Oikos 123: 1384–1390, 2014
doi: 10.1111/oik.01148

Subject Editor: Jessica Abbott. Accepted 11 March 2014

Virus infection alters the predatory behavior of an omnivorous vector

Candice A. Stafford-Banks, Louie H. Yang, Marshall S. McMunn and Diane E. Ullman

C. A. Stafford-Banks (canstafford@ucdavis.edu), Dept of Plant Pathology, Univ. of California, Davis, Davis, CA 95616, USA. – L. H. Yang, M. S. McMunn and D. E. Ullman, Dept of Entomology and Nematology, Univ. of California, Davis, Davis, CA 95616, USA.

The interactions between parasites and their hosts can cause profound changes in host behavior, including changes that can alter other trophic interactions. The western flower thrips *Frankliniella occidentalis* is an important omnivorous insect vector of *Tomato spotted wilt virus* (TSWV), which infects crops worldwide and also infects its thrips vector. Here, we show that tospovirus-infected female thrips become more predaceous, illustrating how the functional role of omnivores may change in response to pathogen infection. Our findings support the hypothesis that increased predation among virus-infected female thrips compensates for the detrimental effects of virus infection. Because predatory behavior is unlikely to increase virus transmission to plants, it is doubtful that this shift in feeding behavior is due to an adaptive parasite manipulation of vector behavior. In this study, increases in predatory behavior were observed in female thrips, but not in male thrips. This sexually dimorphic compensatory response indicates that male and female thrips utilize different feeding strategies to compensate for parasite infection, the expression of which is constrained by resource availability. Our findings demonstrate a novel, but potentially common pathway by which viruses can influence the structure of trophic interactions in food webs.

While parasites often influence host behavior in ways that affect parasite transmission (Hurd 2003, Lefevre and Thomas 2008), it is becoming increasingly clear that the interactions between parasites and their hosts generally reflect a more complex and dynamic interplay of reciprocal effects and compensatory responses rather than simple parasite control (Thomas et al. 2005, Lefevre et al. 2008a, b). In particular, parasite infection can often mimic resource limitation (Anderson and May 1979), causing the host to increase its foraging effort to compensate for the costs of infection by the parasite. If increased host foraging is linked to increased parasite transmission, then natural selection may favor parasite phenotypes that induce such compensatory responses in their hosts (Poulin 1995). For example, numerous vector-borne animal-infecting parasites are known to increase probing rates or feeding duration of their vectors, as has been shown for tsetse flies, sand flies, and mosquitoes transmitting trypanosomes, leishmaniae and malaria, respectively (Killick-Kendrick et al. 1977, Jenni et al. 1980, Koella et al. 1998, Hurd 2003, Lefevre and Thomas 2008). In some cases, it is known that the pathogen physically blocks the feeding apparatus, resulting in difficulty during fluid uptake which necessitates increased feeding attempts or greater time engaged in feeding (Hurd 2003, Lefevre and Thomas 2008). While these changes in host behavior are often associated with an increase in parasite transmission, it may be an oversimplification to interpret these as strictly parasite-controlled behavioral changes. Instead, it seems likely that these behavioral changes in the host result from a combination of parasite effects and host compensatory behaviors.

While omnivory is common in both managed and natural ecosystems (Coll and Guershon 2002, Polis and Strong 1996, Thompson et al. 2007), and parasites are increasingly recognized to be ubiquitous and important components of ecological systems (Lafferty et al. 2008, Kuris et al. 2008), the effects of parasites on omnivores have not been well-studied. Facultative omnivores commonly alter their diets depending on many factors, including nutritional needs, food quality and availability (Eubanks and Denno 1999, Agrawal and Klein 2000, Coll and Guershon 2002, Denno and Fagan 2003, Janssen et al. 2003, Magalhães et al. 2005), and opportunistic predation can greatly enhance omnivore fitness by adding protein-rich diet items to complement a primarily phytophagous diet (Trichilo and Leigh 1988, Whitman et al. 1994, Coll and Guershon 2002, Magalhães et al. 2005, Zhi et al. 2006). While it is clear that changes in the feeding behavior of omnivores can have strong effects on ecological dynamics (Fagan 1997, McCann and Hastings 1997, Agrawal and Klein 2000, Eubanks and Denno 2000, Janssen et al. 2003), we are not aware of any studies that have previously investigated how parasite infection of an omnivore effects its diet choice.

The western flower thrips *Frankliniella occidentalis* is an important insect vector of *Tomato spotted wilt virus* (TSWV), a virus which infects hundreds of plant species and causes extensive crop losses worldwide (Parrella et al. 2003, Whitfield et al. 2005, Pappu et al. 2009). Western flower thrips are cell rupture feeders that ingest the contents of epidermal and mesophyll cells, causing severe feeding damage in the form of silvery lesions and bud distortion, discoloration and abscission (Lewis 1973, Kirk 1997). Tospoviruses replicate in their thrips vectors, and are acquired and inoculated during thrips feeding (Ullman et al. 1993). Infection of thrips with TSWV reduces plant-feeding efficiency, causing male, but not female, thrips to increase their plant-feeding behaviors (Stafford et al. 2011). A previous study showed that infected males fed almost three times more than uninfected males, a parasite-mediated behavior that is likely to result in increased virus transmission (Stafford et al. 2011).

In this paper, we investigate the ecological consequences of parasite infection on multiple trophic levels by examining the effects of TSWV infection on the predatory and plant-feeding behaviors of omnivorous thrips. The predatory and plant-feeding behaviors of omnivores have direct effects on their prey and host plants. At the level of prey, we hypothesized that TSWV-infected thrips would increase their predatory behavior compared to uninfected thrips in order to compensate for the negative effects of virus infection. At the level of plants, we hypothesized that these virus-mediated increases in predatory behavior could affect herbivorous feeding in thrips in at least two ways: virus-infected thrips could either 1) augment their phytophagous diet with increased predatory foraging without decreasing herbivorous feeding (the diet augmentation hypothesis, Fig. 1A) or 2) increase their predatory behavior and decrease their herbivory, switching their feeding emphasis from herbivory to predation (diet switching hypothesis, Fig. 1B). We tested these hypotheses in comparison to the null hypothesis that virus infection does not affect predatory or plant-feeding behavior in omnivorous thrips (Fig. 1C).

Material and methods

Organisms

Frankliniella occidentalis and *Tomato spotted wilt virus* (TSWV) isolate MT2 were originally collected in Hawaii. Thrips were reared on green bean pods *Phaseolus vulgaris* as previously described (Ullman et al. 1992). TSWV was maintained in *Emelia sonchifolia* plants through thrips transmission as in Ullman et al. (1992). Uninfected *E. sonchifolia* plants used for the leaf disc assay were grown separate from infected plants in thrips-free cages to ensure that these plants were free from TSWV infection. *Tetranychus urticae* (two-spotted spider mite) colonies were reared under laboratory conditions on cotton *Gossypium hirsutum*, var. Acala seedlings.

Predation experiment

This experiment was designed to determine how sex and TSWV infection affect mite egg consumption. The predation rate (i.e. proportion of mite eggs consumed) of adult thrips was measured for four treatment groups: 1) TSWV-infected males, 2) uninfected males, 3) TSWV-infected females, and 4) uninfected females. For each trial, the oldest undamaged leaf was selected from each of thirty two-month-old, uninfected *E. sonchifolia* plants. Four 17-mm diameter leaf discs were cut from each leaf and treatment groups were randomly assigned to each disc. Leaf discs were placed with their adaxial side up on top of a 15 mm diameter filter paper disc that had been pre-moistened with 50 µl of deionized water and set inside of the cap of an inverted 32 ml snap-top plastic vial. Vials served as interaction arenas, allowing an individual thrips to move and feed on both sides of the leaf disc, while providing adequate moisture to each leaf disc. Five mite eggs were evenly distributed on the adaxial side of each leaf disc. To prevent hatching during the experiment, only young, clear eggs were used (Agrawal et al. 1999).

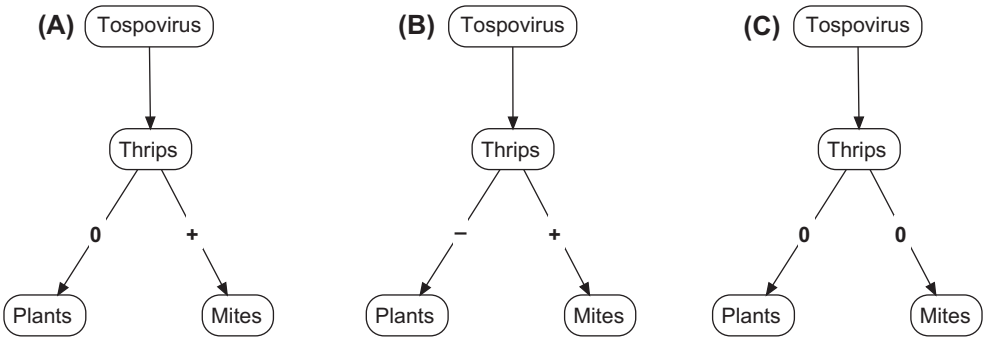

Figure 1. The possible effects of tospovirus infection on thrips omnivory. Omnivory includes effects on herbivory behaviors, measured by plant damage, which may be increased (+), decreased (−), or remain the same (0) and predatory behavior, as measured by mite egg consumption, which may be increased (+), decreased (−), or remain the same (0). We hypothesize that infection with *Tomato spotted wilt virus* and the presence of mite eggs will affect thrips omnivory in one of three ways: augmentation hypothesis (A): infected thrips will increase their propensity to feed on mite eggs (+) and not alter their herbivory (0), augmenting nutritional intake from plant feeding with mite egg consumption; diet switching hypothesis (B): infected thrips will feed less on plant material (−) and more on mite eggs (+), switching their feeding strategies from herbivory to predation; null hypothesis (C): virus infection will not result in a change in thrips herbivory (0) or propensity to feed on mite eggs (0).

TSWV-infected and uninfected thrips were obtained as previously described (Ullman et al. 1992). After a one-hour starvation period, a single thrips was placed onto each randomly assigned leaf disc and vials were sealed. Thrips were allowed to feed for 24 h, after which they were removed and their infection status verified as previously described (van de Wetering et al. 1998). Remaining mite eggs were counted immediately after thrips removal to avoid hatching. The number of mite eggs consumed included partially eaten eggs, with visible evidence of thrips feeding. Hatchings during the trial were rare (4% of mite eggs used), and were counted as uneaten eggs.

Three trials were performed, each with 30 replicates of the four described treatments for a total of 360 leaf discs. All trials were performed under laboratory conditions at 22–25°C. To test for an effect of sex, TSWV infection and their interaction on the proportion of mite eggs consumed, we fit a generalized linear mixed model with a binomially-distributed error variance using maximum likelihood estimation in R ver. 2.15.3 (package lme4). We also used a generalized linear mixed model to test for the effect of sex, TSWV infection and their interaction on the proportion of thrips that consumed at least one mite egg as a measure of predatory behavior. Over-dispersion and zero-inflation were determined to be negligible in both models. We evaluated four variants of each model: 1) no random factors, 2) leaf as the only random factor, 3) trial as the only random factor, and 4) both leaf and trial as random factors. Each of these model variants yielded qualitatively identical results for the fixed factors of interest, and we present the results for the models which minimized the Akaike information criteria (AIC) (Burnham and Anderson 2004) (Supplementary material Appendix 1). Tests of significance for fixed factors were assessed using a likelihood ratio test. If we observed a significant sex × TSWV interaction, we evaluated the effect of TSWV infection separately for male and female thrips.

Predation and herbivory experiment

This experiment was designed to determine a) how TSWV infection and the presence of mite eggs affect leaf damage and b) how TSWV infection affects mite egg consumption by thrips. We used three different measurements to quantify feeding damage: 1) total area of leaf damage, 2) number of individual feeding scars, and 3) average size of feeding scars. Female thrips produce large areas of conspicuous feeding damage on leaves and male thrips do not, therefore this experiment used only female thrips. We measured the amount of feeding damage produced by female thrips in four treatment groups: TSWV-infected thrips with mite eggs present, TSWV-infected thrips without mite eggs present, uninfected thrips with mite eggs present, and uninfected thrips without mite eggs present. For each trial, *E. sonchifolia* leaves and leaf discs were selected for experimentation as described above. Leaf discs with a 13 mm diameter were used to ensure that the whole leaf surface was photographed for digital analysis. Each leaf disc was floated (adaxial side up) on 3 ml of water inside a 12-well tissue culture plate in order to restrict thrips feeding to the upper surface of the leaf disc to enable better visualization of feeding damage. For treatments that contained mite eggs, five mite eggs were evenly distributed on each leaf disc prior to floating.

TSWV-infected and uninfected thrips were placed onto randomly assigned leaf discs after a one-hour starvation period then sealed into the tissue culture plate wells using Parafilm, enabling each well to serve as an individual thrips cage. Thrips were allowed to feed for 24 h, after which they were removed and their infection status verified using a *Vicia faba* leaf disc assay as previously described (van de Wetering et al. 1998). Remaining mite eggs were counted immediately after thrips removal as described above. Each leaf disc was photographed using a digital camera mounted on a microscope. The leaf discs were then analyzed using ImageJ software to calculate the total area of leaf damage which appears as silvery patches in contrast to the dark green color of the leaf tissue. The size and number of individual feeding scars, which indicate uninterrupted bouts of feeding, was also counted from these images. Large areas of damage were broken down into individual scars if areas of silvery tissue were clearly delimited from each other by areas of green tissue.

Eight trials were performed with 20–22 replicates of the four described treatments for a total of 656 leaf discs. Trials 1–3 were performed under laboratory conditions at 20–23°C. Trials 4–8 were performed in an environmental chamber at 25°C with an 8:16 hr light:dark cycle. To analyze leaf damage area and frequency of feeding scars, we used generalized linear mixed models fit by maximum likelihood estimation. Leaf damage models were fit in R ver. 2.15.3 (package lme4) to test for an effect of TSWV infection, mite egg availability and their interaction on these aspects of leaf damage. Total leaf area damaged and scar size models were fit with a normally-distributed error variance while scar frequency models were fit using a Poisson distribution with an observation-level random effect to account for overdispersed data. Following a non-significant mite egg presence × virus interaction in all leaf damage models, this term was removed from all subsequent models (Supplementary material Appendix 1). We evaluated four variants of each of these sets of models, which included leaf, trial and both leaf and trial as random factors. Each of these model variants yielded qualitatively similar results for the fixed factors of interest, and we present the results for the model which minimized the Akaike information criteria (AIC) (Burnham and Anderson 2004) (Supplementary material Appendix 1). To test for an effect of TSWV infection on the proportion of mite eggs consumed and the proportion of thrips consuming at least one mite egg, we used two generalized linear mixed-models with binomially distributed error variances in R ver. 2.15.3 (package lme4). Tests of significance were assessed using a likelihood ratio test.

Results

Predation experiment

Infected thrips consumed significantly more mite eggs than uninfected thrips overall (Fig. 2A, $D = 33.2$, $DF = 2$, $p < 0.0001$, Supplementary material Appendix 1, model

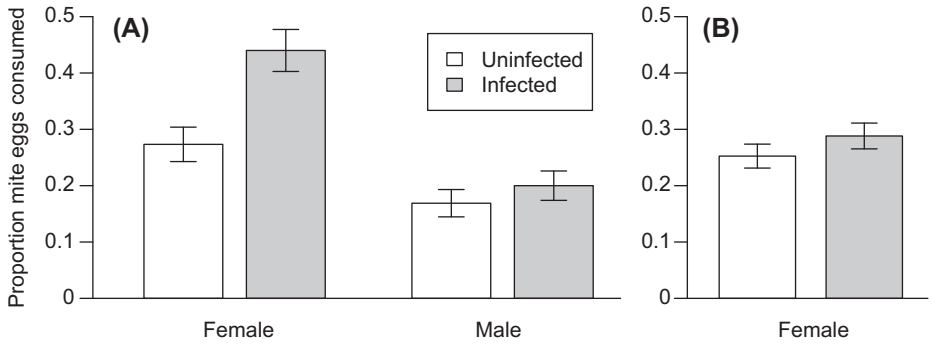

Figure 2. Mite egg consumption by TSWV-infected and uninfected thrips. In the predation experiment (A), infection with TSWV increased the proportion of mite eggs consumed overall. Female thrips consumed a greater proportion of mite eggs than male thrips. Infected female thrips consumed more mite eggs than uninfected females, and virus infection did not alter mite egg consumption by male thrips. In the predation and herbivory experiment (B), TSWV-infected females consumed a larger proportion of mite eggs than uninfected females. Error bars represent ± SE.

1b), and female thrips were more predaceous than male thrips overall (Fig. 2A, $D = 84.6$, $DF = 2$, $p < 0.0001$, Supplementary material Appendix 1, model 1b). Tospovirus infection increased the predatory behavior of female thrips more than male thrips (Fig. 2A, TSWV × sex, $D = 6.8$, $DF = 1$, $p = 0.009$, Supplementary material Appendix 1, model 1b). In sex-specific analyses, virus-infected female thrips significantly increased the proportion of mite eggs consumed from 27 to 44%, compared to uninfected thrips ($D = 25.1$, $DF = 1$, $p < 0.0001$, Supplementary material Appendix 1, model 4b). In contrast, virus-infected male thrips did not significantly increase the proportion of mite eggs they consumed compared to uninfected thrips, although egg consumption did increase slightly from 17 to 20% ($D = 1.6$, $DF = 1$, $p = 0.2$, Supplementary material Appendix 1, model 3b). Over 40% of all male thrips and over 60% of all female thrips tested in this experiment, regardless of infection status, consumed at least one mite egg (Fig. 3A). A larger proportion of females thrips consumed at least one mite egg compared to male thrips (Fig. 3A, $D = 16.3$, $DF = 2$, $p < 0.001$, Supplementary material Appendix 1, model 2b) and a larger proportion of infected thrips consumed at least one mite egg compared to uninfected thrips (Fig. 3A, $D = 6.4$, $DF = 2$, $p = 0.04$, Supplementary material Appendix 1, model 2b).

Predation and herbivory experiment: predation

The proportion of mite eggs consumed was significantly higher for infected thrips versus uninfected thrips (Fig. 2B, $D = 3.92$, $DF = 1$, $p = 0.048$, Supplementary material Appendix 1, model 5a) in this experiment. This difference was not as pronounced as in the predation experiment; infected females increased the proportion of mite eggs consumed from 25 to 29% compared to uninfected thrips (Fig. 2). Over 60% of the thrips tested in this experiment consumed at least one mite egg (Fig. 3B). There was no statistical difference between the infected and uninfected female thrips in the proportion of thrips that consumed at least one mite egg (Fig. 3B, $D = 0.12$, $DF = 1$, $p = 0.73$, Supplementary material Appendix 1, model 6c)

Predation and herbivory experiment: herbivory

Due to the non-significant interaction between thrips virus infection and mite egg presence in predicting total leaf

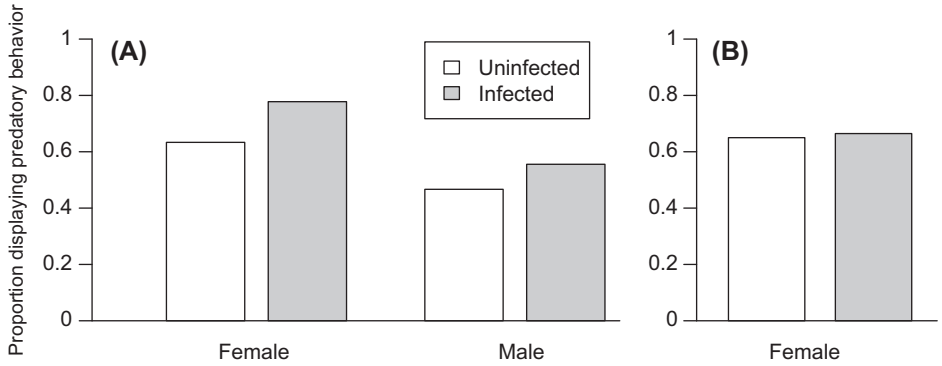

Figure 3. Proportion of TSWV-infected and uninfected thrips displaying predatory behavior by consuming one or more mite egg. In the predation experiment (A) infected thrips displayed predatory behavior with a higher frequency than uninfected thrips. Female thrips displayed predatory behavior more frequently than male thrips. In the predation and herbivory experiment (B) there was no difference between infected and uninfected female thrips.

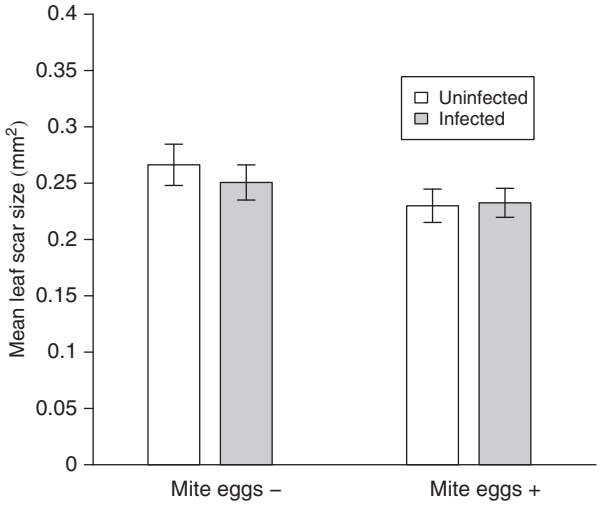

Figure 4. Average size of feeding scars on leaf discs made by TSWV-infected and uninfected thrips. Thrips without access to mite eggs produced marginally larger scars. There was no difference in feeding scar size between infected and uninfected thrips. Error bars represent ± SE.

damage, scar size and scar number, all models presented here exclude this interaction term (for interaction term see Supplementary material Appendix 1, models 10, 11 and 12). The mean size of feeding scars was not affected by thrips virus infection but tended to be larger in the absence of mite eggs (Fig 4. virus infection: $D = 0.21$, $DF = 1$, $p = 0.650$, mite egg presence: $D = 3.72$, $DF = 1$, $p = 0.054$, Supplementary material Appendix 1, model 8a). There was no significant effect of tospovirus infection (Supplementary material Appendix 2, $D = 0.82$, $DF = 1$, $p = 0.3667$, Supplementary material Appendix 1, model 7a) or the presence of mite eggs ($D = 0.36$, $DF = 1$, $p = 0.5478$, Supplementary material Appendix 1, model 7a) on the total area of leaf damage caused by female thrips feeding in this experiment (Supplementary material Appendix 2). Similarly, virus infection and the presence of mite eggs did not significantly affect the number of feeding scars (Supplementary material Appendix 3, virus infection: $D = 0.03$, $DF = 1$, $p = 0.8557$, mite egg presence: $D = 0.50$, $DF = 1$, $p = 0.48$, Supplementary material Appendix 1, model 9a)

Discussion

This study shows a novel mechanism by which pathogens can influence the predator–prey interactions of omnivores. In this system, female tospovirus-infected thrips appear to alter their feeding behavior to compensate for pathogen infection by becoming more predaceous. The results of this paper are consistent with previous findings showing that male thrips compensate for virus infection by increasing plant feeding behaviors (Stafford et al. 2011), but here we show an alternative strategy by which female thrips compensate for virus infection by augmenting their diet through increased predatory feeding behaviors.

The observed increase in the predatory behavior of thrips seems unlikely to directly increase virus transmission. Tospovirus transmission occurs only during plant feeding, therefore the observed tospovirus-mediated increase in female mite egg consumption – without any significant change in herbivory – is unlikely to be an adaptive parasite manipulation of the thrips vector to increase virus transmission. While it is possible that increased predatory behaviors could increase virus transmission through some indirect pathway, such a pathway is not obvious in this system. However, increased predation is known to have positive effects on the survival and fecundity of thrips (Trichilo and Leigh 1988, Zhi et al. 2006), suggesting that this behavioral response may provide a way for the thrips to compensate for the negative effects of virus infection.

Previous studies have shown that tospovirus infection of host plants can have positive effects on the growth rate, survival and fecundity of thrips (Maris et al. 2004, Belliure et al. 2005, 2008), and positive effects on spider mite development and oviposition rate (Belliure et al. 2010). These are likely to be indirect effects driven by virus-mediated changes in the quality of host plants (Maris et al. 2004, Belliure et al. 2005, 2008, 2010). Similarly, changes in the relative quality of available plant material and potential prey resources have previously been shown to change the feeding behaviors of omnivores (Eubanks and Denno 1999, 2000). This study differs from these previous studies in the key finding that tospovirus infection increases the predatory feeding behavior of infected thrips directly, through infection of the vector, as opposed to indirectly through changes in host plant quality.

In this study, virus-infected females showed significant increases in predatory behavior compared to uninfected females, but infection status did not alter the predatory behaviors of male thrips. Combined with the findings of Stafford et al. (2011), this sexually dimorphic compensatory response indicates that male and female thrips utilize different feeding strategies to compensate for parasite infection. This likely occurs because males spend less time feeding on plants and can compensate for virus-mediated reductions in feeding efficiency by increasing herbivorous feeding effort, as previously shown (Stafford et al. 2011). In contrast, females already spend almost all of their time feeding on plants, thus diet augmentation through increased herbivory is difficult. However, female thrips are able to augment their diets through opportunistically consuming nutrient rich foods, such as mite eggs. Thus, male thrips do not show increased predatory behavior, while female thrips appear to follow the expectations of the diet augmentation hypothesis (Fig. 1A). These findings, along with previous work showing that tospovirus infection of thrips impacts herbivory by greatly increasing frequency and duration of male thrips feeding behaviors, shows that virus infection causes increased nutritional requirements for both male and female thrips, and that compensatory responses are sexually dimorphic and constrained by resource availability (i.e. the presence of potential prey) within a community context. These findings highlight the importance of incorporating both male and female omnivores and multiple available food sources to understand how parasite infection may alter both feeding behavior and food choice.

Although both experiments showed a consistent pattern of significantly increased predation among infected female

thrips compared to uninfected female thrips, these effects were more pronounced in the predation experiment compared to the predation and herbivory experiment. These observed differences suggest that the magnitude of these virus-mediated effects may be somewhat context-dependent. The predation experiment used microcosm arenas which differed from those used in the predation and herbivory experiment in several ways, including allowing thrips to move between both sides of the leaf disc and differences in humidity. Although we observed quantitative differences in the size of the infection effect in these two experiments, both experiments showed a qualitatively similar pattern of significantly increased mite egg consumption in infected female thrips compared to uninfected female thrips.

It remains unclear why the infected males do not show a diet shift or diet augmentation in favor of predation when mite eggs are available. We speculate that this may reflect greater carbohydrate limitations on the fitness of males, compared to greater protein limitations on the fitness of females, which need protein to constantly produce eggs (Sang and King 1961, Trivers 1972). *Frankliniella occidentalis* does not actively seek spider mite eggs; instead predation occurs when the eggs are randomly encountered which interrupts normal plant feeding behaviors (Trichilo and Leigh 1988, Wilson et al. 1991). However, as plant quality decreases, the benefits of consuming alternative food sources increases (Agrawal et al. 1999, Magalhães et al. 2005) and as prey quality and availability increases, the need to feed on plant material decreases (Agrawal and Klein 2000). The compensatory responses of female thrips to virus infection is similar to the way in which these thrips respond to poor quality host plants (Agrawal et al. 1999, Magalhães et al. 2005), suggesting that tospovirus infection mimics resource limitation, causing thrips to engage in altered diet mixing. Virus infection appears to increase the benefit of consuming alternative food sources, causing infected female thrips to either increase their acceptance rate of encountered mite eggs or increase searching behaviors for more nutritious food sources.

The results of this study suggest the importance of considering host-parasite interactions in a multi-trophic community context. In this study, observing the expression of this compensatory response required studying omnivorous hosts in a multi-trophic community context that included opportunities for predation as well as herbivory. Even though these experiments were conducted in relatively simple microcosms, the inclusion of a single alternative prey species allowed the omnivorous thrips to express compensatory responses to virus-infection which had not been observed in previous studies that isolated the parasite, plant host and vector interaction. This study illustrates the importance of investigating host–parasite interactions within a community context that is simple enough to be tractable, but complex enough to evaluate the impact on multiple trophic levels. In real-world communities, the potential for complex reciprocal and compensatory responses is likely much greater. In particular, while there are many examples of parasite-induced changes in host behavior that directly increase parasite transmission (Hurd 2003, Lefevre and Thomas 2008), the expectation that parasites generally manipulate host behavior to increase transmission rates is likely to be too simple. Instead, we suggest that the coevolved interactions between parasites and their hosts are more likely to reflect a complex interplay of host responses and parasite counter-responses (Thomas et al. 2005, Lefevre et al. 2008a, b). The results of this study are consistent with this view, showing an increase in the predatory behavior of an omnivorous host in response to virus infection which does not appear to be directly linked with increases in virus transmission.

These observed changes in the predatory behavior of thrips suggest opportunities to study the ecological consequences of parasite infection on multiple trophic levels. In whole ecosystems, parasites can alter food web structure and stability by changing the connectance and energy flow within food webs (Marcogliese and Cone 1997, Lafferty et al. 2006, 2008, Lefevre et al. 2008a). For example, parasites can strengthen predator–prey interactions by increasing the likelihood that an infected host will be consumed by predators (Lafferty 1992, Lefevre et al. 2008a, Sato et al. 2011). Here, we report a unique strengthening of predator–prey interactions in which parasite-induced behavioral changes cause the infected vector to increase predation of a non-host. These changes in predation may also affect other trophic levels within the community via indirect pathways. For example, although the increased rates of predation did not directly reduce rates of herbivory in thrips, increased mite predation may negatively affect herbivorous mite populations, thus indirectly decreasing total herbivory on plant hosts. The findings of this study suggest the intriguing possibility that tospovirus infection may also have positive indirect effects on plants, especially in systems where host plants are more strongly affected by herbivore damage than tospovirus infection. Given the ubiquity of parasites (Lafferty et al. 2008) and context-dependent omnivory (Agrawal et al. 1999) in nature, we suggest that parasite infection may be an important factor influencing the diets of omnivores.

Acknowledgements – We would like to thank Mai Yang for her assistance. This work was supported by the College of Agricultural and Environmental Sciences and the graduate program of the Dept of Plant Pathology, Univ. of California, Davis. This work was also supported by the NIFA/AFRI CAP grant: Advancing innovative technologies and integrated strategies for sustainable management of thrips-transmitted tospoviruses, grant no. GRANT11454594.

References

Agrawal, A. A. and Klein, C. N. 2000. What omnviores eat: direct effects of induced plant resistance on herbivores and indirect consequences for diet selection by omnivores. – J. Anim. Ecol. 69: 525–535.

Agrawal, A. A. et al. 1999. Influence of prey availability and induced host-plant resistance on omnivory by western flower thrips. – Ecology 80: 518–523.

Anderson, R. M. and May, R. M. 1979. Population biology of infectious diseases: Part I. – Nature 280: 361–367.

Belliure, B. et al. 2005. Herbivore arthropods benefit from vectoring plant viruses. – Ecol. Lett. 8: 70–79.

Belliure, B. et al. 2008. Herbivore benefits from vectoring plant virus through reduction of period of vulnerability to predation. – Oecologia 156: 797–806.

Belliure, B. et al. 2010. Vector and virus induce plant responses that benefit a non-vector herbivore. – Basic Appl. Ecol. 11: 162–169.

Burnham, K. P. and Anderson, D. R. 2004. Multimodel interference: understanding AIC and BIC in model selection. – Sociol. Meth. Res. 33: 261–304.
Coll, M. and Guershon, M. 2002. Omnivory in terrestrial arthropods: mixing plant and prey diets. – Annu. Rev. Entomol. 47: 267–297.
Denno, R. F. and Fagan, W. F. 2003. Might nitrogen limitation promote omnivory among carnivorous arthropods? – Ecology 84: 2522–2531.
Eubanks, M. D. and Denno, R. F. 1999. The ecological consequences of variation in plants and prey for an omnivorous insect. – Ecology 80: 1253–1266.
Eubanks, M. D. and Denno, R. F. 2000. Host plants mediate omnivore–herbivore interactions and influence prey suppression. – Ecology 81: 936–947.
Fagan, W. F. 1997. Omnivory as a stabilizing feature of natural communities. – Am. Nat. 150: 554–567.
Hurd, H. 2003. Manipulation of medically important insect vectors by their parasites. – Annu. Rev. Entomol. 48: 141–161.
Janssen, A. et al. 2003. Poor host plant quality causes omnivore to consume predator eggs. – J. Anim. Ecol. 72: 478–483.
Jenni, L. et al. 1980. Feeding behaviour of tsetse flies infected with salivarian trypanosomes. – Nature 283: 383–385.
Killick-Kendrick, R. et al. 1977. *Leishmania* in phlebotomid sandflies. V. The nature and significance of infections of the pylorus and ileum of the sandfly by *leishmania* of the braziliensis complex. – Proc. Biol. Sci. 198: 191–199.
Kirk, W. D. J. 1997. Feeding. – In: Lewis, T. (ed.), Thrips as crop pests. CAB Int., pp. 119–174.
Koella, J. C. et al. 1998. The malaria parasite, *Plasmodium falciparum*, increases the frequency of multiple feeding of its mosquito vector, *Anopheles gambiae*. – Proc. Biol. Sci. 265: 763–768.
Kuris, A. M. et al. 2008. Ecosystem energetic implications of parasite and free-living biomass in three estuaries. – Nature 454: 515–518.
Lafferty, K. D. 1992. Foraging on prey that are modified by parasites. – Am. Nat. 140: 854–867.
Lafferty, K. D. et al. 2006. Parasites dominate food web links. – Proc. Natl Acad. Sci. USA 103: 11211–11216.
Lafferty, K. D. et al. 2008. Parasites in food webs: the ultimate missing links. – Ecol. Lett. 11: 533–546.
Lefevre, T. and Thomas, F. 2008. Behind the scene, something else is pulling the strings: emphasizing parasitic manipulation in vector-borne diseases. – Infect. Genet. Evol. 8: 504–519.
Lefevre, T. et al. 2008a. The ecological significance of manipulative parasites. – Trends Ecol. Evol. 24: 41–48.
Lefevre, T. et al. 2008b. Exploiting host compensatory responses: the 'must' of manipulation? – Trends Parasitol. 24: 435–439.
Lewis, T. 1973. Thrips: their biology, ecology and economic importance. – Academic Press.
Magalhães, S. et al. 2005. Host-plant species modifies the diet of an omnivore feeding on three trophic levels. – Oikos 111: 47–56.
Marcogliese, D. J. and Cone, D. K. 1997. Food webs: a plea for parasites. – Trends Ecol. Evol. 12: 320–325.
Maris, P. C. et al. 2004. *Tomato spotted wilt virus* infection improves host suitability for its vector *Frankliniella occidentalis*. – Phytopathology 94: 706–711.
McCann, K. and Hastings, A. 1997. Re-evaluating the omnivory-stability relationship in food webs. – Proc. R. Soc. B. 264: 1249–1254.
Pappu, H. R. et al. 2009. Global status of tospovirus epidemics in diverse cropping systems: successes achieved and challenges ahead. – Virus Res. 141: 219–236.
Parrella, G. et al. 2003. An update of the host range of *Tomato spotted wilt virus*. – J. Plant Pathol. 85: 227–264.
Polis, G. A. and Strong, D. R. 1996. Food web complexity and community dynamics. – Am. Nat. 147: 813–846.
Poulin, R. 1995. "Adaptive" changes in the behaviour of parasitized animals: a critical review. – Int. J. Parasitol. 25: 1371–1383.
Sang, J. H. and King, R. C. 1961. Nutritional requirements of axenically cultured *Drosophila melanogaster* adults. – J. Exp. Biol. 38: 793–809.
Sato, T. et al. 2011. Nematomorph parasites drive energy flow through a riparian ecosystem. – Ecology 92: 201–207.
Stafford, C. A. et al. 2011. Infection with a plant virus modifies vector feeding behavior. – Proc. Natl Acad. Sci. USA 108: 9350–9355.
Thomas, F. et al. 2005. Parasitic manipulation: where are we and where should we go? – Behav. Process. 68: 185–199.
Thompson, R. M. et al. 2007. Trophic levels and trophic tangles: the prevalence of omnivory in real food webs. – Ecology 88: 612–617.
Trichilo, P. J. and Leigh, T. F. 1988. Influence of resource quality on the reproductive fitness of flower thrips (Thysanoptera: Thripidae). – Ann. Entomol. Soc. Am. 81: 64–70.
Trivers, R. L. 1972. Parental investment and sexual selection. Sexual selection and the descent of man. – Aldine Publishing Co., pp. 136–179.
Ullman, D. E. et al. 1992. A midgut barrier to *Tomato spotted wilt virus* acquisition by adult western flower thrips. – Phytopathology 82: 1333–1342.
Ullman, D. E. et al. 1993. Tospovirus replication in insect vector cells: immunocytochemical evidence that the nonstructural protein encoded by the S RNA of tomato spotted wilt tospovirus is present in thrips vector cells. – Phytopathology 83: 456–463.
van de Wetering, F. et al. 1998. Distinct feeding behavior between sexes of *Frankliniella occidentalis* results in higher scar production and lower tospovirus transmission by females. – Entomol. Exp. Appl. 88: 9–15.
Whitfield, A. E. et al. 2005. Tospovirus–thrips interactions. – Annu. Rev. Phytopathol. 43: 459–489.
Whitman, D. W. et al. 1994. Carnivory in phytophagous insects. – In: Ananthakrishnan, T. N. (ed.), Functional dynamics of phytophagous insects. Sci. Publ., pp. 161–205.
Wilson, L. T. et al. 1991. Natural enemies of spider mites (Acari: Tetranychidae) on cotton: density regulation or casual association? – Environ. Entomol. 20: 849–856.
Zhi, J. et al. 2006. Host plant-mediated interaction between populations of a true omnivore and its herbivorous prey. – Entomol. Exp. Appl. 121: 59–66.

Supplementary material (available as Appendix oik.01148 at <oikosjournal.org/readers/appendix>). Appendix 1.