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PATHOGENESIS ASSOCIATED WITH PHILORNID MYIASIS (DIPTERA: MUSCIDAE) ON NESTLING PEARLY-EYED THRASHERS (AVES: MIMIDAE) IN THE LUQUILLO RAIN FOREST, PUERTO RICO

Leslie S. Uhazy¹ and Wayne J. Arendt²

ABSTRACT: Pathogenesis of myiasis due to the muscid fly *Philornis deceptiveus* in nestling pearly-eyed thrashers (*Margarops fuscatus*) in the Luquillo Rain Forest, Puerto Rico was investigated. Philornid larvae penetrated the host integument, underwent a period of development and growth, and established, as third instar larvae, between the dermis and the body musculature. Movement into this location plus growth and development of the fly larva appeared to be linked to the ingestion of host tissues including red blood cells, mononuclear cells which infiltrated from focal accumulations adjacent to the lesion, and necrotic cellular debris which accumulated in the lesion. The resultant increase in size of the larvae greatly displaced the host integument. Following the evacuation of the larvae for the purpose of pupation, repair of the cavernous lesion was initiated with the production of an intense organized fibrinous exudate. Macrophages and plasma cells predominated with vascular congestion in surrounding tissues. Over the 21-day nest period, nestlings were subject to successive infestations of large numbers of larvae and host responses to these appeared to significantly debit an energy budget responsible for nestling development and growth. Nestling mortality and post fledging survivorship appeared linked to the impact of these energy demands.

INTRODUCTION

Avian myiasis, or the infestation of live nestling and adult birds by dipterous fly larvae of three families (Calliphoridae, Muscidae and Neottiophilidae), is a cosmopolitan phenomenon (Hicks, 1959, 1962; Zumpt, 1965). Zumpt's review of the extensive Old World literature indicates that although there are numerous records of nestling disfigurement and mortality due to what he classified as sanguinivorous or subdermal dipterous larvae, remarkably little is known of their pathogenesis, impacts on host growth and development, and eventual impact on avian populations.

In the Neotropics, 33 species of a unique muscid genus *Philornis* Meinert, 1889 (= *Neomusca* after Malloch, 1921; Bezzi, 1922; Aldrich, 1923) are reported ectoparasites of an as yet undetermined num-

ber of avian species (Pont, 1972). The taxonomy of this group is particularly difficult and the reports of *Philornis* sp. from nestling Aplomado falcons (*Falco femoralis*) (Hector, 1982) plus *Mydaea* (= *Philornis* after Bezzi, 1922) from nestling purplish-backed jays (*Cyanocorax beecheii*) (Winterstein and Raitt, 1983) support this contention. Specific determinations are generally based on adult flies reared from larvae and puparia removed from nestling birds or nesting materials, respectively. In this regard, collections from Trinidad where 10 species of *Philornis* are reported associated with 29 species of avian hosts, suggest the flies are not extremely host specific (Dodge and Aitken, 1968).

Our knowledge of philornid biology and host-parasite relationships is generally poor; however, outstanding in this respect is Nielsen's (1911) investigation of *P. torquans* (Nielsen) (syn. *Mydaea anomala* after Dodge and Aitken, 1968) from an Argentinian finch, *Spermophila gutturalis* and several other species of birds. He described the three larval instars found on nestling and adult birds and "tumors" containing an infiltrate of leucocytes as-

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sociated with the apparent blood sucking activities of these subcutaneous larvae. He further reported that the occurrence of the larvae was not rare and that infested nestlings were killed by the maggots. The impact of this mortality factor on the host population was not investigated.

Recently, based on long term study of some complex ecological interactions involving Hymenoptera, Diptera, nestling chestnut-headed oropendolas (*Psarocolius wagleri*) and yellow-rump caciques (*Cacicus cela*) in Panama, Smith (1968, 1979, 1980) concluded that vertebrate predation was important to these bird populations but philornid myiasis, by an undetermined species of fly, was the "chief source of mortality." Subsequently, in a 4-yr study of the nestling biology of the pearly-eyed thrasher (*Margarops fuscatus*) in the Luquillo Experimental Forest of eastern Puerto Rico, Arendt (1985b) reported that 97% of nestling mortality was due to ectoparasitism by larval *Philornis deceptivus* Dodge and Aitken, 1968 (identified by M. S. Couri of the National Museum of Rio de Janeiro, Brazil). Arendt (1985a) further documented the negative impact of these larvae on nestling growth, development, and fledging success.

Herein, we report on the pathogenesis associated with the presence of larvae of *P. deceptivus* on nestling pearly-eyed thrashers from the Luquillo Forest.

MATERIALS AND METHODS

Eighteen moribund nestling pearly-eyed thrashers were collected in the field and preserved in methanol or isopropyl alcohol. Upon their arrival in Columbia, Missouri, these fixatives were replaced with 10% neutral buffered formalin. Tissues for histopathological examination were dissected from the birds, embedded in paraplast, sectioned at 7 μ m and stained with hematoxylin and eosin. Larval dimensions were determined on philornid larvae which had withdrawn from nestlings upon their placement into the original fixative. Studies of the morphology of larvae were made on specimens stored in 10% glycerol in 70% ethyl alcohol and

subsequently infiltrated with glycerol following evaporation of the alcohol.

RESULTS

As illustrated in Figures 1 and 2, nestling pearly-eyed thrashers 9 and 21 days old, respectively, were disfigured grotesquely by the presence of larval *P. deceptivus*. Both birds were found dead in their nests and based on daily observations it was determined that the younger bird had been exposed to a total of 64 larvae and the older 77. Their usual smooth body contours were distorted by raised elongate lesions (up to 0.5 cm in height and 1.5 cm in length) associated with partially or completely subintegumental larvae, in addition to open cavities (up to 3 mm in diameter) which remained once larvae withdrew from the bird to pupate. Evidence of lesion repair was also observed. The posterior spiracles on the philornid larvae were observed readily within the integumental openings. It was evident in most cases, that penetration sites were in the apteria adjacent to the pterylae and that larvae extended under the developing feather tracts. On the younger nestling, larvae were distributed primarily over the head and dorsal surfaces; while on the older, they were on the ventral surfaces and at the distal end of the tibiotarsus.

Larvae of *P. deceptivus* were off-white to dark brown in color (Figs. 1, 3). They consisted of 12 segments and ranged in total length from 4 to 18 mm with a maximum width ranging from 1.3 to 6.0 mm ($n = 44$). The largest of these, third instar, had an average length and width of 14.8 ± 1.8 mm and 4.1 ± 0.7 mm, respectively ($n = 36$). Maggot-like in general appearance, the anterior end tapered to a rounded head segment and the much broader posterior end was obliquely truncate. In lateral view, the ventral surface was marked by a bulbous greater curvature and a prominent raised anal opening on the anterior margin of the 12th segment



FIGURE 1. Nine-day-old pearly-eyed thrasher nestling infested with larvae of *Philornis deceptiveus*. Preserved, $\times 0.8$.

(Fig. 3). Paired wart-like tubercles were present on the ventrolateral margins of segments 5 through 11. The paired pointed mouth hooks were readily observed (Fig. 4), were bordered ventrally by up to six large cuticular spines, and associated with a distinct cephalopharyngeal skeleton. Antennae and maxillary palps were situated lateral to the mouth hooks. The paired anterior spiracles were situated at the posterior margin of the 2nd segment; while, the paired posterior spiracles were flush with the cuticular surface of the 12th segment. The latter consisted of three serpentine slits and a stout peritreme (Fig. 5).

With the exception of the head, sharply pointed cuticular spines (Figs. 4, 6) were present on the maggots' thoracic and abdominal segments. The anterior margins of segments 2 through 11 were ringed by short arched rows of spines which de-

creased in number and size from anterior to posterior. On the ventral surfaces of segments 5 through 11, discrete raised spiniferous pads bordered posteriorly by small arched groups of spines were present. Just anterior to the spiniferous pads, i.e., on the posterior margins of segments 4 through 10, short arched rows of spines were present. The posterior third of segment 11 and all the terminal 12th were covered by numerous arched rows of small spines. Generally, the spines were positioned with their points directed toward the posterior end of the larva; however, those on the posterior margins of the ventral surface and some on the spiniferous pads were directed towards the maggots' anterior end.

Representative specimens of infested nestlings, *M. fuscatus*, and third instar larvae of *P. deceptiveus* have been deposited in the U.S. National Parasite Collec-



FIGURE 2. Twenty-one-day-old pearly-eyed thrasher nestling infested with larvae of *Philornis deceptionis*. Preserved, $\times 0.8$. Arrows indicate larvae and open integumental lesions.

tion, Beltsville, Maryland 20705 (USNM Helm. Coll. No. 78943).

Microscopically, the raised margins of the open integumental lesions were characterized by sloughing of the keratin layers, epidermal hyperplasia, some evidence of a mononuclear infiltrate and the absence of dermal melanocytes. The long axis of the larvae traversed the thin tissue layers of the integument and its anterior end was positioned between the body musculature and the dermal tissues (Fig. 7). The maggot had essentially burrowed into a subdermal position (subcutis after Lucas and Stettenheim, 1972) with the posterior spiracles exposed to the atmosphere. There was no evidence of involvement with the body musculature. The normal architecture of these loose subintegumental connective tissues containing blood vessels, smooth muscles associated with the feather follicles, and striated cutaneous muscles

was greatly impacted by the presence of the larvae. A cavernous zone lined by mononuclear cells and basophilic necrotic tissues was associated with the anterior end of the maggot. This zone adjacent to the mouthparts of the larva contained a great deal of basophilic cellular debris. It was also evident that portions of the spinous cuticle molted during philornid development remained in this subdermal location and were involved in the host response. The connective tissues around the area of necrosis were edematous, there was little evidence of hemorrhage and few red blood cells were observed in the vessels of the circulatory system. In contrast, more red blood cells, plus mononuclear cells and cellular debris, were observed within the peritrophic membrane of the philornid alimentary tract (Fig. 8). Bordering these necrotic tissues, within the more fibrous connective tissues of the dermis there was



FIGURE 3. Lateral views of two third instar larval stages of *Philornis deceptiveus*. Infiltrated with glycerol, $\times 9$.

evidence of an intense host response indicated by the presence of many prominent focal accumulations of mononuclear cells (Fig. 7). It was apparent that cells from these foci were infiltrating into the necrotic margins of the cavernous lesion. A few thrombocytes were observed and surprisingly macrophages or giant cell formation was not observed in this location or within the cellular debris.

Unlike the area around the anterior end of the maggot, the lesion associated with the stouter posterior end was characterized by compaction of the dermal and subdermal connective tissues. A narrow border of basophilic necrotic tissue, cellular debris and fibrinous exudate lined its circumference. Although fixation produced shrinkage in the larvae, it was apparent from indentations in these tissues that larval cuticular spines were embedded in the margins of the lesion. There

was no evidence of focal accumulations of mononuclear cells in the surrounding connective tissues.

The basophilic necrotic tissues associated with the anterior end of the maggot extended as tracts into the dermal and subdermal tissues (Fig. 9). Since multiple larval infestations predominated, it was evident that large areas of subintegumental tissues were involved. At the confluence of lesions an organized fibrinous exudate containing mononuclear cells, macrophages, thrombocytes and a few plasma cells was observed.

Once a philornid larva had withdrawn from its developmental site (Fig. 10), there was evidence of wound contraction and the vacated cavity was infiltrated by massive numbers of mononuclear cells and an extensive fibrinous exudate organized by connective tissues originating from the dermis and subdermis (Fig. 11). The in-

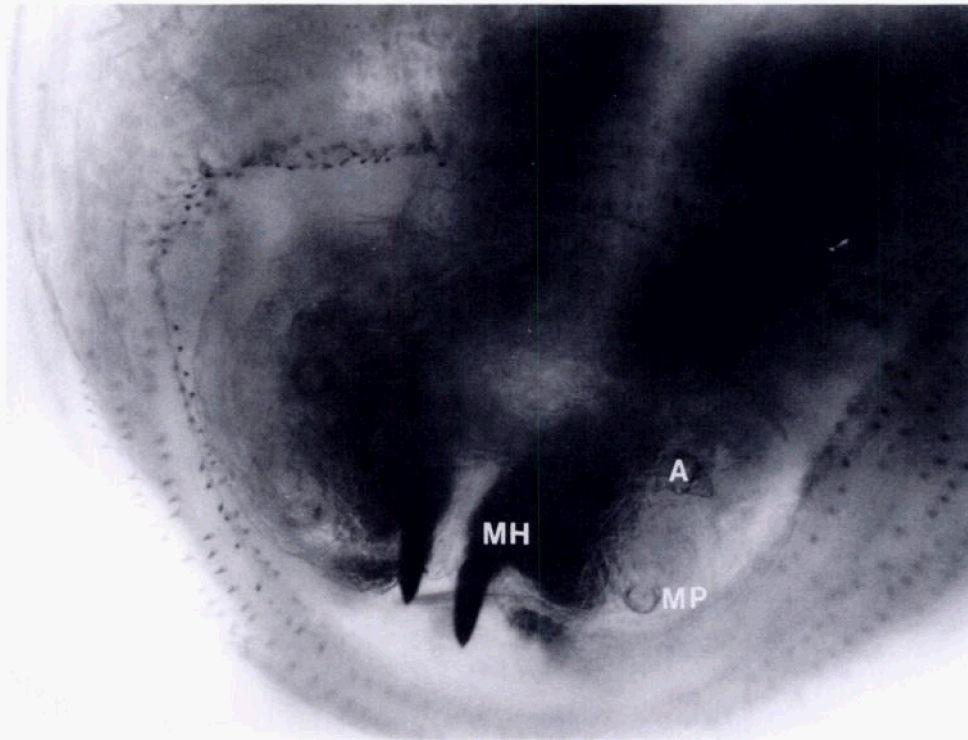


FIGURE 4. Anterior end of the third instar larval stage of *Philornis deceptiveus* showing the mouth hooks (MH), antennae (A), and maxillary palps (MP). Infiltrated with glycerol, $\times 11$.

tegumental opening was covered by a scab while the central component of the lesion consisted of necrotic tissues and macrophages. Tissues on the periphery of this healing lesion were inundated by mononuclear cells and there was evidence of vascular congestion by red blood cells. This contrasted the absence of red blood cells in the more acute lesion. It was also evident that these functions of repair involved extensive regions of the nestlings' subintegumental tissues.

DISCUSSION

Mortality in nestling pearly-eyed thrashers of the Luquillo Experimental Forest in eastern Puerto Rico is primarily due to myiasis by larvae of the muscid fly *Philornis deceptiveus* (Arendt, 1985a). Although the host specificity of this Puerto Rican fly is still under investigation, Pe-

rez-Rivera (1981) reported that some 30 species of Puerto Rican birds are parasitized by *Philornis* spp. In addition, six avian species serve as its host on Trinidad (Dodge and Aitken, 1968). Arendt (1985b) concluded that the reproductive cycle of the fly is highly adapted to the nesting biology of the pearly-eyed thrasher because of the thrasher's abundance in the Luquillo Rain Forest. He described the occurrence of this obligate parasite over four reproductive periods and found 31% of adult birds were infested with a mean intensity of 3.1 larvae per infested bird (range one to seven) while, remarkably, 96% of 448 nestlings harbored a mean intensity of 37 larvae per bird. As few as three and as many as 220 larvae were observed on individual nestlings over their approximate 21-day nestling period. He determined that larval philornid devel-

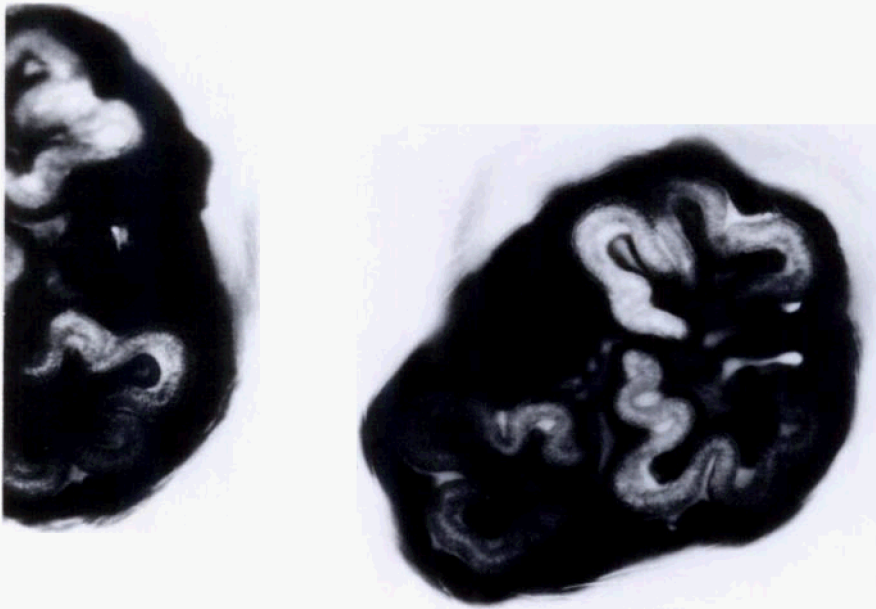


FIGURE 5. Posterior spiracles of the third instar larval stage of *Philornis deceptiveus*. Infiltrated with glycerol, $\times 150$.

opment required about 5 to 7 days, before evacuation from the chick for the purpose of pupation, and that successive infestations were inflicted upon the vulnerable nestlings. During the 4-yr period, 209 of 448 (47%) thrasher nestlings apparently died due to philornid myiasis.

Based on analysis of five growth characters, Arendt (1985a) found that infested nestlings were significantly underweight, showed retarded tarsus growth and delayed emergence of the ninth primary pinfeather. In analyzing these three parameters with respect to intensity of infestation and nestling chronology, he found discrete differences in nestling growth patterns. Generally, nestlings infested with moderate (31–60) and high numbers (>60) of larvae were most affected. However, nestlings infested with lesser burdens (one to 30) during the first

week of life were also developmentally retarded. Interestingly, if a nestling acquired a low intensity after the first week and a half of development, its chances for survival “did not appear substantially jeopardized.” Over the 4 yr, 53.3% (range 44.2–64.4%) of nestlings successfully fledged; however, follow-up field observations indicated that survival of post-fledge juveniles was also influenced by experience with ectoparasitism while in the nest. Based on subsequent mortalities or losses to vertebrate predation, Arendt (1985b) determined that the rate of first year fledgling survival would drop to 20.3%.

As indicated, our knowledge of philornid biology is limited; however, a review of taxonomic studies reveals their close association with nesting birds. Dependent upon the species of *Philornis* in question,

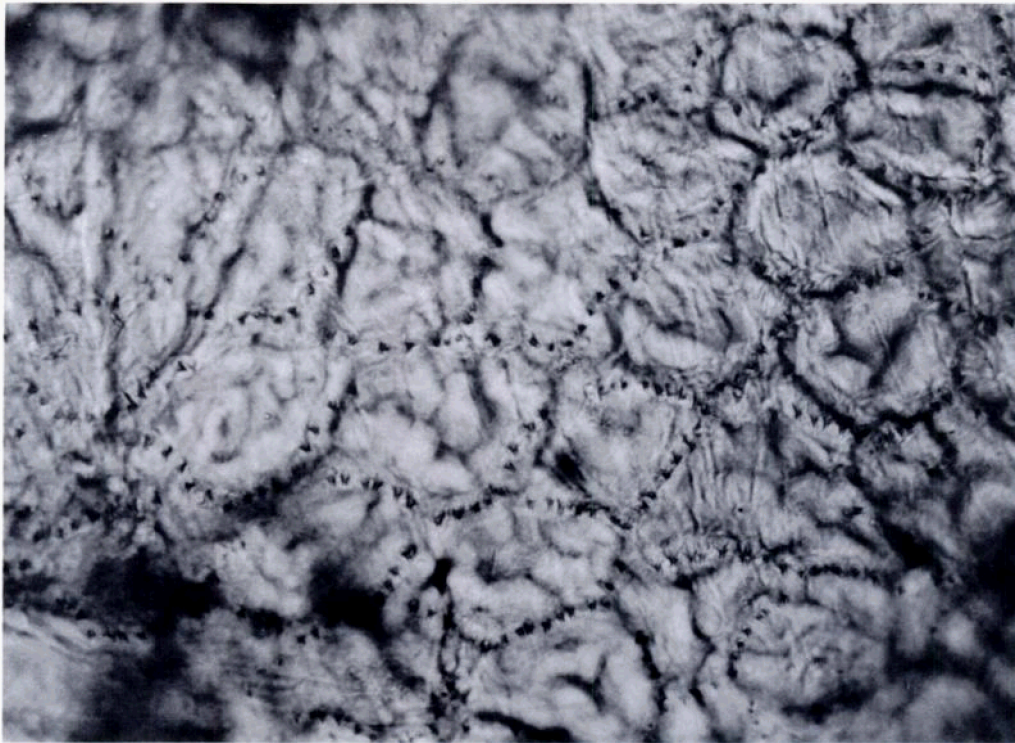


FIGURE 6. Cuticular spination on the third instar larval stage of *Philornis deceptiveus*. Infiltrated with glycerol, $\times 220$.

larval development can occur in nesting materials, on the nestlings or in bird feces. Pupation takes place in the nesting materials (Dodge, 1963; Dodge and Aitken, 1968a, b). Larvae developing in feces are coprophagous scavengers while those in nesting materials apparently feed on the nestlings and return to the nest. With respect to those species which develop in a subintegumental location, it is not clear how they gain access to their hosts. Nielsen (1911) never observed fly eggs on the birds integument, and presumed that adult *P. torquans* deposited first instar larvae directly onto the skin of the bird. We observed fly eggs on nestlings dissected for histological purposes; however, they could not be specifically attributed to *P. deceptiveus*. In considering specific locations of fly larvae on the nestlings with nestling ontogeny, Arendt (1985b) found a signif-

icant correlation between age of nestlings and sites of larval infestation. Young, somewhat nude and immobile nestlings generally had larvae on their head, around the mouth and dorsal surface of the trunk. In contrast, older nestlings with more fully developed feathers and able to stand and move around the nest had more larvae on the ventral surfaces and legs. Circumstantially, the observation of eggs and this change in site specificity suggests adult *P. deceptiveus* exploit readily accessible integumental sites and oviposit directly on the nestlings. Although larvae could hatch within the nesting materials and creep onto the nestling, it is difficult to conceive of random movements of larvae resulting in this pattern of infestation. Arendt (1985b) further indicated that later in the nesting period when populations of both fly and host were the most dense, site

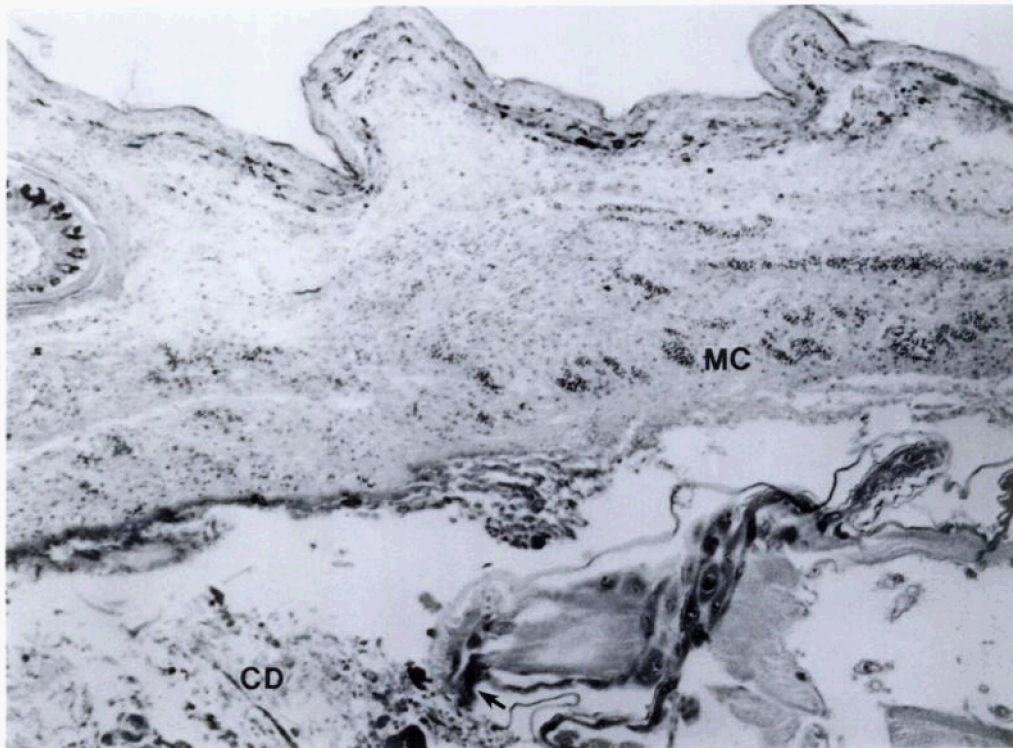


FIGURE 7. Subdermal cavernous lesion, associated with a larval *Philornis deceptiveus*, containing cellular debris (CD), lined by basophilic necrotic tissues, and bordered by focal accumulations of mononuclear cells (MC) associated with a larval *Philornis deceptiveus* in a nestling pearly-eyed thrasher. Arrow indicates a larval mouth hook. H&E, $\times 160$.

specificity disappeared and larvae were observed wherever space was available.

Regardless of how the larvae got onto the host, this study and that of Nielsen (1911) indicate that pathogenesis associated with the presence of ectoparasitic philornid larvae was directly linked to larval feeding activities and their consequent growth and development through three instars. Clearly the heavily spined maggots are adapted to the subintegumental environment. Nielsen found that the first instar penetrated the hosts integument and bored its way, with the aid of sharply toothed broad mouth hooks, into a subintegumental position forming a "tumor." Once in this position, the larva increased in size and molted twice, to the third instar. Accompanying the molts there were changes in the posterior spiracles, cuticu-

lar spination, and morphology of the internal cephalopharyngeal skeleton and mouth hooks. With the exception of the posterior spiracular pattern, the general morphology of the third instar larva of *P. deceptiveus* was similar to the larva described by Nielsen (1911). In addition to large size, we observed the extensive cuticular spination with the majority of spines directed posteriorly, ventral spiniferous pads and slender pointed mouth hooks. Nielsen indicated the mouth hooks were used to irritate host tissues and further stated, "the food of the parasite consists of the blood of the tissues along the inner surface of the tumor."

Nielsen's use of the term "tumor" implies proliferative changes in integumental and subintegumental connective tissues in proximity to the philornid larvae.

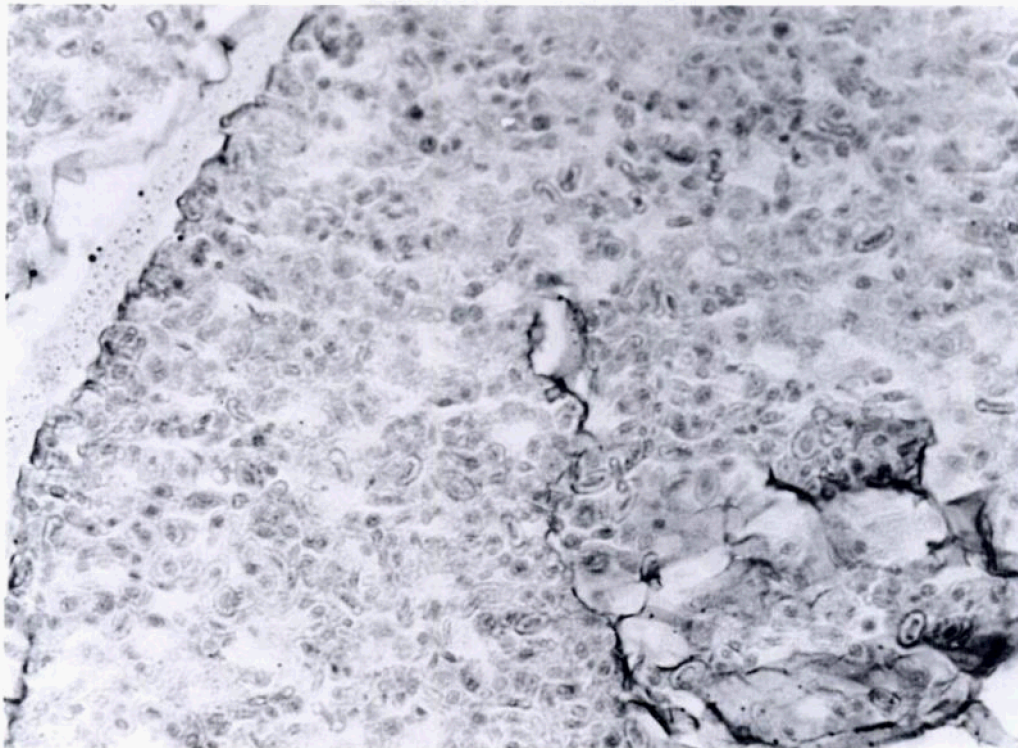


FIGURE 8. Red blood cells, mononuclear cells and cellular debris within the peritrophic membrane of the alimentary tract of a larval *Philornis deceptiveus*. H&E, $\times 610$.

We observed that marked displacement of the integument and compaction of the subintegumental connective tissues, into what grossly appear as tumors, was a consequence of the width of the posterior two-thirds of the larva and their absence from the body musculature. Averaging 4.1 mm in width, philornid larvae were tightly enveloped by host tissues. Other than in instances of tissue repair, epidermal hyperplasia associated with the integumental opening and the occurrence of prominent foci of mononuclear cells were the only proliferative changes we observed.

As indicated by Nielsen (1911), the subintegumental larvae feed on blood and accompanying tissue fluids. But, in addition to red blood cells, we also observed mononuclear cells and basophilic cellular debris within the peritrophic membrane of third instar larvae. Unlike its posterior end, the anterior portions of the larva including the

cephalic segments were positioned in a cavernous space containing tissue fluids and basophilic cellular debris. The cavity was lined by necrotic tissues containing mononuclear cells which may have infiltrated into these tissues from the focal accumulations in the surrounding connective tissues. Presence of the cavity suggested that it is formed as a result of larval movements and lacerating actions of their mouth hooks. Salivary secretions of larvae containing anticoagulants were probably involved in these feeding activities and the presence of tracts of necrotic tissue extending into the subintegumental connective tissues suggested the presence of histolytic enzymes. Although first and second instar larvae were not studied, it seems clear that these actions facilitated the movement of the larvae into the deeper tissues and continued larval feeding.

Nielsen indicated that "no trace of mat-

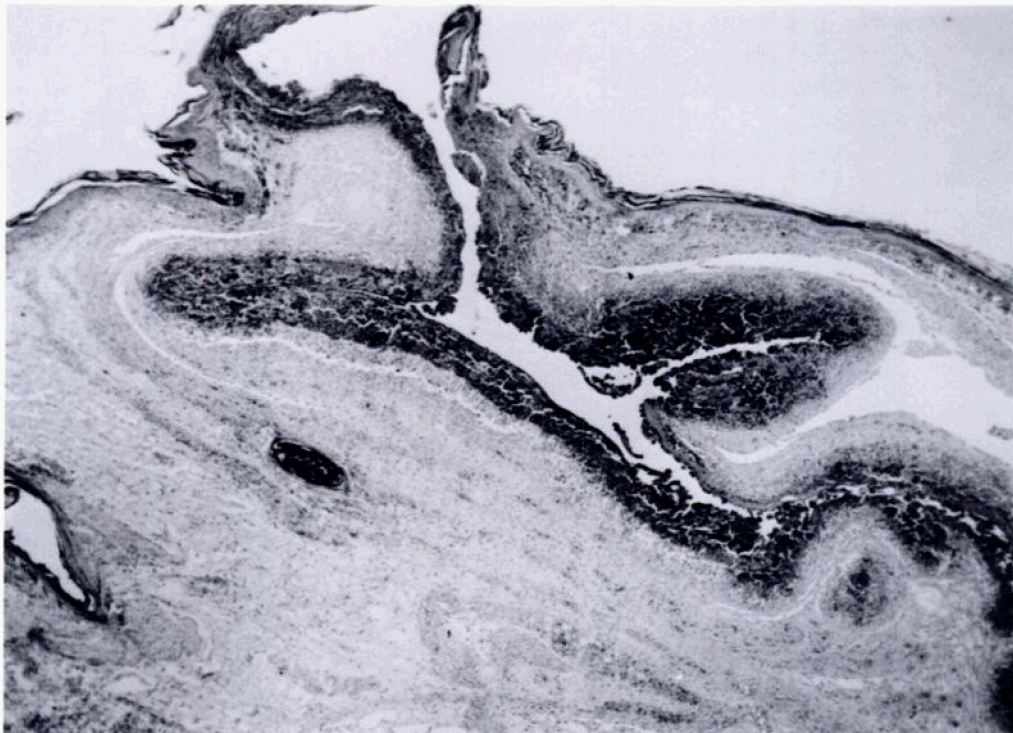
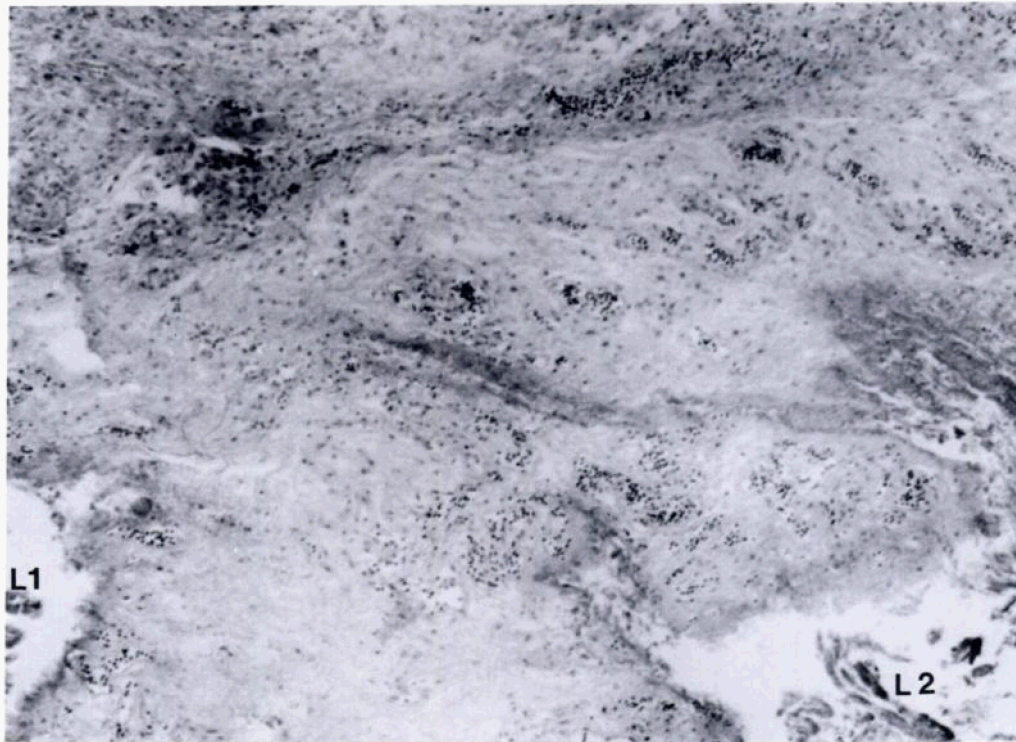
ter is seen in the tumor and also the excrements and cast larval skins of the parasite are removed from it." Contrasting this statement, we observed portions of the molted spinous cuticle within some lesions. In one instance it was involved with an extensive infiltration of plasma cells and a fibrinous exudate. Since the posterior half of the larva was tightly bound by host tissues it is hard to visualize how portions of the larval cuticle could get out of the lesion. As such, the absence of these materials from other lesions suggests the larvae may utilize them as a food source. With respect to larval excrements, the 12th abdominal segment which houses the anus and the posterior spiracles is exposed to the atmosphere and excretory products could be deposited onto the integument of the bird or remain in the integumental opening. In this latter instance it is possible that bacteria could secondarily enter the lesion. Bacteria were not observed in tissue section and this aspect of pathogenesis was not investigated.

Arendt (1985a, b) has shown that the presence of high densities and successive infestations of larvae of *P. deceptivus* have a direct negative impact on growth, development and survivorship of nestling pearly-eyed thrashers. Further, if nestlings successfully fledge, experience with philornids while in the nest impact on juvenile survival. It is very evident from studies of the pathogenesis that under the above conditions of infestation, a severe burden is placed on the metabolic capabilities of a recently hatched chick. The

pearly-eyed thrasher is an altricial nidicolous bird. Nestlings weigh on average 7 g at hatching and grow to be on average 97 g at fledging, i.e., 97% of adult weight at 21 days post hatch (Arendt, 1985a). Clearly, the loss of red blood cells, subintegumental connective tissues and tissue fluids due to the feeding activities of the philornid larvae are serious debits to an energy budget responsible for nestling growth and development. Of equal importance to this balance sheet is the nestlings' redirection of resources to compensate for these losses, to combat the larvae and to initiate healing once the larvae had evacuated the lesions. The high rate of mortality, in all intensity categories, during the first week and a half of development, when about 75% of fledging weight is achieved, indicates these are costs which can not be afforded (Arendt, 1985a). It is interesting to note that following this initial period of time and if the nestling is exposed to a few larvae (one to 30) the chances of nestling survival are greatly improved. Our observation of plasma cells in the hosts response to the parasites suggest the nestling is developing immunocompetency at this time. This conclusion is further supported by the observation of foci of mononuclear cells in the surrounding tissues. Bell (1974) has shown that experimental injection of antigenic materials into the subintegumental tissues of ducks yields a similar cellular response. Moderate and high intensities of infestation are sufficient to overwhelm this putative immune response.

FIGURE 9. Subdermal tracts of necrotic tissues extending between two lesions (L1, L2) associated with larval *Philornis deceptivus* on the pearly-eyed thrasher. H&E, $\times 170$.

FIGURE 10. Open lesion in the integument of a nestling pearly-eyed thrasher following the withdrawal of a larval *Philornis deceptivus*. Note the intensely organized host response in the margins of the lesion. H&E, $\times 50$.



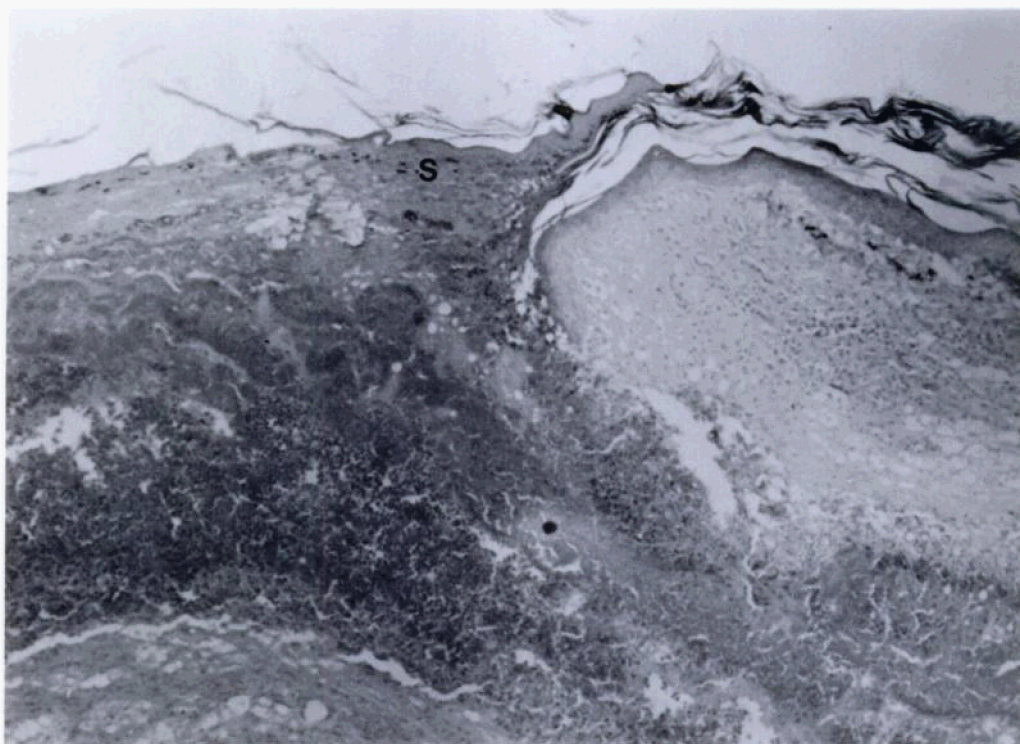


FIGURE 11. Repair of an integumental lesion in a nestling pearly-eyed thrasher in the absence of a larval *Philornis deceptiveus*. Note the covering scab (S) and the intensely organized fibrinous exudate filling the cavity. H&E, $\times 155$.

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