6-26-2018

When Does Less Equal More? Assessing The Mechanisms Driving Compensatory Mortality And The Hydra Effect

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WHEN DOES LESS EQUAL MORE? ASSESSING THE MECHANISMS DRIVING COMPENSATORY MORTALITY AND THE HYDRA EFFECT

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53 Pages

Many populations across taxa are regulated by negative density-dependence, wherein increased population sizes lead to decreased birth rates or increased mortality. By releasing such populations from some level of these density-dependent effects, extrinsic mortality can lead to counter-intuitive results, such as no change in population size (compensation), or an increase in population size (overcompensation). These results have been documented experimentally, but there currently exists a dearth of empirical studies exploring the mechanisms behind the phenomenon. We tested the relationship between extrinsic mortality level and (over)compensation in four mosquito species – *Aedes aegypti, A. albopictus, A. triseriatus,* and *Culex pipiens* – by exposing larvae to a range of mortalities by artificial harvest or predation and analyzing the numbers of adults produced. Additionally, we examined the ability of three functionally diverse predators – *Mesocyclops longisetus, Anopheles barberi,* and *Corethrella appendiculata* – to induce (over)compensation in *A. aegypti* by exposing larvae to predation by either single or multiple predator species treatments. We found overcompensation across all levels of mortality in *A. triseriatus* and *C. pipiens* and at intermediate levels of mortality in *A. aegypti*. Low-to-intermediate levels of mortality were compensatory in *A. albopictus*, followed by a decrease in adult production at the highest mortality level. Predation induced compensation in the second experiment and an increase in population equilibrium, a phenomenon known as the

‘hydra effect.’ These results provide a better understanding of the conditions under which mortality may increase adult production or population equilibrium. Because overcompensation and the hydra effect are predicted to affect a wide range of taxa and food webs, our findings present implications for developing proper population management and pest reduction strategies.
KEYWORDS: hydra effect; compensation; overcompensation; *Aedes; Culex pipiens*; predator-prey; MPEs; index of performance; density dependence

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A Thesis Submitted in Partial
Fulfillment of the Requirements
for the Degree of

MASTER OF SCIENCE

School of Biological Sciences

ILLINOIS STATE UNIVERSITY

2018

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ACKNOWLEDGMENTS

I am eternally grateful to my friends and family who have provided a lifetime of support and guidance upon which I can always rely. Thank you to the members of the Juliano lab, especially Geoff Ower for training me in mosquito rearing protocols and sharing his problem-solving abilities. This project would not have been possible without the assistance of the undergraduate researchers in the lab, especially Amy Gensler, Keenan Longan, and Kaitlyn Frederick, who dedicated many hours to these experiments. Two fearless women in my life – Rebecca Neale and Whitney Morelli – braved the Florida jungle to assist me during my predator collection trips, for which I cannot thank them enough. Thank you to Vickie Borowicz and Ben Sadd for serving on my thesis committee and offering me their expertise. Finally, I would like to thank my advisor, Steve Juliano, for the enormous amount of guidance and support he has provided throughout my time at ISU. He has instilled in me an even greater appreciation of scientific discovery and the natural world which I will further cultivate in the course of my education and career.

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Abstract

Extrinsic mortality applied to negatively density-dependent populations can result in no change in number of survivors (compensation) or an increase in number of survivors (overcompensation) by releasing the survivors from some level of density-dependent effects. Theoretical studies have attempted to elucidate the mechanisms behind this phenomenon, but there is little empirical work testing the predictions of those efforts. The purpose of this study is to examine the relationship between the level of extrinsic mortality (*i.e.,* percentage of mortality) and the level and likelihood of overcompensation. We test the hypotheses that 1) overcompensation is induced at low-to-intermediate levels of extrinsic mortality with additive mortality occurring above a threshold, and 2) different species exhibit varying levels and likelihood of (over)compensation due to differential responses to density. These hypotheses were tested in four container mosquitoes species (Diptera: Culicidae) – *Aedes aegypti, A. albopictus, A. triseriatus,* and *Culex pipiens*. Cohorts of 150 larvae were exposed to either artificial harvest ranging from 0-70% on day 2 or exposure to predation by 1, 2, or 3 *Mesocyclops longisetus* (Crustacea: Copepoda). The number of adults produced per container was recorded and analyzed by species using mixed-effects generalized linear models. *Aedes triseriatus* and *C. pipiens* demonstrated overcompensation across all mortality levels. Overcompensation was induced in *A. aegypti* up to the 50% mortality level, followed by compensation at 70%. Mortality was compensatory from 0-50% in *A. albopictus*, followed by partial compensation above 50%. Mortality from predation by *M. longisetus* led to greater adult production compared to artificial harvest in *A. aegypti* and *A. albopictus* and less adult

production in *C. pipiens*. Our results do not provide full support for our first hypothesis, but rather demonstrate three species-specific patterns in the relationship between extrinsic mortality level and (over)compensation. Our second hypothesis was supported. The relative levels of (over)compensation demonstrated in the four species is inversely related to their competitive abilities and responses to density. These results provide further insight on the mechanisms driving a phenomenon that is predicted to affect many taxa and food webs, and they present practical implications for developing effective strategies to control pest populations.

Keywords: overcompensation; density-dependence; predator-prey; *Aedes*; *Culex pipiens*; hydra effect.

Introduction

Population responses to extrinsic sources of mortality, such as harvesting and predation, have traditionally been predicted to result in a net reduction in population size. Extrinsic mortality, it would be reasoned, interacts additively with intrinsic mortality sources such as intraspecific resource competition. Under certain circumstances, however, extrinsic mortality may produce less intuitive results. Populations regulated by negative density-dependent effects are constrained by intrinsic mortality or reductions in reproduction rates as population sizes approach and exceed the carrying capacity of their habitats (Sibly et al. 2005). Extrinsic mortality affecting such populations can yield the "hydra effect," wherein an increase in population equilibrium density is produced (Abrams and Matsuda 2005). This depends on the production of the same (compensation) or a greater (overcompensation) number of surviving individuals in the following life-stage as would occur without the extrinsic mortality. Compensation may occur when extrinsic mortality kills individuals that would have otherwise

died of intrinsic mortality sources. If removal of these individuals leads to greater per capita resource levels for the surviving population, overcompensation may occur.

The hydra effect was first postulated in Ricker (1954), in which fishery-based models yielded population density increases in response to extrinsic mortality applied to immature life stages. Since Ricker's (1954) publication, this phenomenon has been the subject of multiple theoretical studies to determine the conditions under which the hydra effect may occur (reviewed in Abrams 2009). The timing of extrinsic mortality relative to density-dependent events on a population is predicted to be a key factor in determining whether increases in population density will occur (Jonzen and Lundberg 1999, Ratikainen et al. 2008, Abrams 2009, Zipkin et al. 2009, Hilker and Liz 2013, McIntire and Juliano 2018). Mortality occurring before the onset of density-dependence is predicted to lead to overcompensatory responses in population density (Jonzen and Lundberg 1999, Abrams 2009, McIntire and Juliano 2018). Life history traits can further influence the occurrence of overcompensation fluence the occurrence of overcompensation (De Roos et al. 2007, Zipkin et al. 2009, Karatayev and Kraft 2015). Population regulation by maturation versus reproduction determines whether overcompensation can occur in the juvenile and adult stage (De Roos et al. 2007), and high reproduction rates and static maturation rates are postulated to increase the chances of overcompensation in adults (Zipkin et al. 2009, Karatayev and Kraft 2015).

Overcompensatory mortality and the hydra effect are predicted to occur in a wide variety of food web structures (Cortez and Abrams 2016). However, despite the number of theoretical studies examining the hydra effect, there are relatively few empirical examples in natural or laboratory populations. Compensatory mortality has been demonstrated in response to harvest (Weber et al. 2016), predation (Nannini and Juliano 1998), and parasitism (Washburn et al.

1991), and overcompensation has been demonstrated in response to harvest (Nicholson 1954, Cameron and Benton 2004, Zipkin et al. 2008), parasitism (Washburn et al. 1991), and real or simulated toxin exposure (Agudelo-Silva and Spielman 1984, Moe et al. 2002). While these empirical studies document the occurrence of the phenomenon, the mechanisms underlying the hydra effect remain elusive.

The extent of extrinsic mortality impinging on a population (*i.e.* the percentage of the population killed) should affect whether compensation or overcompensation occurs (Boyce et al. 1999, Ratikainen et al. 2008, Abrams 2009, Zipkin et al. 2009). There should exist a threshold above which extrinsic mortality is additive to intrinsic mortality sources (Boyce et al. 1999, Ratikainen et al. 2008, Abrams 2009). Above this threshold, extrinsic mortality removes a proportion of the population that exceeds the proportion that would have been removed due to density-dependent effects, thus lowering survivorship and population density. Understanding the relationship between level of mortality and overcompensation would provide a better understanding of the population processes occurring and enable better decisions about managed populations. However, we have only found one empirical study that examines this relationship (Sandercock et al. 2011). Sandercock *et al.* (2011) found a partially compensatory response to harvest (harvest reduced population size, but at a lower magnitude than the amount harvested) in willow ptarmigan populations at 15% harvested and an additive response at 30% harvested. This additive response is predicted to occur when extrinsic mortality exceeds the level of density-dependent mortality (Sandercock et al. 2011). While this study did not demonstrate full compensation nor overcompensation, the results are none-the-less informative about the relationship between level of mortality and compensation, and it provides support for predictions from theory (Boyce et al. 1999, Ratikainen et al. 2008, Abrams 2009, Zipkin et al. 2009).

The strength of density-dependent effects regulating a population should affect the level of overcompensation induced by extrinsic mortality, as overcompensation occurs when extrinsic mortality releases a population from some level of density-dependent effects. Stronger density-dependent effects result in greater suppression of population sizes at high densities than do weak density-dependent effects. This greater suppression provides the potential for a larger increase in population size due to overcompensation. By this logic, populations facing stronger density-dependence are predicted to have greater levels of overcompensation than similar populations facing weaker density-dependence. However, this predicted relationship between the strength of density-dependent effects and overcompensation has yet to be tested in any published study we have found.

The purpose of this study is to test the effects of multiple levels of extrinsic mortality of larvae on induction of overcompensation of adult production. This question was tested in four container mosquito species (Diptera: Culicidae): *Aedes aegypti, A. albopictus, A. triseriatus,* and *Culex pipiens*. These species are suited for studies examining overcompensation because they are highly negative-density dependent in their aquatic larval stages (Dye 1984, Léonard and Juliano 1995, Lord 1998, Alto et al. 2012). To compare responses to artificial and natural mortality sources, we included in our experiment random harvest and predation from *Mesocyclops longisetus* (Crustacea: Copepoda) as sources of extrinsic mortality. While past empirical studies on overcompensation and the hydra effect have used artificial harvesting or predation as sources of mortality, none to date have compared the two. Differences between the two mortality sources (*e.g.,* selectivity of mortality, the amount of biomass left behind in the form of partially consumed victims, or anti-predator behavior modifications in prey) may result in different likelihoods or levels of overcompensation.

Based on past theoretical predictions (Boyce et al. 1999, Ratikainen et al. 2008, Abrams 2009, Zipkin et al. 2009) and the work of Sandercock (2011), we hypothesize that overcompensation occurs at low to intermediate levels of extrinsic mortality, with additive mortality occurring above a threshold. If this hypothesis is correct, we predict 1) low-to-intermediate levels of extrinsic mortality applied at early larval stages of container mosquitoes will yield an increase in adult production, 2) above a threshold of extrinsic mortality, the number of adults will decrease with added extrinsic mortality, and 3) if mortality imposed by real predators and artificial harvest are equally random with respect to individuals' traits, they will impose similar levels of overcompensation in adult production for similar levels of mortality; alternatively if real predators selectively remove individuals more or less likely to die due to intrinsic mortality, then we expect the two mortality sources to yield different levels of overcompensation. Our second hypothesis is that due to differential competitive abilities and responses to larval density, different species will exhibit varying likelihood and levels of (over)compensatory mortality. Resource competition appears to be strong in larvae of container mosquitoes (reviewed by Juliano 2009, 2010), and general theory (Tilman 1982, Chase and Leibold 2003) and experiments with these mosquito species (Murrell and Juliano 2012) indicate that ability to maintain survival and population growth at low per capita resource levels is the main determinant of competitive ability. Based on the competitive abilities of the four species included in this experiment (Peters et al. 1969, Barrera 1996, Grill and Juliano 1996, Juliano 1998, 2009, 2010, Carrieri et al. 2003, Braks et al. 2004, Costanzo et al. 2005, Reiskind and Lounibos 2009, Murrell and Juliano 2012, O'Neal and Juliano 2013), we predict the respective levels of (over)compensation demonstrated in the four species will be, from highest to lowest, *C. pipiens, A. triseriatus, A. aegypti,* and *A. albopictus*, as this coincides with their competitive

abilities.

Methods

Laboratory colony rearing

The mosquitoes used in this study were from laboratory colonies at Illinois State University, Normal, IL, that were initiated with field-collected larvae and pupae from: New Orleans, LA (*A. aegypti*); Tyson Research Center in Eureka, MO (*A. triseriatus*); Harrisburg, PA (*A. albopictus*); and Normal, IL (*C. pipiens*). The colonies had been maintained in the lab for approximately 3 years (*A. aegypti*), 2.5 years (*A. albopictus*), and 1 year (*A. triseriatus* and *C. pipiens*) Adults were provided blood meals from anesthetized guinea pigs and mice (IUCAC# 842043). Eggs from the three *Aedes* species were deposited on seed germination paper and allowed to embryonate for at least two weeks. *Culex pipiens* eggs were deposited in a timothy hay/oak leaf infusion.

Mesocyclops longisetus occurs in South and Central America as well as parts of the southern United States (Reid 1993). They are voracious predators of mosquito larvae, and individuals are capable of killing up to 38 larvae per day (Marten et al. 1994). *M. longisetus* used in this study were taken from a laboratory colony maintained at Illinois State University in Normal, IL, which originated from a colony maintained at the Florida Medical Entomology Laboratory (FMEL) in Vero Beach, FL. The colony was housed at room temperature in 2-quart plastic storage containers and provided *Paramecia caudata* approximately bi-monthly as food resources.

Experimental setup

Four days prior to the beginning of the experiment, 500 ml plastic containers were filled with 400 ml ultrapure water, 1g dried live oak leaves (*Quercus viriginiana*) collected from Vero

Beach, FL, 0.05 g dried decorated crickets (*Gryllodes sigillatus*), and 100 µl microbial inoculum collected from a rain-filled bucket in Merwin Nature Preserve, Lexington, IL. Lids were placed on the containers with holes punched for ventilation. Containers were housed in an environmental chamber at 25°C until the beginning of the experiment to allow the establishment of a microbial community to serve as food resources for mosquito larvae.

Eggs from the three *Aedes* species were hatched by placing strips of egg papers in vials containing 0.4 g/l Difco™ nutrient broth (Becton, Dickinson and Company, Sparks, MD) mixed with ultrapure water and stored for 24 hours at 25°C. *Culex pipiens* eggs were collected on the day as oviposition and stored in ultrapure water at 25°C for 24 hours. At the start of the experiment, hatchling larvae of all species were rinsed in ultrapure water prior to counting.

Containers (n=204) were randomly assigned a species and mortality treatment. Mortality treatments consisted of either a percentage of the cohort (0, 10, 30, 50, or 70%) to be removed at random on day 2, or the addition of 1, 2, or 3 female *M. longisetus* as predator treatments. On day 0 of the experiment, 150 mosquito hatchlings of one species were added to appropriate containers, and *M. longisetus* were added to predator treatment containers. This initial density of mosquito larvae was chosen because Containers were returned to the environmental chamber, which maintained a 14:10 light:dark photoperiod.

Artificial mortality treatments were applied on day 2, as this timing has been demonstrated to be most likely to induce overcompensation in this system (McIntire and Juliano 2018). Each container was emptied into an enamel pan and leaves were removed and set aside. Surviving larvae in predator containers were counted and returned to the containers with the leaves, and the percent mortality by day 2 due to predation was determined as the proportion of the initial 150 larvae that were missing or dead. Predators were difficult to detect with certainty

in these containers, so they were not removed. Artificial mortality treatments were applied by randomly removing surviving larvae until the number of remaining individuals equaled the appropriate percentage of the original cohort size (150). After stirring the water with larvae in the pan, an array of 6 PVC pipes 4 cm long and 2.5 cm in diameter were placed in the water, trapping larvae within the pipes. A random number generator was used to select one of the numbered pipes, and larvae were removed from that section. This process was repeated three times before removing the pipes, stirring the contents, and replacing the pipes. This process was repeated until the desired number of survivors remained in each container. Larvae removed were set aside for each species and added, as needed, to any artificial mortality container with fewer survivors than needed for its assigned mortality treatment. The average number of survivors by day 2 in artificial mortality treatments was 138, and all 0% mortality containers required an addition of mosquitoes to reach 150. Thus, at 2 days, we precisely controlled the numbers of larvae surviving in artificial mortality containers.

On days 16 and 30, 0.5g dried live oak (*Quercus virginiana*) leaves and 0.025g dried decorated crickets were added to each *Aedes* container, and on days 9, 16, 23, and 30, the same amounts were added to the *C. pipiens* containers. More frequent resource additions were used for *C. pipiens* because initial trials using the same feeding schedule as the *Aedes* containers failed to produce adults in all *C. pipiens* treatments. Containers were checked daily for pupae, which were removed and placed in 0.25 dram vials with cotton stoppers. Pupae were stored in the environmental chamber and checked daily for emergence, and the date of emergence for each individual was recorded. All individuals reaching adulthood were counted as survivors.

Statistical analyses

This experiment was conducted in incomplete time blocks (n=8) over a period of 9 months. Blocks were incomplete due to egg availability. A single quantitative mortality variable was created to enable comparison of artificial mortality and predator-induced mortality. This mortality variable was equal to the percent of individuals removed on day 2 for artificial mortality treatments and the percentage of mortality due to predation observed on day 2 for predator treatments. Because *Aedes* and *Culex* species were given different food treatments, they were analyzed separately. The number of survivors per container was analyzed using a mixed-effects generalized linear model with PROC GLIMMIX in SAS 9.4. Models including predator presence/absence, species (*Aedes* analysis only), mortality, mortality^2, mortality^3, and all interactions, with block included as a random effect, were evaluated. Higher-order polynomial terms were not tested in models without lower order terms. To correct for over-dispersion, a negative binomial error distribution was used. Because the species*mortality interaction was significant in the *Aedes* analysis (see Results), separate regressions were run to select the best model for each species. Corrected Akaike's information criteria (AICc; Sugiura 1978) were compared to determine the best model, and the model with the lowest evidence ratio was selected in the case of *A. aegypti, A. triseriatus,* and *C. pipiens.* The two *A. albopictus* models with the lowest AICcs were similar in value (Table 1.1), so excluding the second-best model would discard valuable information. The most likely model contained the predator effect alone, while the second-most likely model contained predator and a cubic relationship to mortality. Since the third and fourth best models contained the linear and quadratic relationships to mortality, respectively, and the evidence ratio for the fourth model was relatively low (E=2.044), these four models were averaged following methods described in Anderson (2008) to calculate predicted

numbers of survivors and standard errors, which were weighted by their respective model weight (w_i) values when all unselected models were excluded (Table 1.2). These four models had a combined weight of w_i=0.7037 when all possible models were considered (Table 1.1, sum of w_i from top 4 models).

Results

The mortality*species interaction was significant in the analysis combining all three *Aedes* species ($F_{2,132}$ = 15.55, p<0.0001). Therefore, we report here the results from the single-species analyses. The model average prediction for *A. albopictus* incorporated predictions from the predator-only (w_i=0.3376), predator + a cubic relationship to mortality (w_i=0.3054), predator + linear effect of mortality (w_i=0.1919), and predator + a quadratic relationship to mortality (w_i=0.1651) models (Table 1.1). This model-averaged prediction showed no change in number of survivors with added mortality (compensation) through the 50% mortality level, followed by a decrease in survivorship (Table 1.1, Figure 1.1). Mortality by predation resulted in greater number of survivors than did artificial mortality, with predator treatments yielding 3.74-7.19 more survivors depending on the model (Table 1.2).

The *A. aegypti* and *C. pipiens* data were each best characterized by the models containing predator and a cubic relationship to mortality (Tables 3 and 4), while the *A. triseriatus* data were best fit by a model containing only the linear effect of mortality (Table 1.5). The effect of predator was significantly positive for *A. aegypti* with an estimate of 5.80 more adults produced in predator treatments and negative for *C. pipiens* with an effect of 1.88 fewer adults produced in predator treatments (Table 1.6, Figure 1.1). *Aedes aegypti* showed a slight decrease in survivors with low mortality levels compared to the 0% mortality level, but this trend reversed between 10% and 30% mortality, resulting in compensation at 30% (Table 1.6, Figure 1.1). Intermediate

levels of mortality were overcompensatory, with more adults produced compared to 0% mortality level, and the number of survivors dropped to approximately equal numbers compared to no mortality by the 70% level (Table 1.6, Figure 1.1). *Aedes triseriatus* demonstrated overcompensation across the entire range of mortality applied, with a linear increase in survivors as greater levels of mortality were applied (Table 1.6, Figure 1.1). *Culex pipiens* showed an overcompensatory response at all mortality levels, with the greatest number of survivors produced in the 30% mortality level (Table 1.6, Figure 1.1). Survivors dropped in the higher mortality levels compared to 30%, but the number remained greater than that at the 0% level.

Discussion

Relationship between mortality level and number of survivors

Overcompensation was observed in the production of *A. aegypti*, *A. triseriatus*, and *C. pipiens* adults at some (*A. aegypti*) or all (*A. triseriatus* and *C. pipiens*) ranges of extrinsic mortality imposed, whereas *A. albopictus* cohorts demonstrated compensatory, followed by partial compensation. Our first prediction of our first hypothesis – that overcompensation would occur at low-to-intermediate levels of extrinsic mortality – was supported in *A. aegypti*, *A. triseriatus,* and *C. pipiens*, but not *A. albopictus*. The partial compensation at the 70% extrinsic mortality level in *A. albopictus* trended towards supporting our second prediction, but we did not see support for this prediction in the other three species. Mortality imposed by both predation and artificial harvest induced overcompensation in *A. aegypti, A. triseriatus,* and, *C. pipiens*, supporting our third prediction, but not in *A. albopictus*. Because all three predictions were not confirmed for any single species, we did not find full support for our first hypothesis. Our results demonstrated three patterns in responses to mortality: 1) overcompensation induced at all levels of extrinsic mortality (seen in *A. triseriatus* and *C. pipiens* data), 2) overcompensation

induced at low-to-intermediate levels of extrinsic mortality followed by compensation above a threshold mortality level (seen in *A. aegypti* data), and 3) compensation induced at low-to-intermediate levels of extrinsic mortality followed by partial compensation above a threshold (seen in *A. albopictus* data). The variation in patterns between species is likely a result of differences in responses to density (see 'effects of competitive ability on overcompensation' section).

Mortality by predation versus artificial harvest

Mortality by predation led to greater adult production than did artificial mortality in *A. aegypti* and *A. albopictus*. Artificial mortality in this experiment was imposed by randomly removing individuals from the containers, resulting in the complete loss of biomass from the harvested individuals. However, predation by *M. longisetus* releases feces and unconsumed body parts to the system, potentially enhancing resources for the microbial communities on which mosquitoes feed, which may explain the greater numbers of adults produced in predator treatments. Furthermore, the selectivity of the artificial and predator-induecd mortalities likely influenced the difference in adult production as well. Individuals were selected at random for removal in artificial mortality treatments, while lower-quality individuals (e.g., smaller, slower, less robust) were likely to be consumed in the predator treatments. If this selective removal of less robust larvae occurred with real predation, it may have resulted in higher-quality survivors than did the random removal treatments, with correspondingly greater likelihood of survival to adulthood.

In contrast to *A. aegypti* and *A. albopictus*, fewer *C. pipiens* adults were produced when exposed to predation than to artificial mortality. *Culex pipiens* has a longer larval development period than the three *Aedes* species used in this experiment. Since *M. longisetus* is a size-

selective predator, primarily feeding on 1st and 2nd instar larvae (Marten and Reid 2007), this longer development time would result in an increased duration of vulnerability to predation. Most larvae in *Aedes* treatments appeared to have grown sufficiently to have low risk of predation by day 2, when survivors were counted, and the percent mortality induced by predation was calculated. If fewer *C. pipiens* larvae reached this refuge by day 2, predation may have continued after the census of survivors. If predation continued beyond the point at which density-dependence became important, it would likely interact additively with intrinsic mortality (Abrams 2009, McIntire and Juliano 2018). This additive mortality may have counteracted overcompensation in predator containers, thus leading to a lower number of survivors induced by predation compared to artificial mortality.

Effects of competitive ability on overcompensation

Our second hypothesis – different species exhibit varying likelihoods of (over)compensatory mortality due to differential competitive abilities and responses to harvest – was supported. The extent of overcompensation differed among the three *Aedes* species in a pattern consistent with past studies examining their relative competitive abilities and responses to density. Under natural settings and in microcosms simulating natural settings, *A. albopictus* is the superior competitor of the three, followed by *A. aegypti*, and *A. triseriatus* is the weakest competitor (Barrera 1996, Grill and Juliano 1996, Juliano 1998, 2009, 2010, Braks et al. 2004, Reiskind and Lounibos 2009, Murrell and Juliano 2012, O'Neal and Juliano 2013), and competitive abilities are determined by the a species' ability to persist at low per capita resource levels (Tilman 1982, Chase and Leibold 2003, Murrell and Juliano 2012). Because overcompensation occurs when extrinsic mortality releases a surviving population from some level of density-dependent effects, stronger density-dependent regulation of the population

should be associated with greater levels of overcompensation, or a greater range of conditions leading to overcompensation. *Culex pipiens* is the weakest competitor of the four species (Peters et al. 1969, Carrieri et al. 2003, Costanzo et al. 2005); however, because of the different food regimes used for the *C. pipiens* and *Aedes* experiments, we are not confident of comparability of the responses of the two genera.

The patterns observed in the responses to mortality between the four species suggest competitive ability and associated strengths of density-dependent effects regulating a population may influence which of the three alternative hypotheses previously described occurs in response to extrinsic mortality. This would indicate the likelihood of (over)compensation differs not only between species, but also with variation in per capita resource levels among conspecific populations. Thus, raising the strength of density-dependent effects in this experiment by reducing resource levels or increasing cohort sizes may be expected to lead to greater levels of overcompensation. Whereas *A. albopictus* did not show overcompensation in this experiment, we predict decreasing the per capita resource level would increase the likelihood of overcompensation in response to mortality. McIntire and Juliano (2018) conducted an experiment with *A. albopictus* with the same resource levels and nearly the same level of random mortality on day 2 using a cohort size of 250 larvae. They found strong overcompensation in *A. albopictus* at 48 % mortality, which is consistent with our expectation that per capita resource levels and population density are strong determinants of likelihood and strength of overcompensation, at least for *A. albopictus.* Further experimentation is necessary to understand the general relationship between strength of density-dependent effects and (over)compensation.

The shift to partial above a threshold in *A. albopictus* is consistent with the findings in Sandercock et al. (2011) and Pardini et al. (2009), as well as past theoretical models (Boyce et al.

1999, Ratikainen et al. 2008, Abrams 2009, Zipkin et al. 2009). The absence of this result in our *A. aegypti, A. triseriatus*, and *C. pipiens* data suggests the range of extrinsic mortality included in this study was not sufficiently high to induce additive mortality. The level of mortality that serves as the threshold for additive mortality is predicted to be determined by the proportion of mortality due to density-dependent effects in the absence of extrinsic mortality (Sandercock et al. 2011). Populations exhibiting higher density-dependent mortality levels in the absence of extrinsic mortality would demonstrate a higher threshold for extrinsic mortality to become additive (Sandercock et al. 2011). Our findings support this prediction, as only the species believed to be the best competitor and postulated to have the lowest level of density-dependent mortality (*A. albopictus*) demonstrated a threshold to additive mortality within the range of extrinsic mortalities included in our study. According to the level of density-dependent mortality that occurred at the 0% mortality level, *A. albopictus* would be predicted to exhibit a threshold to additivity at approximately 59%, which is close to the 50% threshold demonstrated here. Mortality would be predicted to become additive in *A. aegypti* at the 62% mortality level, which we did not see. However, the drop to compensation at the 70% mortality level suggests the additive threshold was not much higher. *Aedes triseriatus* and *C. pipiens* would be predicted to exhibit thresholds to additive mortality at 88% and 97%, respectively, which exceed the mortality range tested in this study. Decreasing the density-dependent mortality of the other three species by increasing the per-capita resource levels may lower the threshold for additive mortality to exist within the 0-70% mortality range used here, but further experimentation is needed to explore this possibility.

Practical implications

Determining the mechanisms by which overcompensation and the hydra effect can occur is critical for effectively managing pest populations. Interventions aimed at control of density-dependent populations can lead to counter-productive results (Agudelo-Silva and Spielman 1984, Buckley et al. 2001, Pardini et al. 2009). The four mosquito species used in this study are important vectors of mosquito borne viruses (Eldridge et al. 2000) and are targets of control efforts within their ranges. The results of this study demonstrate the potential importance of using pest-control strategies that minimize the risk of imposing mortality levels that can be offset by a release from density-dependence. To avoid inducing (over)compensation, control strategies must result in sufficiently high levels of mortality to surpass the threshold for additive mortality demonstrated by the target population. *Mesocyclops longisetus* has been successfully used as a biocontrol agent of several mosquito species, including the three *Aedes* species tested in this study (Marten et al. 1994, Soumare and Cilek 2011). Our results suggest the success of *M. longisetus* at reducing mosquito population sizes is not only dependent on its predation rate but is also likely to depend on its numerical response. Inoculations of *M. longisetus* to mosquito habitats must include large numbers of copepods and provide a suitable environment for maintenance of a large copepod population to avoid overcompensation in mosquito adult production.

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Tables

Table 1.1 Mixed effects generalized linear models testing survivorship in *A. albopictus*. Early-instar larvae were exposed to a range of mortality from harvest or predation (pred).

Model Effects	AICc	ΔAICc	exp(-.5*ΔAICc)	w_i[a]	E[a]	w_i[b]	E[b]
Pred	308.91	0.00	1.00	0.24	1.00	0.34	1.00
Pred mortality $mortality^2$ $mortality^3$	309.11	0.20	0.90	0.21	1.11	0.31	1.11
Pred mortality	310.04	1.13	0.57	0.14	1.76	0.19	1.76
Pred mortality $mortality^2$	310.34	1.43	0.49	0.12	2.04	0.17	2.04
None	310.69	1.78	0.41	0.10	2.44		
Mortality	311.33	2.42	0.30	0.07	3.35		
Pred mortality $mortality^2$ $mortality^3$ mortality*pred	312.17	3.26	0.20	0.05	5.10		
Pred mortality mortality*pred	312.41	3.50	0.17	0.04	5.75		
Pred mortality $mortality^2$ pred*mortality	313.23	4.32	0.12	0.03	8.67		
Pred mortality $mortality^2$ $mortality^3$ mortality*pred $mortality^2$*pred	315.53	6.62	0.04	0.01	27.39		
Pred mortality $mortality^2$ $mortality^3$ mortality*pred $mortality^2$*pred $mortality^3$*pred	317.10	8.19	0.02	0.00	60.04		
Sum	-	-	4.21	-	-		

Note: Weight of evidence (w_i) is calculated as exp(-0.5*AICc)/∑exp(-0.5*ΔAICc) and estimates the probability the model is correct. The evidence ratio (E) is calculated as $w_{(max)}/w_i$ and expresses how much more likely the best model is than the given one. Higher values indicate a less likely model.

[a] indicates w_i and E values when all models are included in calculations.

[b] indicates w_i and E values when only the 4 models incorporated in the model average prediction are included in calculations.

Table 1.2 Parameter estimates for the four models used to calculate model-averaged prediction values for *A. albopictus*.

Model Effects	Intercept	p>0	Pred	p>0	Mortality	p>0	Mortality^2	p>0	Mortality$_3$	p>0
Pred	39.53	**0.00**	7.19	**0.04**	-	-	-	-	-	-
Pred mort	42.19	**0.00**	7.14	0.05	0.00	0.22	-	-	-	-
Pred mort mort^2	39.50	**0.00**	3.74	0.29	0.01	0.25	0.00	0.12	-	-
Pred mort mort^2 mort^3	43.34	**0.00**	6.26	0.11	-0.02	0.16	0.00	0.08	0.00	**0.04**

Note: Values were produced in a mixed-effects generalized linear model testing the effects of predator (pred) and mortality up to the cubic term on survivorship.

Table 1.3 Mixed effects generalized linear models testing survivorship in *A. aegypti*. Early-instar larvae were exposed to a range of mortality from harvest or predation (pred).

Model Effects	AICc	ΔAICc	exp(-.5*ΔAICc)	w_i	E
Pred mortality $mortality^2$ $mortality^3$	377.39	0.00	1.00	0.42	1.00
Pred mortality	378.90	1.51	0.47	0.20	2.13
Pred mortality mortality*pred	379.98	2.59	0.27	0.12	3.65
Pred mortality $mortality^2$ $mortality^3$ mortality*pred	380.07	2.68	0.26	0.11	3.82
Pred mortality $mortality^2$	381.46	4.07	0.13	0.05	7.65
Pred mortality $mortality^2$ $mortality^3$ mortality*pred $mortality^2$*pred	382.51	5.12	0.08	0.03	12.94
Pred mortality $mortality^2$ pred*mortality	382.65	5.26	0.07	0.03	13.87
Mortality	382.81	5.42	0.07	0.03	15.03
Pred mortality $mortality^2$ $mortality^3$ mortality*pred $mortality^2$*pred $mortality^3$*pred	384.59	7.20	0.03	0.01	36.60
Pred	391.89	14.50	0.00	0.00	1408.10
None	395.62	18.23	0.00	0.00	9090.63
Sum	-	-	2.38	-	-

Note: Weight of evidence (w_i) is calculated as exp(-0.5*AICc)/∑exp(-0.5*ΔAICc) and estimates the probability the model is correct. The evidence ratio (E) is calculated as $w_{(max)}/w_i$ and expresses how much more likely the best model is than the given one. Higher values indicate a less likely model.

Table 1.4 Mixed effects generalized linear models testing survivorship in *C. pipiens*. Early-instar larvae were exposed to a range of mortality from harvest or predation (pred).

Model Effects	AICc	ΔAICc	exp(-.5*ΔAICc)	w_i	E
Pred mortality mortality^2 mortality^3	294.08	0.00	1.00	0.30	1.00
Pred mortality mortality*pred mortality^2 mortality^3	295.68	1.60	0.45	0.13	2.23
pred mortality mortality*pred mortality^2	295.85	1.77	0.41	0.12	2.42
Pred mortality mortality*pred mortality^2 mortality^2*pred mortality^3 mortality^3*pred	296.18	2.10	0.35	0.10	2.86
pred mortality	296.25	2.17	0.34	0.10	2.96
pred mortality mortality*pred mortality^2 mortality^2*pred	297.23	3.15	0.21	0.06	4.83
pred mortality mortality*pred mortality^2 mortality^2*pred mortality^3	297.33	3.25	0.20	0.06	5.08
mortality	297.76	3.68	0.16	0.05	6.30
pred mortality mortality*pred mortality^2 mortality^2*pred mortality^3	297.78	3.70	0.16	0.05	6.36
mortality mortality^2 mortality^3	300.83	6.75	0.03	0.01	29.22
Pred	301.64	7.56	0.02	0.01	43.82
None	303.32	9.24	0.01	0.00	101.49
Sum	-	-	3.34	-	-

Note: Weight of evidence (w_i) is calculated as exp(-0.5*AICc)/$\sum$exp(-0.5*ΔAICc) and estimates the probability the model is correct. The evidence ratio (E) is calculated as $w_{(max)}/w_i$ and expresses how much more likely the best model is than the given one. Higher values indicate a less likely model.

Table 1.5 Mixed effects generalized linear models testing survivorship in *A. triseriatus*. Early-instar larvae were exposed to a range of mortality from harvest or predation (pred).

Model Effects	AICc	ΔAICc	exp(-.5*ΔAICc)	w_i	E
Mortality	420.61	0.00	1.00	0.54	1.00
Pred mortality	422.48	1.87	0.39	0.21	2.55
Pred mortality mortality^2	424.23	3.62	0.16	0.09	6.11
Pred mortality mortality*pred	424.48	3.87	0.14	0.08	6.92
Pred mortality mortality^2 pred*mortality	426.48	5.87	0.05	0.03	18.82
Pred mortality mortality^2 mortality^3	426.62	6.01	0.05	0.03	20.19
Pred mortality mortality^2 mortality^3 mortality*pred	428.43	7.82	0.02	0.01	49.90
Pred mortality mortality^2 mortality^3 mortality*pred mortality^2*pred	429.34	8.73	0.01	0.01	78.65
Pred mortality mortality^2 mortality^3 mortality*pred mortality^2*pred mortality^3*pred	429.97	9.36	0.01	0.01	107.77
None	434.93	14.32	0.00	0.00	1286.91
Pred	437.22	16.61	0.00	0.00	4044.04
Sum	-	-	1.85	-	-

Note: Weight of evidence (w_i) is calculated as exp(-0.5*AICc)/∑exp(-0.5*ΔAICc) and estimates the probability the model is correct. The evidence ratio (E) is calculated as $w_{(max)}/w_i$ and expresses how much more likely the best model is than the given one. Higher values indicate a less likely model.

Table 1.6 Parameter Estimates from *A. Aegypti, A. Triseriatus*, and *C. Pipiens* models.

A. aegypti					
Effect	**Estimate**	**Std Er**	**DF**	**t Value**	**Pr > |t|**
Intercept	3.7286	0.1168	4	31.93	**<.0001**
Pred -	-0.1502	0.06018	43	-2.5	**0.0165**
Pred +	0	.	.	.	.
Mortality	-0.01939	0.01198	43	-1.62	0.1128
Mortality2	0.000873	0.000376	43	2.32	**0.0249**
Mortality3	-8.16E-06	0	43	-2.7	**0.0099**
A. triseriatus					
Intercept	2.4634	0.1948	4	12.65	**0.0002**
Mortality	0.01327	0.002984	50	4.45	**<.0001**
C. pipiens					
Intercept	0.3699	0.313	4	1.18	0.3027
Pred -	0.8311	0.2416	43	3.44	**0.0013**
Pred +	0	.	.	.	.
Mortality	0.114	0.01701	43	6.7	**<.0001**
Mortality2	-0.00339	0.000239	43	-14.14	**<.0001**
Mortality3	0.000027	0	43	2.15	**0.0374**

Figure

Figure 1.1 Observed and predicted numbers of mosquito survivors across a mortality range. Artificial mortality was applied to no predator treatments (circles) on day 2 by randomly removing 0, 10, 30, 50, or 70% of the initial cohort size (150) on day 2. Predator treatments (triangles) received 1, 2, or 3 female *M. longisetus* at the start of the experiment, and the percent mortality by day 2 was determined by counting the number of surviving mosquito larvae. Predicted lines for the no predator (solid line) and predator (dashed line) treatments plot the function produced by a mixed-effects generalized linear model. Predicted values were calculated using the models with the lowest AICc's for the *A. aegypti, A. triseriatus,* and *C. pipiens* data. The predicted values for *A. albopictus* were calculated by using model weights to average the four models with the lowest AICc's. The effect of predator was significant for all species except *A. triseriatus.*

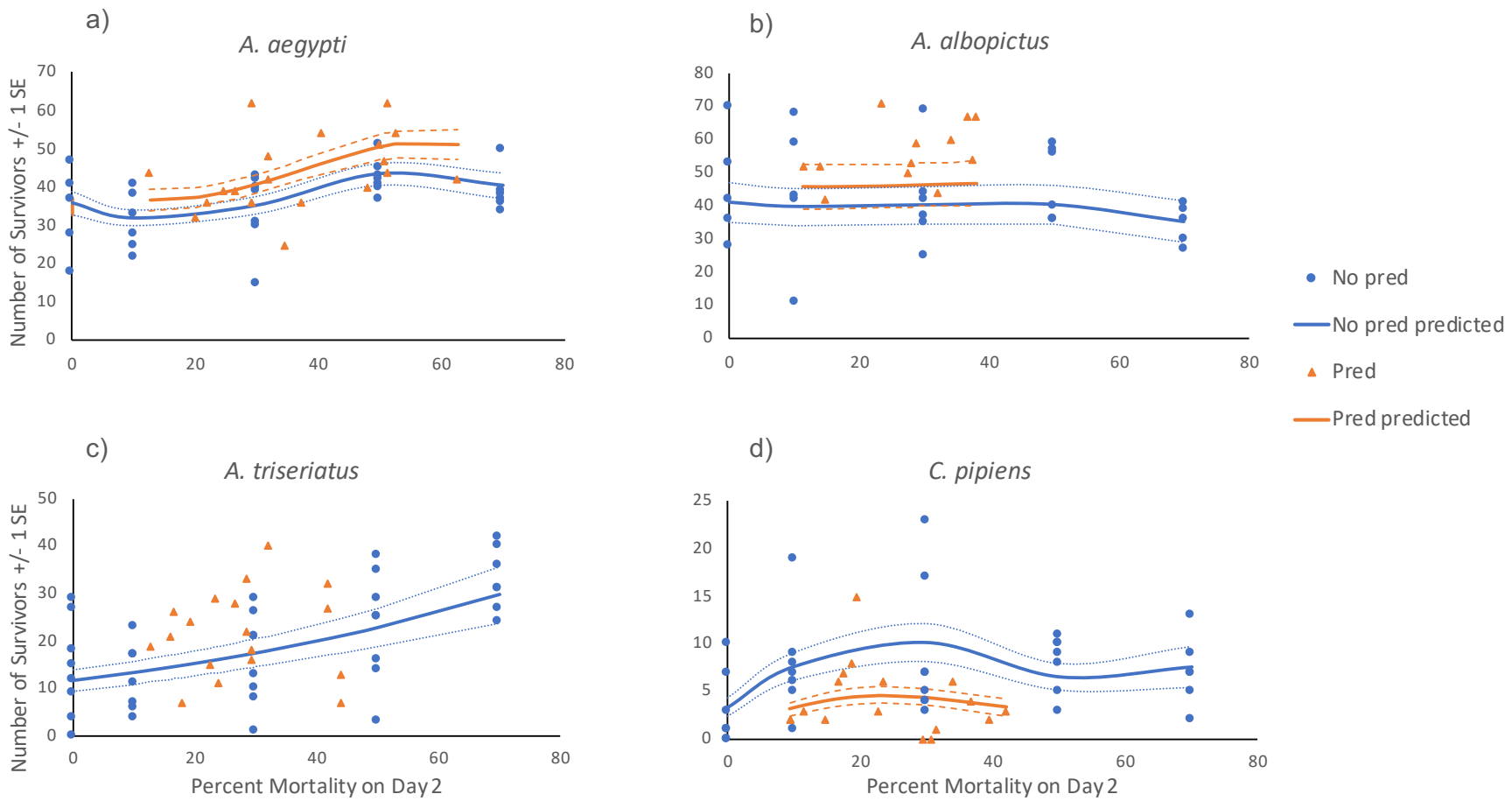
a)
A. aegypti
Number of Survivors +/- 1 SE
b)
A. albopictus
c)
A. triseriatus
Number of Survivors +/- 1 SE
Percent Mortality on Day 2
d)
C. pipiens
Percent Mortality on Day 2
No pred
No pred predicted
Pred
Pred predicted

Figure 1.1

CHAPTER II: THE ENEMY OF MY COMPETITOR IS MY FRIEND

Abstract

Population responses to extrinsic mortality can lead to no change in population size (compensation) or an increase in population size (overcompensation) when the population is negatively density dependent. This intriguing response has been the subject of various theoretical studies, but few experiments have explored the how ecological context may modify the phenomenon. The purpose of this study is to test the ability for three species of predators alone to induce (over)compensation on a prey population and to compare predation from single species to predation from a functionally diverse predator assemblage, which can lead to nonlinear effects on prey populations known as emergent multiple predator effects (MPEs). Larval *Aedes aegypti* (Diptera: Culicidae) were exposed to predation by *Mesocyclops longisetus* (Crustacea: Copepoda)*, Anopheles barberi* (Diptera: Culicidae)*, Corethrella appendiculata* (Diptera: Corethrellidae), or all three. The total number of survivors to adulthood, the numbers of males and females, as well as a composite index of performance r' were separately analyzed in ANOVAs. Predator treatment did not have a significant effect on survivorship across sexes, suggesting mortality by predation was compensatory, as it did not result in a change in the number of adults produced. However, the overall effect of predation on the number of female survivors was significant, in contrast to the effect on males. Predator treatment had a significant effect on r' with predation yielding a higher r' than the no-predator control. This suggests that, while predation did not lead to significantly greater production of adults, it did release survivors from sufficient levels of density-dependent effects to raise the population equilibrium, a phenomenon that has been coined the 'hydra effect.' We did not find evidence for emergent MPEs, as the diverse predator treatment was not significantly different from the single-species

treatments. This study serves as one of the first empirical examples of predation yielding the hydra effect, a phenomenon that is predicted to occur across many taxa and food webs.

Keywords: Hydra effect; MPEs, compensation, index of performance, predator-prey

Introduction

Extrinsic mortality (*e.g.*, due to natural enemies, harvesting, or other human interventions) impinging on populations has traditionally been predicted to interact additively with intrinsic mortality sources, with greater levels of extrinsic mortality leading to reductions in population densities. However, populations regulated by negatively-density dependent effects may demonstrate counter-intuitive responses. By initially reducing the population density, extrinsic mortality may reduce detrimental density-dependent effects on the survivors. This may result in the production of the same number (compensation) or a greater number (overcompensation) of individuals surviving to the following life stage as would have been the case in the absence of extrinsic mortality. Extrinsic mortality that results in an increase in the equilibrium density of a population has been termed the 'hydra effect' (Abrams and Matsuda 2005).

Compensatory and overcompensatory responses to mortality have been demonstrated in both field and laboratory studies (Nicholson 1954, Agudelo-Silva and Spielman 1984, Washburn et al. 1991, Moe et al. 2002, Cameron and Benton 2004, Zipkin et al. 2008, Weber et al. 2016, Neale and Juliano, *in review*). Numerous theoretical studies have attempted to elucidate the mechanisms under which the phenomenon occurs (reviewed in Abrams 2009). The timing of extrinsic mortality relative to the onset of density-dependent effects is predicted to influence the likelihood of overcompensation, with mortality occurring prior to density-dependence postulated to lead to overcompensation and increased population sizes (Jonzen and Lundberg 1999, Abrams

2009, Pardini et al. 2009). This hypothesis, known as the 'temporal separation of mortality and density dependence hypothesis', was recently supported in an empirical study on container mosquitoes (McIntire and Juliano 2018). Furthermore, the extrinsic mortality rate (i.e., proportion killed) is expected to have an effect on whether additive, compensatory, or overcompensatory effects are observed (Sandercock et al. 2011, Neale and Juliano, *in review*) and these effects appear to be related to competitive abilities of the species involved (Neale and Juliano, *in review*). We have found only a few published studies empirically examining the mechanisms of the hydra effect (Sandercock et al. 2011, McIntire and Juliano 2018), and more empirical studies are needed to determine the conditions under which it occurs.

Predation is a common source of extrinsic mortality for animal populations in the wild, and mathematical models predict that predation can lead to the hydra effect in prey populations (Cortez and Abrams 2016). However, only two of the aforementioned empirical examples included predation as a mortality source, both in container mosquito systems (Nannini and Juliano 1998, Neale and Juliano, *in review*). In natural food webs, many prey populations face predation from multiple predators (Sih et al. 1998). Understanding how predation by multiple predator species differs from a single predator species in hydra effect studies is critical to predicting the occurrence of the phenomenon in nature. However, we have found no published studies examining the effects of multiple predators on overcompensation and the hydra effect. Increasing predator functional or phylogenetic diversity can result in emergent multiple predator effects (MPE's), which are characterized by nonlinear effects (*i.e.,* risk reduction or risk enhancement) on prey populations, which often result in an increase or decrease in predation rates relative to that observed with single predators (Sih et al. 1998, Schmitz 2007, Bruno and Cardinale 2008, Greenop et al. 2018). A recent meta-analysis of studies on terrestrial arthropod

systems found predator functional diversity was more important in determining the outcome on prey than phylogenetic diversity (Greenop et al. 2018). The degree of overlap in foraging domains and hunting modes between the different predator functional groups is predicted to determine if emergent MPE's will occur and whether they will be risk enhancing or reducing, as foraging domain and hunting mode determine the likelihood of intraguild predation and availability of prey refugia (Schmitz 2007). Since the level of extrinsic mortality influences the likelihood of overcompensatory responses, emergent MPE's may result in levels of overcompensation (including absence of overcompensation) that deviate from responses to single predator species.

The purpose of this study is to test the ability of predators to induce compensation or overcompensation in a prey species. We hypothesize that predation from a single species occurring early in the development of a density-dependent prey population leads to overcompensatory mortality, and predation from multiple predator species leads to either increased or decreased strength of overcompensation due to emergent MPEs that alter mortality rates.

We tested our hypotheses using *Aedes aegypti* (Diptera: Culicidae) as the prey species. The complex life-cycle and negatively density dependent survival of the larval stage (Dye 1984) are consistent with the assumptions of the models of populations developed by Abrams (2009). Overcompensation has been demonstrated in this species (Neale and Juliano,*in review*) as well as its congeners, *A. sierrensis* (Washburn et al. 1991), *A. albopictus* (McIntire and Juliano 2018), and *A. triseriatus* (Neale and Juliano, *in review*). The predators we included were *Mesocyclops longisetus* (Crustacea: Copepoda), *Anopheles barberi* (Diptera: Culicidae), and *Corethrella appendiculata* (Diptera: Corethrellidae). All three predators have been demonstrated to be

efficient predators of *Aedes* larvae under similar conditions (Marten et al. 1994, Nannini and Juliano 1998, Alto et al. 2009). They are size-selective, feeding primarily on early instar larvae (Nannini and Juliano 1998, Soumare et al. 2004, Alto et al. 2009). This size-selectivity is ideal for inducing overcompensation, as it concentrates the mortality early in prey development and potentially separates mortality due to predation temporally from the density-dependent effects, which are expected to increase as immatures grow. The three predators differ in hunting domains. *Mesocyclops longisetus* swims throughout the water column, lunging at prey when it passes within ~ 1mm (Marten and Reid 2007). *Anopheles barberi* sits in the surface tension and ambushes larvae as they surface (Clements 1992). *Corethrella appendiculata* primarily sits at the bottom of the water column and preys on mosquito larvae when they browse in the substrate (Kesavaraju et al. 2007).

Material and Methods

Organism Collection

Aedes aegypti used in this study were from a laboratory colony originating from pupae and larvae field-collected from Vero Beach, FL approximately 1 year before the start of this experiment. To maintain the colony, larvae were reared in plastic pans at 25°C and provided bovine liver powder. Adults were given a constant supply of 20% sucrose solution, and blood meals were provided from anesthetized guinea pigs (IUCAC# 842043).

Mesocyclops longisetus were from a laboratory colony maintained at Illinois State University in Normal, IL, which originated from a colony maintained at the Florida Medical Entomology Laboratory (FMEL) in Vero Beach, FL. *Corethrella appendiculata* were 4th instars field collected from tree holes on the FMEL grounds. Larvae were housed in water from the tree holes at 25°C until the start of the experiment. *Anopheles barberi* were collected as larvae in

rain-filled buckets at Parklands Merwin Nature Preserve near Lexington, IL. To maximize the number of late-instar larvae available at the start of the experiment, 3rd and 4th instars were housed at 22°C to delay pupation, while 1st and 2nd instars were housed at 25°C.

Experimental Setup

Four days prior to the beginning of the experiment, 500 ml plastic containers were filled with 400 ml ultrapure water, 1 g dried live oak (*Quercus virginiana*) leaves collected from Vero Beach, FL, 0.05 g dried decorated crickets (*Gryllodes sigillatus*) from a colony maintained at Illinois State University, and 100 µl microbial inoculum, from rain-filled buckets in Merwin Nature Preserve, Lexington, IL. Lids were placed on the containers with holes punched for ventilation. The containers were housed in an environmental chamber at 25°C until the beginning of the experiment to allow the establishment of a microbial community to serve as food resources for mosquito larvae.

Aedes aegypti eggs were hatched 24 hours prior to the start of the experiment by placing strips of egg papers in 4 dram glass vials containing 0.4g/l Difco™ nutrient broth (Becton, Dickinson and Company, Sparks, MD) at 25°C. At the start of the experiment, hatchling larvae were rinsed in ultrapure water and 150 were placed in each experimental container (n=15). Containers were randomly assigned one of five predator treatments: no predator, *M. longisetus*, *C. appendiculata*, *A. barberi*, and diverse. The single-species treatments received three predator individuals, whereas the diverse treatment received one individual of each predator species. Only non-gravid adult female *M. longisetus*, 4th instar *C. appendiculata*, and 3rd and 4th instar *A. barberi* were used. Multiple *A. barberi* instars were included because of a limited number of larvae available. Since 4th instars consume greater numbers of *Aedes* prey than 3rd instars (Nannini and Juliano 1998), the *A. barberi* treatment received one 4th and two 3rd instars, and

diverse containers each received a 4th instar. Once prey and predators were added to each container, they were placed in an environmental chamber set to 25°C and a 14:10 light:dark photoperiod.

Containers were checked daily for *A. aegypti* pupae and survival of predators. *A. aegypti* pupae were removed, placed in 0.25 dram vials with cotton stoppers, and returned to the environmental chamber, and any dead or missing predators were replaced. All predators were removed on day 6 because the replacement stock of *A. barberi* larvae was depleted. Due to the size selectivity of the three predators and the developmental stage of prey by day 6, only minimal amounts of predation would have occurred if the predators remained. On days 16 and 30 0.5 g dried live oak leaves and 0.025g dried decorated crickets were added to replenish resources for bacteria and fungi that are the food of *A. aegypti*.

Pupae were checked daily for eclosion. Water was removed from vials containing adults and the vial was placed in a drying oven at 70°C for >48 hours. All individuals reaching adulthood were counted as survivors. Female wings were dissected and photographed with a digital camera, and wing lengths were measured in Image J 1.51.

Index of Performance

Using data collected on female survivorship to adulthood, development time to adulthood, and predicted fecundity based on body size, Livdahl and Sugihara's (1984) index of performance r' was calculated for each container (Equation 1). This index synthesizes information on these variables in a manner analogous to calculations of net reproductive rate (R_0) and cohort generation time (T_c) from a cohort life table. This index of performance provides an estimate of cohort rate of change and was used to assess how predator treatments affected population growth for experimental cohorts in each container. We infer that cohorts of 150

larvae receiving a particular predator treatment are farther below equilibrium density if their index of performance is farther above 0.

Equation 1 Livdahl and Sugihara's (1984) index of performance

$$r' = \left[\frac{\ln\left[(1/N_0) \sum_x A_x f(w_x) \right]}{D + \left[\sum_x x A_x f(w_x) \Big/ \sum_x A_x f(w_x) \right]} \right]$$

N_0 is the initial number of females (assumed to be 50% of the initial 150 larvae), A_x is the number of new females emerging on day x, w_x is the mean wing length of new females emerging on day x, and D is the estimated days between female eclosion and oviposition, (estimated to be 12 days; Grill and Juliano 1996). Production of female offspring $f(w_x)$ was estimated as a function of wing length using the regression provided by Briegel (1990): $f(w_x) = 0.5(2.5w_x^3 - 8.616)$.

Statistical Analysis

One-factor ANOVA's were used to analyze the effects of predator treatment on overall survivorship, female survivorship, male survivorship, r', female size, and female development time using PROC GLM in SAS 9.4. Contrast statements were used as a *post hoc* tests for the analyses of index of performance, female survivorship, and female development time, and sequential Bonferroni methods were used to correct for multiple comparisons (Holm 1979). The contrasts we tested were predator versus no predator, single-predator versus diverse, and pairwise comparisons of each of the three single-predator treatments.

Results

The no predator treatment produced the lowest number of survivors across both sexes, but the overall treatment effect was not significant ($F_{4,14}$=2.82, p=0.0838, Figure 2.1). The effect of predator on the number of adult males produced was not significant ($F_{4,14}$=0.72, p=0.5994), but the effect on the number female adults produced was significant ($F_{4,14}$=4.03, p=0.0337). However, none of the *post hoc* contrasts produced significant differences after correcting for multiple tests (Table 2.1, Figure 2.1).

The overall ANOVA on r' values indicated a significant effect of predator treatment ($F_{4,14}$=4.24, p=0.029). No predator treatments produced the lowest value of r' at 0.0107, whereas *C. appendiculata* produced the largest r' at 0.0438 (Figure 2.2). *Post hoc* analyses indicated predation led to an increase in r' compared to no predation (Table 2.1, Figure 2.2). Predation by a single predator treatment was not significantly different from the diverse treatment, and there were no significant pairwise comparisons among the three single-predator treatments (Table 2.1).

Predation from *M. longisetus* produced the largest average female wing length, but the overall treatment effect was not significant ($F_{4,14}$=2.17, p=0.1457, Figure 2.3a). The predator treatment had a significant effect on the average number of days to adulthood for females ($F_{4,14}$=3.56, p=0.0469), with the average time to adulthood in predator treatments significantly lower than that in control (Table 2.1, Figure 2.3b).

Discussion

The absence of a significant differences in adult production among the predator treatments and the control indicates mortality from predation induced compensation in the *A. aegypti* cohorts. We did not observe significant overcompensatory mortality in any treatment, and the adult production in the diverse predator treatment was not significantly different from

any single-species predator treatment; therefore, our results do not support our hypothesis. The compensatory response suggests predation removed individuals that would have otherwise died from density-dependent effects, but this removal did not release the surviving population from a sufficient level of these density-dependent effects to increase significantly production of adults.

Predation led to a significantly larger index of performance (r'), suggesting that predation had the counter-intuitive effect of increasing equilibrium compared to the no-predator treatment (Livdahl and Sugihara 1984). The overall F test on female survivorship was significant, and female adult production trended to be greater with predation, but this contrast was marginally non-significant. This may be explained by insufficient power to detect differences due to the small sample size. The differential effects of predation on male versus female survivorship is consistent with contrasts in resource requirements between the two sexes. Females require more time to reach adulthood and emerge as larger adults, indicating they have larger resource demands than males (Wormington and Juliano 2014a, 2014b). Females would thus receive a greater benefit to conspecific mortality, as they are the most resource limited.

All three components of the index of performance – survivorship, development time, and fecundity – displayed trends consistent with the differences in r'. Predation led to higher mean number of survivors, higher mean fecundity, and lower mean development time. However, since the effect of predation on female development time was the only one that led to significant differences in *post hoc* comparisons between predator versus no predator, the greater population equilibrium density in response to extrinsic mortality was primarily mediated by females reaching adulthood faster in the presence of predation. The faster development of females was likely caused by the weakening of density-dependent effects by reductions in population density. The difference in r' suggests that equilibrium population densities would be greater when

exposed to predation compared to no predator if the experimental populations were allowed to persist for multiple generations.

Overcompensation has been induced in *A. aegypti* by predation from *M. longisetus* (Neale and Juliano, *in review*). However, the present study failed to produce the same result. The relatively small sample size may limit our power to detect differences among treatments despite the tendency for predator treatments to produce more adults, particularly more females (Figure 2.1). Furthermore, the *A. aegypti* population tested in the past study originated in New Orleans, LA, while the population tested in this study originated from Vero Beach, FL. Population-level differences in intraspecific competitive abilities and responses to predation may have influenced the contrasting results in the two experiments, as well as differences in exposure to predation in the two experiments. The compensatory response to *A. barberi* predation is consistent with Nannini and Juliano (1998), in which predation by *A. barberi* induced compensation in *A. triseriatus*, a congener to the *A. aegypti* tested in this study. However, comparisons of (over)compensatory responses between species should be made with caution, as differences in responses to population density and competitive abilities can lead to interspecific variation in the level and likelihood of (over)compensation (Neale and Juliano, *in review*).

The single-species predator treatments produced the same response as the diverse predator treatment; therefore, we did not find evidence for emergent MPE's. Our results indicate the effects of the three predators were substitutable. Predator substitutability is predicted to occur when the predators exhibit non-overlapping habitat domains and prey exhibit broad habitat domains (Schmitz 2007). However, this prediction may be complicated when prey demonstrate predator-specific avoidance behaviors, which may result in risk enhancement by predator facilitation. In our experiment, habitat domains for *A. barberi* and *C. appendiculata* have little

or no overlap, but the domains of each overlap with the uppermost (*A. barberi*) and lowermost (*C. appendiculata*) portions of the domain for *M. longisetus*, which hunts throughout the entire water column (Clements 1992, Kesavaraju et al. 2007, Marten and Reid 2007). Scenarios in which the habitat domains of multiple predators overlap are predicted to lead to emergent MPE's, the nature of which depend on the respective hunting modes of the predators and the degree of overlap with prey habitat domain (Schmitz 2007). However, since the degree of overlap between *M. longisetus* and either of the other two predators is small, the chances for interactions between predators may have been minimal. We found no evidence of intraguild predation, one mechanism which can lead to risk reduction when predator habitat domains overlap (Schmitz 2007). Habitats with shorter water columns would compress the habitat domain of *M. longisetus* and increase the proportion of overlap with *C. appendiculata* and *A. barberi*, thus increasing the likelihood of predator interactions and emergent MPEs.

We have demonstrated predation on larval *A. aegypti* by three predator species, alone and in polyculture, can induce compensation in the production of adults. Our evidence suggests predation may relieve *A. aegypti* populations of a sufficient level of density-dependent effects to increase population equilibrium and induce the hydra effect. Since this effect is predicted to occur in a variety of food web structures, these results provide insight on a phenomenon that affects many taxa (Cortez and Abrams 2016). Further work should be conducted to elucidate the mechanisms mediating (over)compensation and the hydra effect to better predict their occurrence in nature, allowing more effective pest management, conservation, and harvest strategies (Abrams 2002, Ratikainen et al. 2008, Zipkin et al. 2009, Sandercock et al. 2011).

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Table

Table 2.1 *Post hoc* tests comparing treatment effects on *r′,* female adult production, and female development time. Contrast statements compared effects predator versus no predator, single predator versus diverse, and pairwise comparisons of individual species treatments on *A. aegypti* index of performance, number of female adults produced, and female days to adulthood. Bold p values indicate significant comparisons after correcting for multiple comparison.

Table 2.1

Index of performance (r')			
Contrast	DF	F Value	Pr > F
Predator vs no predator	1	12.1	**0.0059**
Single predator vs diverse	1	0.06	0.8123
A. barberi vs *C. appendiculata*	1	4.81	0.053
M. longisetus vs *C. appendiculata*	1	1.4	0.2635
A. barberi vs *M. longisetus*	1	1.02	0.3371
Number of female adults			
Predator vs no predator	1	7.78	0.0192
Single predator vs diverse	1	0.01	0.9153
A. barberi vs *C. appendiculata*	1	7.14	0.0234
M. longisetus vs *C. appendiculata*	1	5.16	0.0464
A. barberi vs *M. longisetus*	1	0.16	0.6969
Female days to adulthood			
Predator vs no predator	1	11.71	**0.0065**
Single predator vs diverse	1	1.97	0.1902
A. barberi vs *C. appendiculata*	1	0.55	0.4736
M. longisetus vs *C. appendiculata*	1	0.07	0.7987
A. barberi vs *M. longisetus*	1	0.23	0.6396

Figures

Figure 2.1 Mean numbers of adult *A. aegypti* produced by predator treatment. Larvae were exposed to no predators, 3 *A. barberi*, 3 *M. longisetus*, 3 *C. appendiculata*, or 1 of each predator species.

Figure 2.2 Mean indices of performance (*r'*) by predator treatment. Cohorts of larvae were reared in the presence of no predators, 3 *A. barberi*, 3 *M. longisetus*, 3 *C. appendiculata*, or 1 of each predator species. Brackets indicate the significant difference between predator vs. no predator.

Figure 2.3 Mean female wing length (a) and days to adulthood (b) by predator treatment. Larval *A. aegypti* were reared in the presence of no predators, 3 *A. barberi*, 3 *M. longisetus*, 3 *C. appendiculata*, or 1 of each predator species. Brackets indicate the difference between predator vs. no predator.

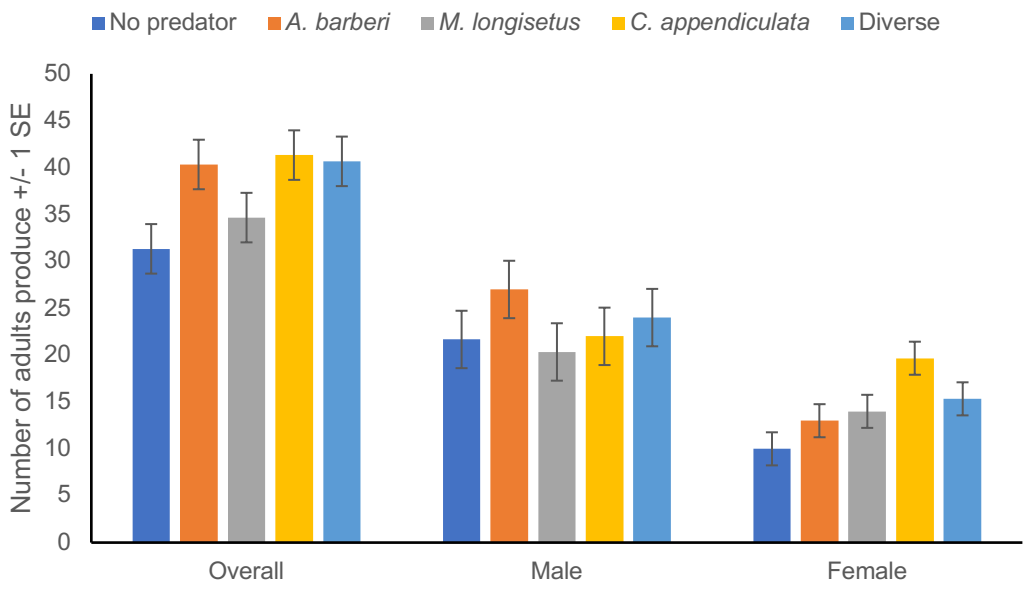
No predator
A. barberi
M. longisetus
C. appendiculata
Diverse
Number of adults produce +/- 1 SE
50
45
40
35
30
25
20
15
10
5
0
Overall
Male
Female

Figure 2.1

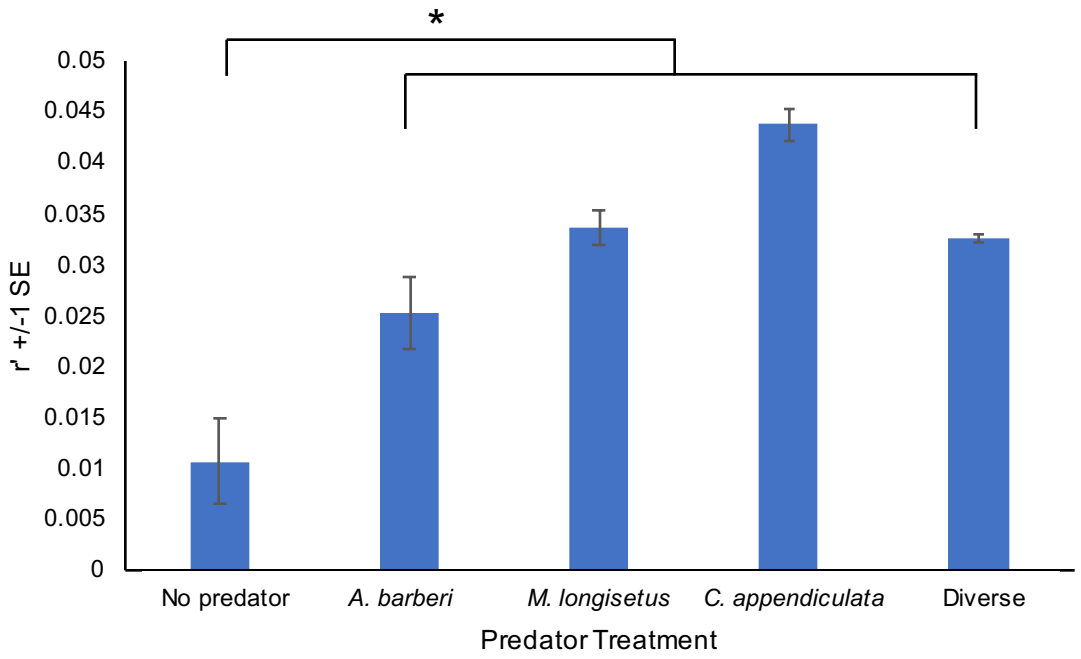
*
0.05
0.045
0.04
0.035
0.03
0.025
0.02
0.015
0.01
0.005
0
r' +/-1 SE
No predator
A. barberi
M. longisetus
C. appendiculata
Diverse
Predator Treatment

Figure 2.2

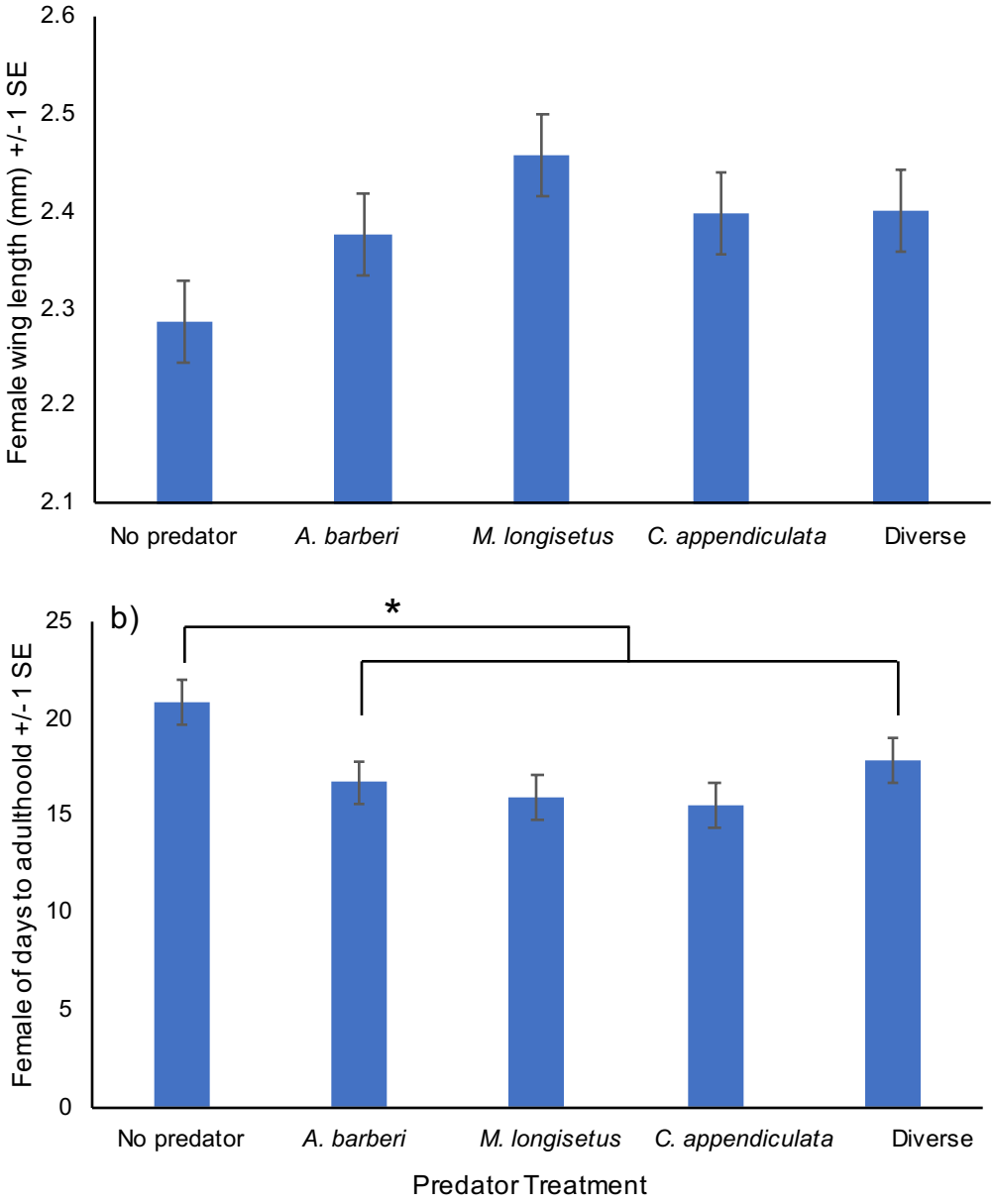
Female wing length (mm) +/- 1 SE
2.6
2.5
2.4
2.3
2.2
2.1
No predator
A. barberi
M. longisetus
C. appendiculata
Diverse
b)
*
Female of days to adulthood +/- 1 SE
25
20
15
10
5
0
No predator
A. barberi
M. longisetus
C. appendiculata
Diverse
Predator Treatment

Figure 2.3