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Physical and physiological costs of ectoparasitic mites on host flight endurance

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Abstract. 1. Dispersal is essential for locating mates, new resources, and to escape unfavourable conditions. Parasitism can impact a host's ability to perform energetically demanding activities such as long-distance flight, with important consequences for gene flow and meta-population dynamics.

2. Ectoparasites, in particular, can adversely affect host flight performance by diminishing flight aerodynamics and/or by inflicting physiological damage while feeding on host tissue.

3. Experimental flight assays were conducted using two fruit fly-mite systems: *Drosophila nigrospiracula* (Patterson and Wheeler) – *Macrocheles subbadius* (Berlese) and *D. hydei* (Sturtevan) – *M. muscaedomesticae* (Scopoli). Flies that are burdened by mites are expected to exhibit lower flight endurance compared to uninfected flies.

4. The results show that the presence of mites (attached) significantly decreased flight endurance by 57% and 78% compared to uninfected *D. nigrospiracula* and *D. hydei*, respectively. The physiological damage caused by *M. subbadius* was revealed through a 53% decline in flight time among previously infected flies (mites removed just prior to flight assay). Surprisingly, the presumably phoretic *M. muscaedomesticae* also caused a 62% reduction in flight endurance among previously infected *D. hydei.*

5. These results suggest a strong deleterious effect of ectoparasitic mites on host flight performance, mediated by a reduction in flight aerodynamics and damage to host physiology. Adverse effects on host flight and/or dispersal may have broad implications for gene flow, population genetic structure, and local adaptation in both host and parasite meta-populations.

Key words. Dispersal, *Drosophila*, host–parasite interaction, infection, insect flight, *Macrocheles*, parasitism.

Introduction

Animal dispersal and long-distance movements are essential for locating mates, breeding sites, food, and for escaping deteriorating environments. Dispersal is defined as the movement from the natal or breeding area to another area (Clobert *et al.,* 2001). Dispersal has important consequences for genetic exchange between local populations, population genetic structure, and species distribution (Slatkin, 1985, 1987; Powell, 1997; Whitlock, 2001). In particular, studies have demonstrated a clear relationship between insect dispersal and local genetic differentiation, i.e. gene flow increases with mobility (Peterson & Denno, 1998). Differences in dispersal capacity among insects have been attributed to a suite of intrinsic and extrinsic factors, including body size, age, lipid content as well as population density, habitat patchiness, and resource availability (Doak, 2000; Coll & Yuval, 2004; also see Palmer & Strathmann, 1981; Levin *et al.,* 1984; Elliott & Evenden, 2009). While studies have examined the role parasites play in the host's decision to disperse as a means of escaping parasitism, relatively few have considered the impact of pathogens and parasites on the capacity to disperse or dispersal distance (Boulinier *et al.,* 2001). For example, monarch butterflies infected by protozoan parasites exhibit lower dispersal distance compared to healthy butterflies, which is attributed to shorter flight distances and slower flight speeds among infected compared to uninfected

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individuals (Bradley & Altizer, 2005). Resource extraction and reduced body condition as a result of parasitism can influence a host's ability to perform energetically demanding activities such as long-distance flights.

Some while other studies have documented a reduced flight performance among infected insect hosts compared to uninfected or lightly infected individuals (Humphry & Linit, 1989; Simmons & Rogers, 1991; Akbulut & Linit, 1999; Villacide & Corley, 2008; Dorhout *et al.,* 2011), few have attempted to separate out the physical and physiological costs of parasitism on host movement. For hosts infected by internal pathogens (e.g. viruses, bacteria, protozoa, or fungi) and parasites (e.g. nematodes, tracheal mites), the costs of parasitism are manifested primarily at the physiological level (Schiefer *et al.,* 1977; Marden & Cobb, 2004). For example, the microsporidian *Nosema pyrausta* reduces flight distance and duration in the European corn borer by depleting the host's energy reserves (Dorhout *et al.,* 2011). For ectoparasites, however, it is not always clear whether the deleterious effects are a result of physiological damage or the physical presence of the ectoparasite itself. Ectoparasites typically feed on host haemolymph or tissue (Reinhardt, 1996), potentially draining the host of nutrients necessary for energetically expensive activities (Abro, 1992); the additional physical burden of having one or more parasites attached to the body's external surface may exacerbate these physiological costs. The aim of this study was to disentangle experimentally two non-mutually exclusive hypotheses for the cost of parasitism on host flight performance, direct (physical) costs, and resource allocation (physiological) costs.

Species of the genus *Drosophila* disperse long distances, ranging from a few metres to several kilometres per day and show a wide range of genetic structures (Powell, 1997). We conducted experimental flight assays to measure flight endurance, a proxy for dispersal ability, using two different host–parasite systems. The first system involved the fruit fly, *Drosophila nigrospiracula* (Drosophilidae, Patterson and Wheeler) and a naturally occurring parasitic mite *Macrocheles subbadius* (Macrochelidae, Berlese). This relatively large mite (600–700 μm) primarily attaches to the abdomen where it feeds on host haemolymph (Fig. 1a) (Polak, 1996). These mites have been shown to have adverse effects on host body condition, survival, and reproduction (Polak & Markow, 1995; Polak, 1996, 1998). The second system involved the fruit fly *Drosophila hydei* (Sturtevan) and a naturally occurring phoretic mite, *Macrocheles muscaedomesticae* (Scopoli; Fig. 1b). *Macrocheles muscaedomesticae* is also fairly large (800–1000 μm) relative to the adult flies and occasionally feeds on fly eggs and first instar larvae, but is thought to be strictly phoretic on adult flies (Filipponi, 1955; Wade & Rodriguez, 1961; Axtell, 1964). Both mite species have been shown to have negative effects on the dispersal of stable flies *Stomoxys calcitrans.* Based on trap data, the authors showed that flies dispersing between farms were less likely to harbour mites compared to flies collected at the farms (Beresford & Sutcliffe, 2009).

In this study, we examined two possible mechanisms by which parasitism can reduce flight endurance. The presence of mites may impose a physical burden on flight via mite loading or by physically interfering with flight aerodynamics. In addition, the feeding action of mites may incur physiological costs in the form of resource allocation. Hosts may also suffer indirect physiological costs associated with energy allocated towards grooming or repairing damaged tissue. The physical burden hypothesis predicts that if the presence of the mites alone is sufficient to impede flight performance, then flies that are infested (i.e. mites attached) for even just a brief period should show diminished flight capacity compared to uninfected flies. More specifically, we expected both *M. subbadius* and *M. muscaedomesticae* to reduce host flight endurance when attached to the host. The physiological hypothesis predicts that if flies incur sustained physiological damage from the feeding mites, then flies infested for an extended period of time should exhibit reduced flight time, even if mites are experimentally removed immediately before the flight assays. In contrast, the phoretic mite *M. muscaedomesticae* is not expected to incur a physiological cost. Still, there is anecdotal evidence suggesting that *M. muscaedomesticae* may actually feed on the haemolymph of adult flies (Wade & Rodriguez, 1961). If this is the case, we would also expect flies that *had* been infested with *M. muscaedomesticae* also to exhibit reduced flight performance.

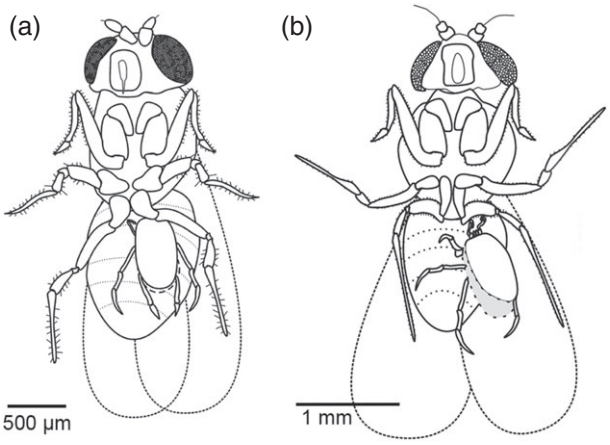

Fig. 1. Illustration of a single female mite attached to the ventral abdomen of an adult fly. (a) *Macrocheles subbadius* mite parasitising a *Drosophila nigrospiracula* fruit fly. (b) *Macrocheles muscaedomesticae* mite attached to *Drosophila hydei*.

Materials and methods

Study systems

The facultative, ectoparasitic mite *M. subbadius* Berlese (Acari: Macrochelidae) occurs naturally with its host *D. nigrospiracula* (Diptera: Drosophilidae) at necrotic cacti [mainly saguaro (*Carnegiea gigantea)* and cardón (*Pachycereus pringlei*)] of the Sonoran Desert (Heed, 1978; Markow, 1988). The mean prevalence and intensity of infection (the number of mites per fly) among *D. nigrospiracula* populations vary depending on the extent of deterioration in the plant tissue. As rot age increases so does prevalence ($\bar{x} = 21\%$, range: 0–50%) and parasite abundance ($\bar{x} = 0.98$, range: 0–5 mites/fly) (Polak & Markow, 1995). Parasitised females

experience attenuated longevity and fecundity, and infested males suffer reduced mating success. *Macrocheles muscadomesticae* (Acari: Macrochelidae) is a free-living, cosmopolitan mite found in association with various fly species, including fruit flies (*Drosophila spp.*), house flies (*Musca domestica*), stable flies (*Stomoxys calcitrans*), and bot flies (*Dermatobia hominis*). Mite prevalence and intensity of infection also varies spatiotemporally (Wade & Rodriguez, 1961).

Adult *M. subbadius* ($n = 120$ per sex) were collected in the field at necrotic saguaro cacti at the Sonoran Desert (Tucson, Arizona, U.S.A.) and used to established laboratory cultures in 2013. Flies were first cleared of mites and then mass-cultured at standard laboratory light and temperature conditions (12 h light, 25 °C: 12 h dark, 24 °C). The flies from which mites were removed were combined with unparasitised hosts to find the base population. Flies were cultured in media containing potato spuds, drosophila instant (Formula 4-24 Instant Drosophila Medium, Carolina Biological Supply Company, Burlington, North Carolina), active yeast, and a small amount of autoclaved necrotic cactus. The *M. muscaedomesticae* population was founded and maintained in a similar manner, except that adult flies ($n = 120$ per sex) were collected from residential compost bins in Edmonton, AB (approx. coordinates: 53.527815°N, 113.483924°W) and cultured on standard agar–molasses–yeast-based fly media.

For both systems, mites were harvested from infected flies collected in the field, and reared on artificial media consisting of wheat bran, wood shavings, and bacteriophagical nematodes which served as a source of food for the mites (Polak, 1996). Mites were maintained in separate incubators under similar conditions (12 h light, 25 °C: 12 h dark, 23 °C).

Mite attachment and flight assays

Infected and control flies (4–14 days old) were selected from the base population and randomly assigned to either the control or infection group. All flies were assayed for flight endurance either: (i) soon after mites were attached or (ii) after an extended period of attachment, with mites removed an hour before the assay.

Experiment 1 (physical burden). *Drosophila hydei* flies were transferred to shortened 200-µl micropipette tips using a fly aspirator. This confined space severely limits the fly's capacity to escape or fight off mites; fly resistance is based primarily on behavioural defences (Luong *et al.,* 2007). Hence, restricting host movement precludes any differences arising from host susceptibility. Adult flies were individually parasitised by transferring five *M. muscaedomesticae* mites to each micropipette tip and stoppered on both ends with cotton; the exposure period lasted 50–60 min. Under light CO_2 anaesthesia (<1 min), flies were tethered by attaching a fine nylon thread (0.1 mm diameter) to the thorax using super glue. *Drosophila nigrospiracula* flies were handled slightly differently because *M. subbadius* mites were more sensitive to CO_2 exposure and tended to detach from flies under anaesthesia. As such, *D. nigrospiracula* flies were tethered first under CO_2 (<1 min), the anaesthesia was then shut off, and four mites were individually transferred onto the tethered fly with a fine paintbrush. Attachment success varied from 1 to 4 mites per fly, comparable to the flies infected in the pipet tips. Infected flies were allowed to recover from anaesthesia for another 50–60 min before commencing the flight assay.

Fig. 2. Photograph of *Drosophila hydei* hovering on a tether with *Macrocheles muscaedomesticae* mite attached to the ventral side of the abdomen.

Experiment 2 (physiological cost). Adult flies were selected from the base populations and parasitised as described above; after 1 h of exposure, infected flies were transferred to separate 35-ml vials containing agar–molasses–yeast substrate and maintained in individual vials at standard rearing conditions for 48 h prior to assay. Flies in this group were inspected for mites 24 h post-parasitising. Two days post-infection, flies from this group were anaesthetised and tethered as described above. At this time, all remaining mites were counted and carefully removed from the infected flies prior to the flight assay. All flies were allowed at least 1 h to recover from anaesthesia prior to the assay. The intensity of infection among this group of flies was estimated using a mite accumulation index = $\sum$(number of days infested × number of mites), over the course of 2 days. *Drosophila hydei* flies were assayed in two replicated blocks.

Control, uninfected flies from each experiment were handled in a similar manner as the infected flies but were not exposed to mites. For each experiment, control and infected flies were simultaneously assayed in random order. Flies were suspended and allowed to hover at 22–23 °C until they reached exhaustion, defined here as failure to sustain flight for a minimum of 10 s after three consecutive gentle puffs of air (Fig. 2). The exhausted fly was freed from its tether by cutting the thread close to the thorax. Flies in Experiment 1 were inspected under a stereomicroscope at the end of the assay to verify the mite count in case any became dislodged in mid-flight. Fly sex, age, and body mass (weighed to the nearest 0.01 mg with a Mettler Toledo XP-105 analytical balance) were also recorded. Experiments 1 and 2 were replicated over two blocks for each of the respective fly-mite study systems.

Statistical analyses

Data were analysed separately for the two experiments and each of the two fly-mite systems. The effect of mites on host flight endurance (log-transformed) was analysed with a generalised linear model (GLM) and backward selection techniques (R statistical package, http://www.r-project.org). We used a drop-in-deviance (from now on = 'deviance') chi-square test to compare competing models ($P \leq 0.05$, chi-square statistic). The full model included the following fixed explanatory variables: replication block, fly body mass, age, sex, infection status, and the interaction between treatment and sex. The relationship between parasite abundance (the number of mites per fly) or mite accumulation index and flight duration was analysed in separate GLMs to test for density-dependent effects of the mites on flight time.

Results

Experiment 1 (physical burden)

Ectoparasitic mites significantly reduced the flight endurance of both species of fruit flies. For *D. nigrospiracula* infected with *M. subbadius*, block (deviance = −9.11, $P < 0.001$) and infection status (deviance = 4.35, $P = 0.002$) were the only significant factors in the minimal model; host body mass, sex, and the interaction of treatment × sex were all non-significant factors ($P > 0.05$) in the model selection process. Infected flies harboured on average 2.21 mites per fly (SD = 0.86, range: 1–4 mites). The mean flight time for flies with mites attached was 57% lower compared to unparasitised, control flies (Fig. 3a). The average flight time for control and infected flies was 57.6 ($n = 30$, SD = 72.0, range: 11.2–238.6) min and 25.0 ($n = 33$, SD = 48.2, range: 0.05–187.6) min, respectively. Among infected flies, there was no density-dependent effect of mite abundance (the number of mites per fly) on flight endurance (Coef. estim. = 0.03, $P = 0.82$).

The impact of *M. muscaedomesticae* mites on the flight endurance of *D. hydei* was relatively more pronounced, even as mite load was comparable ($\bar{x} = 2.30$, SD = 1.2, range: 1–5 mites/fly). Infection status (deviance = −24.7, $P < 0.001$) and block (deviance = −29.0, $P < 0.001$) were the only significant explanatory factors in the minimal model. Host body mass, sex, age, and the interaction between treatment and sex were all non-significant ($P > 0.05$). The flight endurance of flies burdened by mites decreased by 78% compared to flies with no mites attached (Fig. 3a). The average flight time for control flies was 53.2 ($n = 91$, SD = 54.1, range: 0–180.0) min, while flies carrying mites hovered on average of only 11.9 ($n = 91$, SD = 25.5, range: 0–120.0) min. The flight endurance of infected flies did not change significantly with increasing mite load (Coef. est. = −0.05, $P = 0.39$).

Experiment 2 (physiological cost)

Flies that had been fed on by the ectoparasitic *M. subbadius* for 24–48 h exhibited lower flight endurance even although the mites were removed an hour prior to the flight assay (Fig. 3b). The mite accumulation index ranged from 1 to 9 ($\bar{x} = 3.58$, SD = 2.22). The minimal model providing the best fit to the data included the explanatory variables block (deviance = −2038, $P < 0.001$), sex (deviance = −3.63, $P = 0.008$), and infection status (deviance = −2.41, $P = 0.029$). Body mass and the interaction between treatment and sex were not significant ($P > 0.05$). The flight endurance of previously infected flies ($\bar{x} = 52.2$ min, SD = 73.6, range: 0.05–216.5, $n = 33$) was 53% lower compared to control flies ($\bar{x} = 111.3$ min, SD = 118.4, range: 0–392.1, $n = 36$). Overall, the mean flight

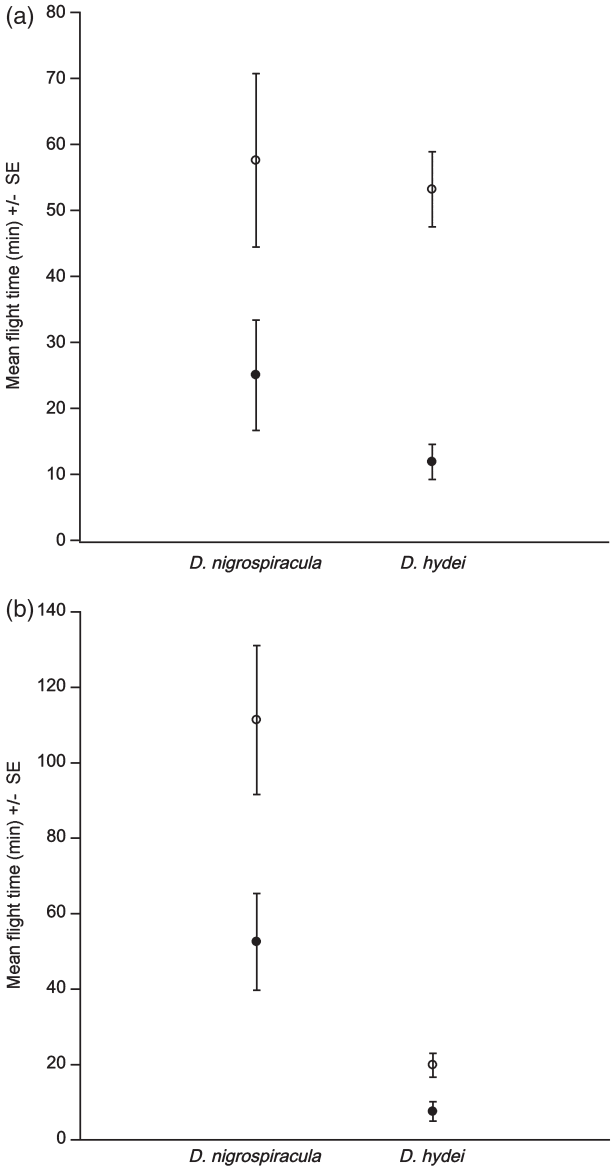

Fig. 3. Flight endurance (mean ± SE) for *Drosophila nigrospiracula – Macrocheles subbadius* and *Drosophila hydei – M. muscaedomesticae* study systems. (a) Experiment 1: flight endurance for control (○) and infected flies (●) with mites still attached during the flight assay, (b) Experiment 2: mites were detached an hour before commencing the flight assay.

time for male flies ($\bar{x} = 110.0$ min, SD = 109.7) was higher than female flies ($\bar{x} = 55.3$ min, SD = 89.4). Flight endurance among infected flies did not vary significantly with increasing mite load (Coef. est. = 0.01, $P = 0.89$).

Surprisingly, flies that harboured the supposed phoretic mite, *M. muscaedomesticae* in the 48 h leading up to the flight assay also experienced a significant decline in flight endurance (Fig. 3b). The mean mite accumulation index was 4.43 (SD = 2.49, range: 1–10 days). The best-fit model included the explanatory variables block (deviance = −3.60, $P = 0.006$) and infection status (deviance = −4.71, $P = 0.002$). The inclusion of the infection by sex interaction, host age, and body mass did not improve the fit of the model ($P > 0.05$). Even although the mites were removed an hour before the assay, flight time for infested flies ($\bar{x} = 7.53$ min, SD = 23.8, range: 0–117.3, $n = 86$) was reduced by nearly 62% compared to control flies ($\bar{x} = 19.8$ min, SD = 34.6, range: 0–181.0, $n = 123$). Among infected flies, there was a slight and marginally significant relationship between flight time and the mite index (Coef. est. = −0.05, $P = 0.04$). Taken together, these results indicate that the mites exert an additional cost beyond just the physical burden of mite attachment, one that is incurred over an extended period of association (presumably feeding on host haemolymph) and manifested in the form of reduced flight endurance.

Discussion

We investigated two non-mutually exclusive mechanisms, physical and physiological harm, by which ectoparasitic mites can influence host flight endurance. The mites adversely affected host flight in both the fly-mite study systems, *D. nigrospiracula – M. subbadius* and *D. hydei – M. muscaedomesticae*. The presence of mites probably affected flight aerodynamics by increasing wing load, drag, and/or by introducing asymmetry depending on the attachment site (McLachlan *et al.,* 2008). Both fly species suffered physiological damage as evidenced by the reduced flight time in the second experiment, even as mites were experimentally removed an hour prior to the assay. A study on ectoparasites of coral reef fish showed that fish parasitised by isopods exhibited higher metabolic rates and lower swimming speeds owing to reduced streamlining, but this effect disappeared when the isopods were experimentally removed (Binning *et al.,* 2013). The physiological damage imposed by the isopods was temporary and only detectable while the isopods were attached. In contrast, the negative physiological/energetic costs of the ectoparasitic mites on *Drosophila* flight endurance appear to be sustained and long term. In addition to the direct physiological damage caused by the feeding action of mites, the time and energy allocated towards grooming off mites or repairing damaged tissue could translate into less energy budget for flight with potentially important consequence of long-distance dispersal. Further studies are needed to tease out precisely how mites interfere with flight aerodynamics, as well as the physiological/energetic costs of infection. Whether *M. muscaedomesticae* is actually feeding on host tissue and/or haemolymph also needs experimental verification.

In both experiments, the impact on flight endurance was more severe among flies harbouring *M. muscaedomesticae* compared to flies infected with *M. subbadius*. This may be due to the considerably larger body size of *M. muscaedomesticae*, particularly given that their corresponding hosts are similar in size: *D. nigrospiracula* mean body length = 3.3 mm (range: 2.7–4.0), and *D. hydei* mean = 3.2 mm (range: 2.9–3.6). Larger mites may also consume bigger bloodmeals and/or appropriate more nutrients from the host. Nagel *et al.* (2010) suggested that the shorter flight distance of infected damselflies was not associated with mite load *per se* but rather the engorgement size of the mites, indicative of a strong physiological cost of feeding. Alternatively, *D. nigrospiracula* may just be better adapted to tolerate parasitism by mites than *D. hydei*. In addition to differential resistance to infection, hosts can minimise the harm caused by a given parasite load via a tolerance response (Read *et al.,* 2008; Schneider & Ayres, 2008; Raberg *et al.,* 2009).

Parasites that negatively affect host dispersal capability have the potential to impact directly gene flow and genetic differentiation among local populations, and ultimately evolutionary processes such as adaptation and speciation. Both species of fruit flies live and reproduce on highly ephemeral habitats including rotting plant material and animal manure characterised by variable spatio-temporal distribution. Natural populations *Drosophila* generally exhibits a wide range of habitat and species-specific dispersal capabilities (Powell, 1997). For instance, the average dispersal distance for *D. nigrospiracula* is 100–300 m day^{-1} (Markow & Castrezana, 2000; Pfeiler *et al.,* 2005). Given these relatively high dispersal distances, any impairment in flight capability could adversely influence the ability of the host to locate food and oviposition sites. For both the fruit flies and mites, which rely entirely on the flies for dispersal to fresh substrates and food resources, a diminished flight capacity can potentially limit gene exchange between local populations, increase genetic differentiation, and promote sexual isolation leading to speciation. Indeed, Pfeiler *et al.* (2005) showed that the stability of genetic structure throughout the geographical range of *D. nigrospiracula* is attributed in part to a capacity to disperse over long distances, along with other behavioural traits. As flies typically disperse before mating (Pfeiler *et al.,* 2005), meta-populations subject to persistent parasitism by mites will experience diminished dispersal capabilities with significant implications for gene flow.

Parasitism can also introduce heterogeneity in the distance travelled by any individual, generating a multimodal dispersal kernel, i.e. varying probability distributions of distance travelled depending on infection status (Fronhofer *et al.,* 2013). While studies have identified a number of factors potentially influencing an animal's dispersal, few have considered the role that parasitism may play in shaping the dispersal kernel, and its implications for population genetics and host–parasite interactions (Iritani & Iwasa, 2014). In a microcosm study, infection by bacterial parasites reduced the short-distance dispersal of the ciliate *Paramecium caudatum* (Fellous *et al.,* 2011). Natal dispersal distances of great tits were shorter for birds leaving nets infested with hen fleas compared to uninfested nests (Heeb *et al.,* 1999). Future studies, particularly under field conditions, are needed to understand the consequences of reduced flight performance on the long-distance dispersal and population structure of natural *Drosophila* populations.

In many cases, parasites are only able to achieve long-distance dispersal when their host disperses (Boulinier *et al.,* 2001). Given that the mites rely entirely on flies for dispersal, a strong reduction in host flight capability could also have important consequences for parasite spread and persistence, gene flow, and the population genetic structure. Blouin *et al.* (1995) found that gene flow among parasitic nematode populations was dependent on host vagility on geographical scales. Similarly, ectoparasitic ticks that infected seabirds with longer dispersal distances exhibited greater genetic differentiation than ticks that parasitise birds with relatively short dispersal distances (McCoy *et al.,* 2003). In the extreme case where prevalence and intensity of infection is moderate to high, mite populations could crash as carrier flies fail to disperse, ultimately leading to elevated risks of parasite extinction (Elzinga & Broce, 1988). Further research is needed to address directly the extent to which a parasite-mediate reduction in host dispersal influences gene flow. Parasite-mediated differences in dispersal rates also have potentially important implications for host–parasite interactions, including disease prevalence, rate of spread, local adaptation, and the evolution of virulence (Folstad *et al.,* 1991; Gandon *et al.,* 1996; Altizer *et al.,* 2000; Boulinier *et al.,* 2001).

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