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Author: Warren, Yvonne

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Protocalliphora braueri (Diptera: Calliphoridae) Induced Pathogenesis in a Brood of Marsh Wren (*Cistothorus palustris*) Young

Yvonne Warren, Department of Biology, Utah State University, Logan, Utah 84322-5305, USA

ABSTRACT: Infestation with blow fly larvae (*Protocalliphora* (*Trypocalliphora*) *braueri* Hendel) was pathogenic to marsh wren (*Cistothorus palustris*) young. The mechanism of pathogenicity was *Pseudomonas* spp. infection of subdermal myiasis-induced lesions and subsequent sepsis. Neither internal organ involvement nor muscle destruction was seen on necropsy of fledglings. Multifocal hepatic necrosis was seen histologically and *Pseudomonas* sp. was isolated from myiasis sites, liver, and peritoneal cavities.

Key words: Pathogenicity, blow fly larvae, *Protocalliphora* (*Trypocalliphora*) *braueri*, *Pseudomonas*, marsh wren, *Cistothorus palustris*, histopathology, myiasis.

Larvae of *Protocalliphora* species (Diptera: Calliphoridae) are obligate hematophagous parasites of nestling and fledgling birds. Invasion of the host body to feed on blood, defined as sanguinivorous myiasis (Zumpt, 1965), occurs when larvae form a wound by rasping and expanding a hole in skin to induce blood flow (Bennett and Whitworth, 1991). Larvae of almost all *Protocalliphora* species live in nesting material and feed intermittently on their hosts (Rogers et al., 1991). *Protocalliphora* (*Trypocalliphora*) *braueri* Hendel are unusual in that they can produce a subdermal myiasis by burrowing into subcutaneous tissue and remain embedded there throughout larval development (Sabrosky et al., 1989).

Observations of nestling mortality associated with *P. braueri* infestation (Sabrosky et al., 1989) have prompted recent studies directed at determining if these larvae are pathogenic to their hosts; they appear to have no deleterious effects on mass, fledging success, or hematological parameters of sage thrashers (*Oreoscoptes montanus*) and house wrens (*Troglodytes aedon*) (Eastman et al., 1989; Howe, 1992). Theoretically, *Protocalliphora* might directly transmit pathogens to their hosts or increase host susceptibility to secondary infections (Leclercq, 1969) but few data are available to support or refute these hypotheses. Although Rausch (1972) reported the gross pathology and epidermal histopathology for a *P. braueri* infested Wilson's warbler (*Wilsonia pusilla*), there generally are few published descriptions of the lesions of larval infestation resulting in host mortality; adverse effects on hosts are assumed to be the result of extensive tissue destruction or exsanguination (Sabrosky et al., 1989). Here I describe the structural and functional manifestations of disease of a brood of marsh wren (*Cistothorus palustris*) young infested with *P. braueri* larvae and present evidence that *P. braueri* infestation increased susceptibility to a bacterial infection that resulted in host mortality.

In conjunction with a study to determine the pathogenicity of *Protocalliphora interrupta* (Whitworth), marsh wren nests were located in the freshwater marshes of the Bear River in Cache County, Utah (USA, 41°53′N, 111°55′W). Young wrens were assessed daily for 8 days after hatching to determine mass, ectoparasite load, and morphological development. At day 8 post-hatch all nestlings were banded with a United States Fish and Wildlife aluminum band and a unique color band combination; 20 μl blood samples were collected by the technique of Jain (1986) in heparinized microhematocrit tubes, chilled for transportation, then centrifuged for 5 min at 11,500 rpm in a microhematocrit centrifuge (International Equipment Company, Needham Heights, Massachusetts, USA). Microhematocrit (packed cell

volume expressed as a percentage of total blood volume) was determined with an International Equipment Company microcapillary tube reader with an accuracy, according to the manufacturer, of ±1%.

At day 10 post-hatch the young wrens of this population often leave the brood nest for short periods of time, climb about in vegetation, and attempt to fly across small spans of water while soliciting their parents for food (Warren, unpubl.). The number of young present on parental territories, either in the nest or fledged, was monitored until day 14 post-hatch. Of 117 broods examined in the years 1990 to 1992, one brood of four young was found to be parasitized by *P. braueri* at day 11 post-hatch. At day 12 post-hatch, 10 μl blood samples were taken from all four young to determine post-infestation hematocrit. The first young wren was found dead on day 13 post-hatch; on day 14 post-hatch, the remaining three young were found dead in the brood nest.

At necropsy all four young had perforations of the skin on the crown, neck, prodorsal region, wrist, and forearm. A thick dark brown and green malodorous exudate was present in the subcutaneous areas around the perforations. One to six fly larvae were associated with each perforation with a mean (±SE) of 12.5 (±2.5) *P. braueri* larvae per nestling. Larvae collected from the young were reared in individual jars of sawdust; adults that emerged identified to genus and species using keys in Sabrosky et al. (1989). Representative specimens were deposited in the Entomological Collection of Utah State University, Logan, Utah; no accession numbers are available, but researchers may borrow specimens. No internal organ or muscle damage was seen. The proventriculus was full in three of the four wrens, indicating recent ingestion. Peritoneum, myiasis lesions, and livers of all four birds were cultured aerobically at 37 C on sheep blood agar and MacConkey agar (Oxoid Australia, Hurstville, New South Wales, Australia): This yielded a heavy growth of *Pseudomonas* sp., identified by the techniques of Strafuss (1988).

The degree of post-mortem autolysis of three birds prohibited histopathological examination. Tissue samples of trachea, lung, esophagus, pancreas, intestine, proventriculus, heart, brain, skin, and liver from one bird were fixed in 10% buffered formalin, embedded in paraffin, sectioned, stained with hematoxylin and eosin, and examined by light microscopy. Sections of skin taken from areas of myiasis displayed necrosis, edema, and influx of heterophils and lymphocytes. Sections of liver displayed multiple areas of necrosis and numerous bacteria. There were no significant alterations in sections of trachea, lung, esophagus, pancreas, intestine, proventriculus, heart, or brain. Using a paired one-tailed t-test, the mean (±SE) post-infestation hematocrit (37.4 ± 1.3) differed significantly ($n = 4$, $P = 0.0059$) from the mean hematocrit (46.4 ± 1.1) prior to infestation (Sokal and Rohlf, 1969). The post-infestation value, however, was still within normal range for this species; mean (±SE) hematocrit of four non-infested day 12 post-hatch young evaluated the same day was 38.3 ± 5.8 (Warren, unpubl.).

Skin lesions at sites of myiasis were compatible with the findings of Rausch (1972) and included edema and focal aggregation of lymphocytes. Unlike Rausch's findings, no organs were physically affected by the larvae and invasion did not affect the young wrens' ability to leave the nest or feed. Were it not for the *Pseudomonas* sp. infection at the myiasis sites, subcutaneous tissue damage might have healed after larval abandonment.

Decomposing plant material is ubiquitous in a marsh habitat, with *Pseudomonas* spp. probably a prominent member of the bacterial community. *Pseudomonas* spp. are pathogenic to guinea pigs, rats, and pigeons, producing death within 24 to 36 hr following intraperitoneal injection (Soltys, 1963). Though all young of this wren population were vulnerable to the bacterial community, those with multiple my-

iasis-induced lesions available for bacterial colonization would be most susceptible to infection. *Pseudomonas* sp. infection of myiasis-induced lesions most likely occurred when these young left the nest to climb about in the vegetation and entered the water. Infection of larval entry sites presumably was a frequent occurrence and the resulting pathogenicity may vary. More studies, with larger sample sizes and inclusion of culture of myiasis-induced lesions and necropsy, will contribute to the understanding of this parasite's role in nestling and fledgling survivorship.

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LITERATURE CITED

BENNETT, G. F., AND T. L. WHITWORTH. 1991. Studies on the life history of some species of *Protocalliphora* (Diptera: Calliphoridae). Canadian Journal of Zoology 69: 2048–2058.

EASTMAN, M. D., L. S. JOHNSON, AND L. H. KERMOTT. 1989. Ectoparasitism of nestling house wrens *Troglodytes aedon*, by larvae of the blow fly *Protocalliphora braueri* (Diptera: Calliphoridae). Canadian Journal of Zoology 67: 2358–2362.

HOWE, F. P. 1992. Effects of *Protocalliphora braueri* (Diptera: Calliphoridae) parasitism and inclement weather on nestling sage thrashers. Journal of Wildlife Diseases 28: 141–143.

JAIN, N. C. 1986. Schalm's veterinary hematology, 4th ed. Lea and Febiger, Philadelphia, Pennsylvania, 1221 pp.

LECLERCQ, M. 1969. Entomological parasitology: The relations between entomology and the medical sciences. Pergamon Press, Oxford, England, 158 pp.

RAUSCH, R. L. 1972. Cutaneous myiasis in a bird by the larval *Protocalliphora h. hirudo* Shannon and Dobroscky, 1924 (Diptera: Calliphoridae). Aquilo, Series Zoologica 13: 1–4.

ROGERS, A. R., R. J. ROBERTSON, AND B. J. STUTCHBURY. 1991. Patterns and effects of parasitism by *Protocalliphora sialia* on tree swallow nestlings. *In* Bird-parasite interactions: Ecology, evolution, and behavior, J. E. Loye and M. Zuk (eds.). Oxford University Press, New York, New York, pp. 123–139.

SABROSKY, C. W., G. F. BENNETT, AND T. L. WHITWORTH. 1989. Bird blow flies (*Protocalliphora*) in North America (Diptera: Calliphoridae), with notes on the Palearctic species. Smithsonian Institute Press, Washington, D.C., 312 pp.

SOKAL, R. R., AND J. ROHLF. 1969. Biometry. W. H. Freeman and Company, San Francisco, California, 776 pp.

SOLTYS, M. A. 1963. Bacteria and fungi pathogenic to man and animals. Williams and Wilkins Company, Baltimore, Maryland, 540 pp.

STRAFUSS, A. C. 1988. Necropsy: Simplified procedures and basic diagnostic methods for practicing veterinarians. Charles C. Thomas, Springfield, Illinois, 244 pp.

ZUMPT, F. 1965. Myiasis in man and animals in the Old World. Butterworth Incorporated, Washington, D.C., 245 pp.

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