originated from cluster flies (*P. rudis*) and is highly infective and virulent for *M. domestica.* This isolate of *E. muscae* has primary conidia that average 22.2 μ in length and 17.9 μ in width (n = 100), contain an average of five nuclei and have the 'muscae-type' shape, e.g., broadly ellipsoidal conidia with a papillate apex and flattened base.

Test species were inoculated by a method described elsewhere [14]. Test flies were exposed to an average of 34 conidia/mm^2. The density of conidia was determined by placing three coverslips at the bottom of a cage, allowing conidia to fall from five cadavers for three days, then counting the conidia from nine arbitrarily selected fields of view from each coverslip using a compound microscope (160 ×, Nomarski differential contrast, field of view = 0.627 mm^2).

Experimental flies were held at 18°–20°C, 12-12L:D photoperiod, 40–60%RH, fed a moist yeast extract-sucrose-non-fat dry milk mixture, and supplied with water. The same isolate of *E. muscae* and procedure were used in all cases. Six years of experience with this *E. muscae* isolate has shown that this procedure and quantity of inoculum suffices to infect and kill 100% of inoculated *M. domestica* within seven days. Since the only variable in this study was the insect species (technique, fungal isolate and inoculum level were held constant), the differences in infection rates between the various test species reflect differences in species susceptibility. The inoculating method used duplicates the way in which conidia contact and infect insects in nature; therefore, the fungus encounters all natural host barriers to infection. Thus, the host range determined in this study is more meaningful than it would be if hyphal bodies were inoculated directly into the hemolymph bypassing some of the insect's defenses.

Infection criteria

Cages were checked daily for dead flies, which were removed and examined for signs of *E. muscae.* If no conidiophores with conidia were present on the exterior of a dead fly, its hemolymph was examined,

microscopically, for hyphal bodies. Test insects not dying of the mycosis were maintained for up to six weeks after inoculation and then dissected and examined for hyphal bodies.

For purposes of comparison the post-mortem changes associated with *E. muscae* infections in *P. rudis* (Fig. 1) and *M. domestica* (Fig. 2) were used as a standard and deviations from this standard were noted and described. Infected *M. domestica* and *P. rudis* usually die with the labellae of the proboscis attached to a substrate by hyphal holdfasts (rhizoids) and a sticky secretion. In addition, the legs are stiffly outspread, the wings are slightly lifted, the conidiophores emerge in three large white bands through the dorsal membranous intersegmental areas of the abdomen and coalesce ventrally, the conidia production is copious, and the mycosis kills the host within seven days of inoculation.

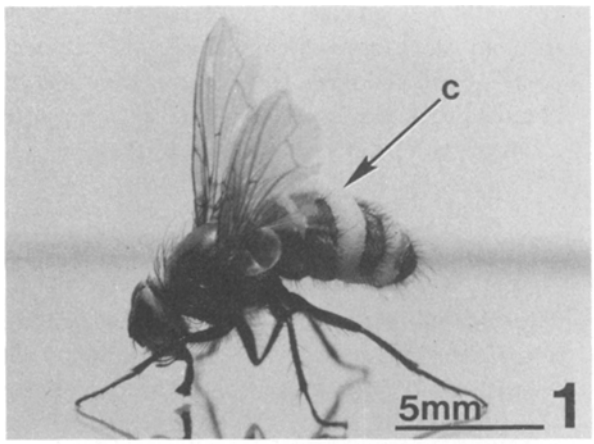
Results and observations

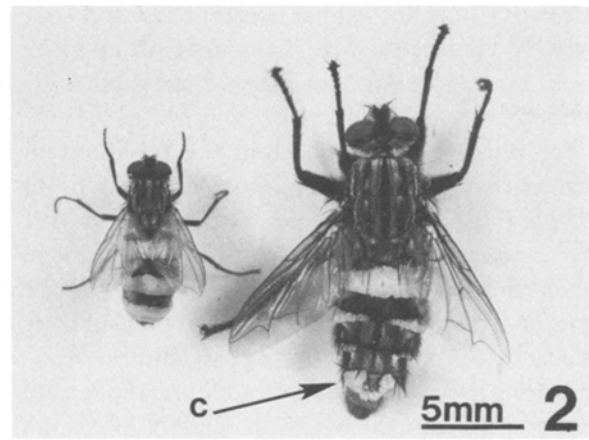
The susceptibility of the test species varied considerably (Table 1). *M. domestica* was the most susceptible with 100% dying with a frank mycosis seven days after inoculation. Other species from several fami-

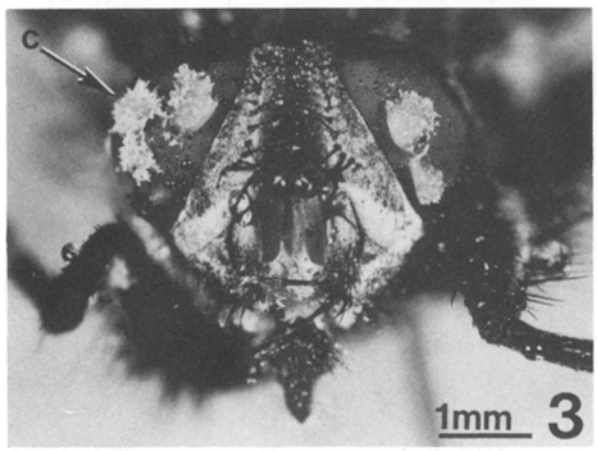
Table 1. Effect of *Entomophthora muscae* on sixteen species of Diptera.

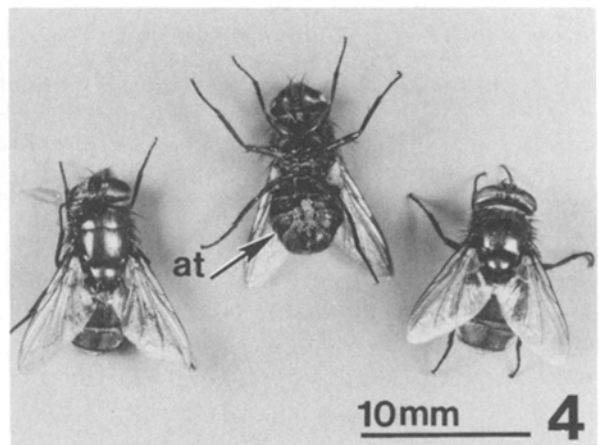
Species	Tested #	Infected #	Frank infections %	Incubation period	Syndrome
(Muscidae)					
Musca domestica	78	78	100	7	typical
Stomoxys calcitrans	90	4[1]	2	11	atypical
(Sarcophagidae)					
Sarcophaga haemorrhoidalis	21	18	86	7	near typical
(Calliphoridae)					
Phaenicia sericata	16	7	44	7	near typical
Phormia regina	24	0	0	-	-
Calliphora vicina	24	0	0	-	-
(Anthomyiidae)					
Scatophaga stercoraria	10	3	30	13	near typical
(Drosophilidae)					
Drosophila melanogaster	100	11	11	7	near typical
(Culicidae)					
Aedes aegypti	31	1	3	17	atypical
(Tephritidae)					
Rhagoletis pomonella	13	0	0	-	-
(Syrphidae)					
Allograpta obliqua	1	0	0	-	-
Eristalis arbustorum	1	0	0	-	-
Eristalis tenax	11	0	0	-	-
Sphaerophoria scripta	2	0	0	-	-
Syrphus sp.	1	0	0	-	-
Toxomerus geminatus	9	0	0	-	-

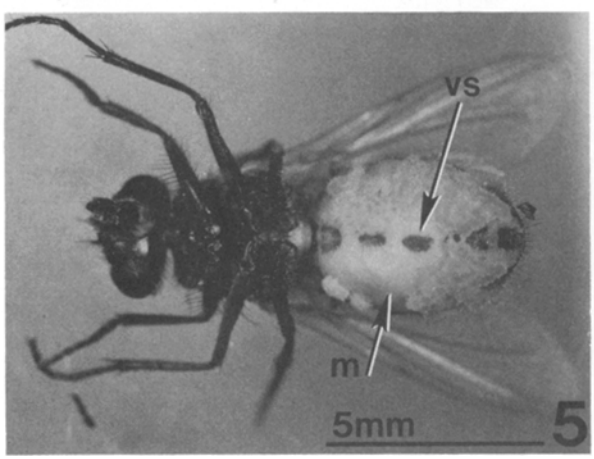
[1] 2 flies produced conidia, 2 had no outward signs and produced abnormal hyphal bodies.

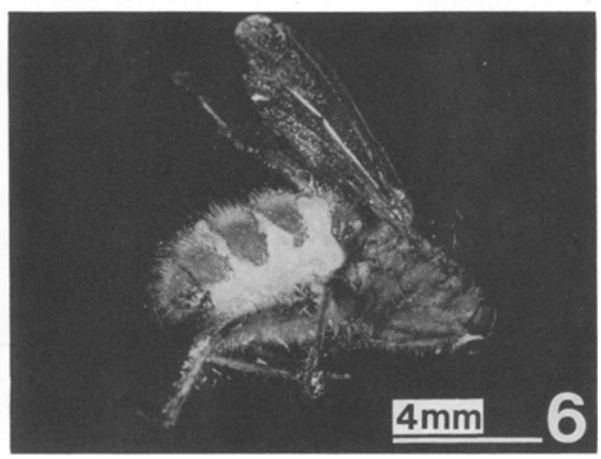












lies were moderately susceptible: *Sarcophaga haemorrhoidalis* (86%), *P. sericata* (44%), *S. stercoraria* (30%), and *D. melagonaster (11%). Two species were slightly susceptible: A. aegypti* (3%) and *S. calcitrans* (2%). The other nine species tested were not susceptible under the conditions of this study. The wide range of susceptibility of the different test insects indicated that insect defenses against infection by *E. muscae* varied in efficience from species to species.

Post-mortem growth of E. muscae *on different host species*

Infection is the result of an interaction between a pathogen and an insect. While the effects of the pathogen upon the host are often obvious, producing disease and death in the host, the effects of the host on the pathgen may be subtle. The host affects the pathogen's growth, development and survival in and/or on the host. Observations made on the growth of *E. muscae* in various host species are important because they provide evidence for the suitability of particular insect species as a substrate for the fungus. Post-mortem growth of *E. muscae* in *P. rudis* (Fig. 1) and *M. domestica* (Fig. 2, at left) was typical. The other susceptible species had one or more atypical features.

Infected *S. haemorrhoidalis* had bands of conidiophores on the dorsum of the abdomen that were incomplete (Fig. 2 at right) and hyphae emerged from the compound eyes (Fig. 3), a fungal growth characteristic never noted in *M. domestica* or any other host. Interestingly, the incubation period of the mycosis in both *M. domestica* and *S. haemorrhoidalis* was seven days even though *S. haemorrhoidalis* is much larger than *M. domestica* (Fig. 2). Fungal growth in *P. sericata* (Fig. 4) was nearly typical; however, the bands of conidiophore were thin and 'waxy' and conidial production was scanty. This feature was also noted in infected calliphorids collected in nature by Graham-Smith [10]. These features may be related to the relatively small membranous areas of the abdomen available for outgrowth of conidiophores as compared to *M. domestica* (compare Fig. 4 with Fig. 5). Fungal growth in *S. stercoraria* was nearly typical, but the legs were not outspread and the proboscis was not attached (Fig. 6). Growth of the fungus in *D. melonagaster* (Fig. 7) was nearly typical, but the production of conidia was relatively meager, probably due to the small size of the host.

The fungus took 17 days to kill *A. aegypti* (Fig. 8) in spite of its comparatively small size. This stands in marked contrast to the seven days the fungus took to destroy the comparatively huge *Sarcophaga haemorrhoidalis* (Fig. 2 on right). The growth of conidiophores was very sparse, typical *E. muscae* conidia were produced but were few in number, the legs were not spread and the proboscis produced no holdfasts. While *A. aegypti* is a new host record for *E. muscae* it would appear that in nature the fungus would not survive well on this host.

Fungal growth in *S. calcitrans* (Fig. 10, on right) was atypical. The legs were not spread, hyphal holdfasts did not grow out of the heavily sclerotized proboscis of this bloodsucking fly, no conidiophores were produced in two out of the four infected individuals (Fig. 10, on left), and the incubation period was 11 days. Abnormal hyphal bodies with large vacuoles (Fig. 11) were found in the two infected *S. calcitrans* that did not produce conidiophores or conidia. In contrast, typical hyphal bodies from *M. domestica* (Fig. 12) lacked these large vacuoles

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Fig. 1. P. rudis infected with *E. muscae*. Typical post-mortem syndrome. Note: raised wings, outspread legs, proboscis extended and attached to substrate, three broad bands of conidiophores (c) emerging through the intersegmental membranes of abdomen.

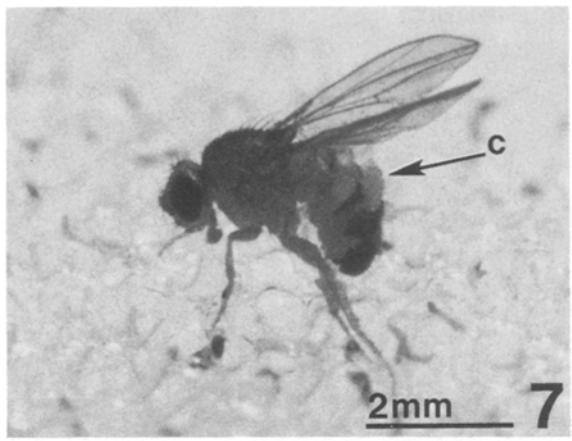
Fig. 2. S. haemorrhoidalis (on right) and *M. domestica* (on left) infected with *E. muscae*. Note typical post mortem growth of fungus on *M. domestica* and nearly typical growth on *S. haemorrhoidalis* except for the bands of conidiophore bands (c) which are incomplete dorsally.

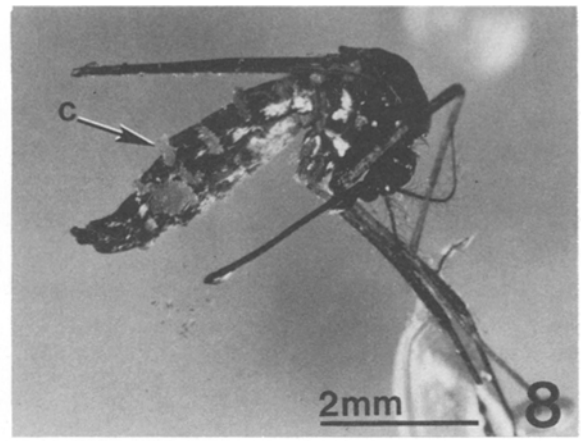
Fig. 3. Conidiophores of *E. muscae* emerging from the compound eyes of an infected *S. haemorrhoidalis*. A characteristic of post-mortem fungal growth not noted in any other susceptible species.

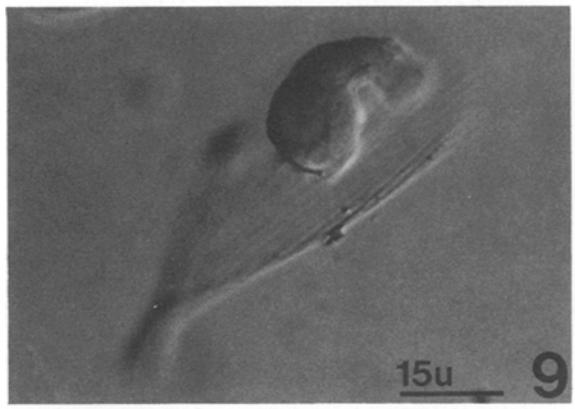
Fig. 4. P. sericata infected with *E. muscae*. The conidiophore bands are thin and waxy and yielded few conidia. Note that the abdominal tergites (at) curve ventrally to meet the ventral sternites leaving relatively small areas of exposed intersegmental membrane. Compare the morphology of the *P. sericata* abdomen to that of *M. domestica* in Fig. 5.

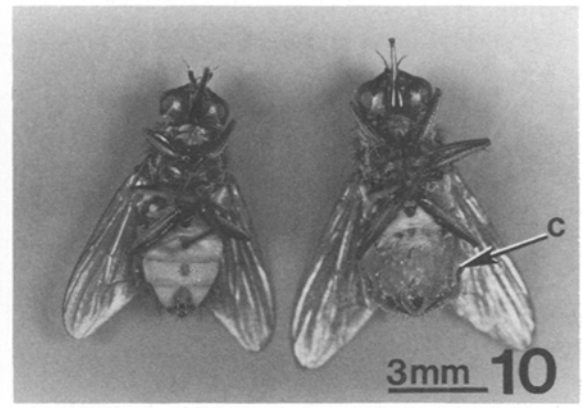
Fig. 5. Ventral view of *M. domestica* infected with *E. muscae*. Note the conidiophores coalescing ventrally and the tiny ventral sternites (vt) surrounded by large areas of thin membranous integument (m). These large areas of thin intersegmental membrane on *M. domestica* seem to make this host especially susceptible to infection and post-mortem growth of the fungus.

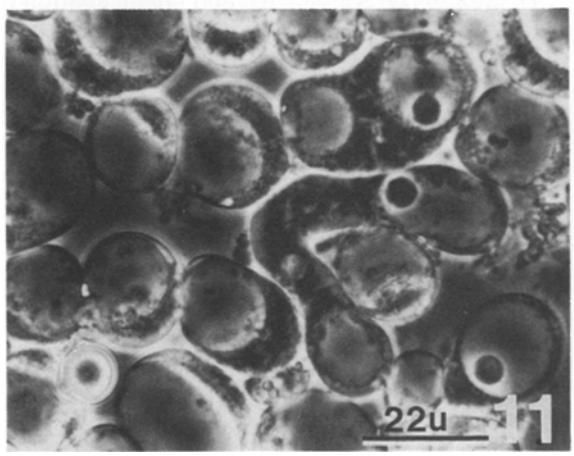
Fig. 6. S. stercoraria infected with *E. muscae*. Note the raised wings, three bands of conidiophores that coalesce ventrally, but the legs are not outspread and the proboscis is not extended.

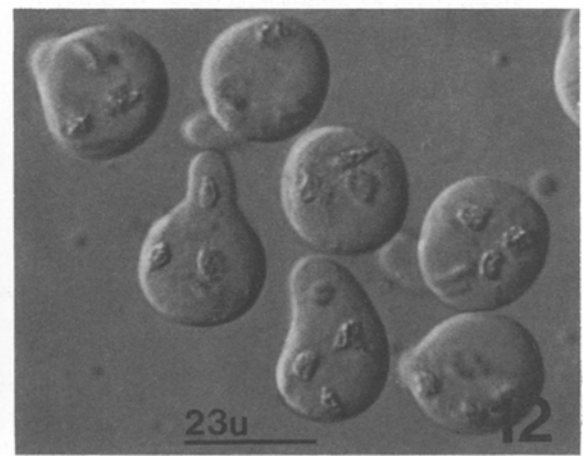












and produced conidiophores. The lack of conidiophores and the presence of abnormal hyphal bodies in two of the infected individuals, and the increase in the incubation period indicated that *S. calcitrans* was not a suitable host for this fungus.

In order to infect an insect, conidia of *E. muscae* must send hyphal germ tubes through the insect integument and into the hemocoel. Zacharuk [25] suggested that conidia of the Entomophthorales probable penetrate the integument in the 'thinner membranous regions.' *A. aegypti* adults are densely covered with overlapping scales on the dor-

sal and ventral surfaces of the abdomen and on the wing veins. Observations with the microscope made on inoculated *A. aegypti* mosquitoes showed that many *E. muscae* conidia landed on the mosquito scales, germinated, but did not penetrate through the scale to the hemocoel (Fig. 9). In these cases. the mosquito scales acted as barriers that protected much of the body surface, reducing the chance that conidia might land on the vulnerable membranous areas. *Phaenicia sericata*, while it does not have scales, does have heavily sclerotized abdominal tergites that curve down around the sides of the abdomen and meet the abdominal sternites so that only a small area of intersegmental membrane is exposed (Fig. 4). In contrast to *A. aegypti* and *P. sericata, M. domestica* not only lacks scales but its abdomen has large expanses of thin intersegmental membrane with only tiny ventral sclerites (Fig. 5). Conidia have a greater chance of landing on thin membranous sites that can be penetrated easily on *M. domestica* than on species like *A. aegypti* or *P. sericata*. This may be one reason why *M. domestica* is so susceptible to *E. muscae*.

Hywel-Jones and Webster[11] noted that rhizoids, cystidia and conidiophores of *Erynia conica* from infected *Simulium sp.* (Simuliidae) emerged only from the thinner parts of the insect's cuticle and rarely emerged through the thick dorsal abdominal tergites. They concluded that the major means of the fungus for rupturing through the host cuticle was by mechanical means. These findings agree with our observations of the growth of *E. muscae* in various hosts. Hosts with large areas of thick sclerites and small areas of thin membrane, such as *S. calcitrans* and *P. sericata*, did not give rise to luxurious exterior growth of the fungal conidiophores. *M. domestica* has large areas of thin membrane on its abdomen that seem to make it very susceptible to penetration by conidia during the infection process and to conidiophores during post-mortem fungal growth.

Discussion

Previous studies of the host range of *E. muscae* were either limited in scope or the researchers had difficulty maintaining the pathogen [1, 7, 23, 24]. Wilding [23] was unable to infect hosts of different taxa and concluded that *E. muscae* 'exists in strains adapted to specific hosts.' We found, to the contrary, that there was no sharply defined correlation between susceptibility to infection and the taxonomic affinities of the flies tested. Species from phylogenetically and ecologically separated families such as the Culicidae, Drosophilidae, Muscidae, Sarcophagidae, Anthomyiidae, and Calliphoridae were all susceptible to some extent to this isolate. In contrast, some closely related hosts such as *M. domestica* and *S. calcitrans* (Muscidae), or *P. sericata* and *P. regina* (Calliphoridae), differed greatly in susceptibility. These results indicate that susceptibility is not related to the pathogen being adapted to closely related hosts per se.

Observations made during this study strongly sug-

Fig. 7. D. melanogaster infected with *E. muscae*. Note typical post-mortem fungal growth of conidiophores (c).

Fig. 8. A. aegypti infected with *E. muscae*. Note atypical post-mortem growth of fungus. The legs were not outspread, no holdfasts were produced from the proboscis, few conidia were produced, wings were not raised, four thin intersegmental bands of conidiophores (c) are visible.

Fig. 9. Scale from abdomen of *A. aegypti* with a germinating secondary conidium of *E. muscae* attached. Such scales were seen in large numbers. No germ tubes from such conidia were ever observed penetrating the mosquito scale. This suggests that these scales play a role in protecting *A. aegypti* from infection by *E. muscae*.

Fig. 10. Two specimens of *S. calcitrans* infected with *E. muscae*. Note the atypical syndome: legs not outspread, no holdfasts produced from proboscis, wings not raised. The specimen on the right procuced conidiophores (c) while the specimen on the left did not. The left specimen contained abnormal, highly vacuolated hyphal bodies (see Fig. 11).

Fig. 11. Abnormal hyphal bodies from *S. calcitrans* (Fig. 10 on left) that died of the mycosis caused by *E. muscae* but did not produce conidiophores or conidia. Note the extremely large vacuoles and compare these hyphal bodies to the normal ones pictured in Fig. 12.

Fig. 12. Hyphal bodies of *E. muscae* produced in *M. domestica*. Note the absence of large vacuoles. These hyphal bodies were in the process of elongating to produce conidiophores.

gest that the morphology of the host integument and the interior biochemical milieu of potential hosts were important factors in susceptibility. The integument has been called a primary barrier to infection [8, 17, 20]. Anti-fungal lipids on the integument are one factor that affect insect resistance to pathogens [13, 21]. David [8] stated that fungi penetrate more readily through the thinner, softer, regions of the integument and that when insect species are compared it is found that those with thin cuticles are more readily invaded than those with thick. We found that the scales that cover the body and wing veins of *A. aegypti* were a protective barrier against *E. muscae* conidia. This phenomenon may constitute another mechanism by which an insect might thwart an invader. The role cuticular structures like scales and/or exposed membranous areas play in resistance deserves more study.

The abnormal hyphal bodies (Fig. 11) observed in two specimens of *S. calcitrans* indicate a second type of barrier to infection. Even if the fungus is able to penetrate into the hemocoel of the host, it may not find conditions suitable for normal growth. The factors responsible for such abnormalities could involve in various combinations: osmotic pressure, pH, nutrient deficiencies, phagocytes, toxins, or other host factors.

We found that the size of different host species was not correlated with the incubation period. The large *S. haemorrhoidalis* and the medium sized *M. domestica* had seven-day incubation periods, whereas the tiny *A. aegypti* had a 17-day incubation period. On the other hand, Mullens [18] worked with *E. muscae* infecting *M. domestica* and concluded that differences in incubation period in *M. domestica* individuals were due to differences in host size only. Clearly the incubation period is influenced greatly by the species of host.

The characteristics of the post-mortem growth of the fungus in a dipterous victim are important because they indicate whether a particular host species is a suitable substrate for the pathogen. A 'good' host from the viewpoint of *E. muscae* is one that is highly susceptible, becomes attached to the substrate by the proboscis and its legs at the time of death, produces large numbers of conidiophores exteriorly and discharges large numbers of conidia. The attachment of the host by the pathogen in a site leading to the infection of further hosts is one of the factors that makes the fungi in the Entomophthoraceae such successful pathogens in nature [2, 3]. Production of large numbers of conidia is essential to ensure that some conidia contact new hosts. We found that *A. aegypti* and *S. calcitrans* were only slightly susceptible, did not become attached via the legs and proboscis, and did not produce large numbers of conidia. Therefore, these two dipterans would not be suitable hosts for *E. muscae* in nature. While *P. sericata* did become attached, it did not produce large numbers of conidia. Hence, it would not be a particularly suitable host. *D. melanogaster, S. stercoraria,* and *S. haemorrhoidalis* were moderately suitable hosts for the fungus. *M. domestica* was the best host species tested because it was highly susceptible, attached to the substrate, and produced large numbers of conidia.

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