

# Not the Usual Suspects: Human Wound Myiasis by Phorids

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**ABSTRACT** Infestation of animal tissues by dipteran larvae (myiasis) commonly occurs in many species, but it is unusual for humans in temperate regions. Nevertheless, human myiasis is regularly observed in many primary care facilities in the United States. Beyond medical issues associated with treating human myiasis, both the causal agent and the longevity of myiasis can have legal implications, for example, as evidence of neglect. Cases of human myiasis in the United States typically involve imported myiasis from torsalo, *Dermatobia hominis* (Linnaeus, Jr.) (Diptera: Oestridae), or facultative myiasis from calliphorids. Here, however, we report two cases of wound myiasis caused by phorid larvae occurred in southeastern Nebraska within 10 mo. Degree-day analysis indicates initial infestation occurred 2 and 3 d before discovery. There are few previous reports of phorid wound myiasis; so, the occurrence of two cases in so short a period suggests that phorids are more important than previously appreciated.

**KEY WORDS** myiasis, Phoridae, medical entomology, forensic entomology

Myiasis is defined as “the infestation of live human and vertebrate animals with dipterous larvae, which, at least for a certain period, feed on the host’s dead or living tissues, liquid body substances, or ingested food” (James 1947). Although flies in the families Oestridae and Calliphoridae are most commonly associated with myiasis, more than a dozen other families of Diptera are known to cause myiasis in humans (Catts and Mullen 2002). Aside from occasional reports of obligatory myiasis in travelers caused by torsalo, *Dermatobia hominis* (Linnaeus, Jr.) (Diptera: Oestridae), the majority of reported human myiasis cases in the United States are facultative wound myiasis caused by calliphorids, or, rarely, by sarcophagids (Sherman 2000).

Phorids (Diptera: Phoridae) are a large family representing >225 genera and ≈3,000 described species worldwide (Disney 1994). The family is represented in North America by ≈356 species and 48 genera (Peterson 1987). Although myiasis caused by phorids has been reported previously, it is most often related to nonwound myiasis such as urogenital (Singh and Rana 1989, Biery et al. 1979), ocular (Wright 1927), and nasopharyngeal (Carpenter and Chastain 1992) myiasis. Relatively few cases of wound myiasis associated with phorids have been reported (Hardey 1951, Sherman 2000, Hira et al. 2004).

Here, we present two similar cases of wound myiasis caused by phorids. Both cases occurred in southeastern Nebraska within a 10-mo period.

## Materials and Methods

**Case 1.** A sample from the York General Hospital was submitted to the University of Nebraska-Lincoln Insect Diagnostic Clinic in August of 2004. The sample, collected from a human leg wound, contained one maggot preserved in isopropyl alcohol and two live puparia in a dry specimen vial. The client stated that maggots were placed in the dry vial at the time of collection and that they must have pupated during transport. The live samples were reared in an environmental chamber (Percival Scientific, Perry, IA) at  $24 \pm 1^\circ\text{C}$  and a photoperiod of 16:8 (L:D) h until adult emergence.

**Case 2.** In June 2005, a patient of St. Elizabeth’s Wound Care Center was diagnosed with wound myiasis. The patient in this case was a 55-yr-old morbidly obese white male with diabetes mellitus type II on chronic renal replacement therapy with long-standing venous stasis ulcers. He had been undergoing weekly dressing changes, although he would sometimes miss his appointments and follow-up 3 d later. He returned 10 d after application of a four-layer compressive dressing, and he was found to have maggots under his dressing. There were no signs of cellulitis. The wounds were redressed with the same dressings: an antimicrobial barrier dressing (Acticoat, Smith & Nephew, Hull, United Kingdom) and a four-layer compression dressing (Profore, Smith & Nephew). Debridements were not performed, because the patient would not allow his wounds to be debrided. The patient’s dressings had been saved, which were heavily infested with hundreds of 3–5.5-mm maggots. Samples of the maggots were taken, placing half into Peterson’s solution (KAAD) (Triplehorn and Johnson 2005). The other

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half were kept alive for rearing purposes. Additionally, one newly formed puparium was collected from the bandages, which also was kept alive for rearing. The live maggots were transferred to raw beef liver held in a foil pouch to allow for continued feeding and development. The live samples were placed in an environmental chamber (Percival Scientific) at  $24 \pm 1^\circ\text{C}$  and a photoperiod of 16:8 (L:D) h until adult emergence.

This was the second occurrence of maggot infestation in this patient. The previous incident was 3 yr earlier, which occurred after the patient failed to follow-up for 3 mo and returned with the same dressing that had been placed originally. The maggots at that time were not sent for identification of species.

### Results and Discussion

The adult flies in the first case were identified as *Megaselia scalaris* Loew by using keys by Disney (1994) and Borgmeier (1964, 1966). The preserved maggot was determined to be a third instar. Degree-day analysis was conducted using thermal requirements published by Greenberg (1991) and assuming normal body temperature ( $37^\circ\text{C}$ ) at the site of development. These data yielded an estimated period of development on the wound between 46 and 71 h. Because the patient in this case is anonymous, treatments that the patient received are unknown.

The adult flies in case 2 also were identified as *M. scalaris* by using keys by Disney (1994) and Borgmeier (1964, 1966). The preserved maggots were again determined to be third instars, although the latest stage of development (and therefore the most accurate indicator of time since colonization) was the newly formed puparium. Degree-day analysis was conducted with the assumption of normal body temperature ( $37^\circ\text{C}$ ), yielding an estimated period of development of  $\approx 72$  h.

To prevent facultative myiasis, wounds should be kept clean and covered, dressings should be changed at least weekly, and entry of flies should be controlled (Steenvoorde and Jukema 2004). The patient in case 2 did keep the wound covered, but he failed to return for his dressing change until a 10-day interval had elapsed. He did not allow debridement of his wounds, but he did allow the wounds to be rinsed with chlorhexidine and rinsed with water before having the dressing reapplied. By report, his living environment was not protected from the entry of flies. He had no blistering that would be a sign of cutaneous myiasis (Goddard 2001), so treatment involved removal of the larvae by removing the dressing and cleansing the wound with chlorhexidine and water to lavage the larvae off the wound. He had no recurrence of myiasis in the intervening time until his death from pulmonary complications >9 mo after this episode.

Among physicians, it is recognized that maggot therapy can be used successfully to treat chronic, long-standing, infected wounds, which have previously failed to respond to conventional treatment. Such wounds are typically characterized by the pres-

ence of necrotic tissue, underlying infection, and poor healing. Maggot therapy uses freshly emerged, sterile blow fly larvae, and it is a form of artificially induced myiasis in a controlled clinical situation (Sherman 2000, 2002; Nigam et al. 2006). The presence of maggots does not confer infection. In fact, Armstrong et al. (2006) found no significant difference in infection prevalence in patients undergoing maggot therapy for diabetic foot wounds versus controls (80 versus 60%). Interestingly, Steenvoorde and Jukema (2004) cultured gram-negative bacteria more often after maggot treatment than before treatment ( $P = 0.001$ ). The opposite effect was found for gram-positive-infected wounds (nonsignificant,  $P = 0.07$ ).

Given an appreciation for the potential therapeutic value of myiasis, it also is important that health care providers recognize injurious forms of myiasis. Beyond the medical implications of myiasis, myiasis also can be an important indicator of wound care neglect, either self-neglect (as illustrated by the cases reported here) or by care givers (Hall and Huntington 2008). In this latter instance, recognizing and reporting myiasis can have important legal implications in cases of criminal neglect.

Although cases of phorid myiasis may not necessarily herald a new threat to wound care patients, the occurrence of two cases in less than a year suggests that such myiasis is not necessarily a rare event. Because so much attention is paid to larger muscoid flies that may cause myiasis, it is likely that the small, unassuming phorid flies buzzing around a wound may go unnoticed or ignored, which may lead to an increase in the occurrence of phorid wound myiasis. Additionally, given the large number of maggots observed in case 2, the potential health implications of phorid myiasis may justify further study. Minimally, patient and doctor education on myiasis should include more information on the diversity of potential causative agents beyond *Dermatobia* and calliphorids.

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