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Can vertebrate predation alter aggregation of risk in an insect host–parasitoid system?

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Summary

1. Insect host–parasite systems allow investigations of the trophodynamics of ecological communities within a well-formed theoretical context. A little explored feature of such systems involves the interplay between generalized consumers and host–parasitoid dynamics. I report a study investigating how the impacts of generalized consumers, viewed here as interaction modifications, may influence the stability of a particular interspecific interaction.

2. In a study involving overwintering oothecae of the praying mantis *Stagmomantis limbata* (Hahn), birds damaged 36% of oothecae, 85% of which had also been parasitized by winter-active, multivoltine torymid wasps of the genus *Podagrion*. Birds preferentially preyed upon oothecae oviposited high on trees, but such predation was often incomplete, leaving both viable and parasitized mantid eggs inside damaged oothecae.

3. Two factors allowed bird damage to influence the distribution of risk of parasitism among oothecae and among hosts. These were (1) that some parasites – but no mantids – emerged successfully prior to bird predation and (2) that extensive post-damage foraging by parasitoids occurred, but that the nature of this foraging was altered little by oothecal damage.

4. In all, bird damage engendered a fourfold increase (from 1·5 to 6·5) in the CV^2 of parasitism risk among hosts (a stability criterion that has been proposed for host–parasitoid interactions) and increased the relative importance of host-density-dependent parasitism. The role of timing of the two natural enemy impacts for stability of the host–parasitoid interaction is discussed.

Key-words: aggregation of risk, *Aphelocoma*, $CV^2 > 1$ rule, Mantidae, Mantodea, percentage parasitism, *Podagrion*, Torymidae, *Stagmomantis*.

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Introduction

Elucidating how species intensify, attenuate or otherwise modify interactions among other species facilitates understanding of food web structure and dynamics. Study of such 'interaction modifications' (Wootton 1993) has received increasing attention in a variety of field systems (e.g. Morin, Lawlor & Johnson 1988; Wilbur & Fauth 1990). Generally, these efforts have focused on determining whether interaction modifications exist and how they might influence interactions between another species pair. Often, these effects are expressed in terms of changes in per capita interaction strength (e.g. fish reducing the predation rate of salamanders on isopods (Huang & Sih 1990; Adler & Morris 1994)) or via similar mechanisms that force the interaction between two species to depend explicitly upon another species (Billick & Case 1994; Abrams 1995).

Interaction modifications that involve changes to the intensity or nature of particular interspecific interactions may qualitatively alter dynamics of the associated food web. For example, Haemig (1999) suggested predation risk could reverse the sign of an interaction between birds and ants. More generally, when predation risk alters a consumer's behaviour (e.g. Werner 1992), interaction modifications may engender behaviourally mediated trophic cascades (Moran, Rooney & Hurd 1996; Beckerman, Uriarte & Schmitz 1997; Schmitz, Beckerman & O'Brien 1997).

Within a mathematical context, such qualitative alterations of food web dynamics often involve identification of shifts between stable or unstable states. Theoretical models addressing external resource subsidies or dispersal

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explore mechanisms through which ecological complications can determine the stability of particular species interactions. Likewise, theoretical studies have repeatedly demonstrated that adding new species to a food web can influence its stability (e.g. May 1973; Drake 1990). However, such food web models have generally addressed stability issues via the addition of new trophic links rather than modifications of existing ones. Analyses of field studies assessing impacts of interaction modifications on stability of species interactions or food webs are also rare. Ives, Carpenter & Dennis (1999) recently touched upon the issue as part of an investigation of community level effects of lake perturbations.

One arena in which field data have been linked to the stability of species interactions involves insect host–parasitoid systems. For such systems, extensive theoretical efforts have identified aggregation of parasitism risk among hosts as a key stabilizing mechanism in host–parasitoid interactions (e.g. Hassell 1978; May 1978; Chesson & Murdoch 1986; Hassell *et al*. 1991). One line of research (Pacala, Hassell & May 1990; Pacala & Hassell 1991; Hassell *et al*. 1991) has suggested that characterizing the distribution of parasitism among patches permits quantification of the stabilizing potential of aggregation of risk among hosts. This work has led to the so-called '$CV^2 > 1$ rule', which suggests that when the coefficient of variation squared of the density of searching parasitoids per host exceeds one, the host–parasitoid system is stable. Although the CV^2 measure captures, at least roughly, the stabilizing influence of several forms of heterogeneity, others have argued that the links between the distribution of parasitism among patches and aggregation of risk are too tenuous to draw any firm conclusions about system stability (Taylor 1993; Hochberg *et al*. 1996; Gross & Ives 1999). These authors argue instead for assessments of host–parasitoid stability via direct study of the density dependence of the interactions.

If the distribution of parasitism among patches is well-connected to the stability of a host–parasitoid interaction, then impacts from other species that substantially alter that distribution should influence the stability of the host–parasitoid interaction. Experimental tests of this prediction would be difficult undertakings, and would best be pursued after a particular species has been shown capable of altering the distribution of parasitism within a host–parasitoid interaction. Here I provide field data demonstrating the potential for such a shift in parasitism in a case in which the host species is itself a predator, namely a praying mantis. In this system bird damage to overwintering mantis egg cases shifts the distribution of parasitism among patches. This effect, coupled with bird-induced changes in effective patch sizes, leads to a fourfold increase in the theoretical CV^2 criterion and to an increase in the relative importance of host-density-dependent parasitism. The relative timing of the two natural enemy impacts proves to be of considerable importance to understanding how these changes come about.

Methods

STUDY ORGANISMS AND FIELD SITE

Stagmomantis limbata (Hahn) is a common, large-bodied (adult length 50–70 mm) mantis native to the south-western United States and northern Mexico (Helfer 1987). A detailed study of nymphal development (Roberts 1937) and investigations of parasitoid attack (Breland 1941; Fagan & Folarin 2001) comprise most of what is known about the ecology of *S. limbata*. In southern Arizona, *S. limbata* is univoltine, with nymphs hatching in the spring and oothecae being deposited from August to November by adults that do not overwinter. Oothecae of *S. limbata*, which typically harbour between 50 and 250 eggs, are visually obvious to trained observers, especially during winter and spring when the mantids' preferred oviposition substrates (branches of acacia and mesquite trees) lack leaves.

Oothecae of *S. limbata* are regularly parasitized by torymid wasps of the genus *Podagrion* (Breland 1941; Grissell & Goodpasture 1981). *Podagrion* wasps are highly specialized to attack mantid eggs developing inside oothecae, although individual wasp species may attack more than one species of mantid. Female *Podagrion* spp. (about 3 mm body length) deposit eggs inside mantid oothecae via slender ovipositors (an additional 2–3 mm) that penetrate through the hardened oothecal exterior. Wasp larvae develop in a 1 : 1 relationship with individual mantid eggs inside oothecae, with wasps maturing as external parasites of the mantid eggs (Breland 1941). *Podagrion* are winter-active, attacking mantid oothecae from mid-autumn until spring (Breland 1941; Coombs 1994, W.F. Fagan, unpublished data). Unlike their hosts, *Podagrion* wasps are multivoltine, with generation times of 35–60 days (Williams 1914; W.F. Fagan, unpublished data) depending on temperature. However, staggered parasite emergence times within and among oothecae during the winter substantially blurs the bounds of the parasitoid generations.

Parasites emerge by chewing tunnels through the oothecal pith. Consequently, parasite emergence holes can be readily distinguished on mantid oothecae as characteristic, small, circular openings, usually on the sides of the oothecae (Breland 1941; Fig. 1a). After mantid emergence has ceased, counting parasite emergence holes allows identification of the *cumulative* degree of parasitism suffered by each ootheca during the winter (Fagan & Folarin 2001). Similarly, repeated wintertime examinations of oothecae in the field give a good indication of the progress of parasitism. However, save for eyewitness observation of parasitoid oviposition, it is not yet possible to determine how many developing mantids within an ootheca have been parasitized before the wasps complete development and dig emergence tunnels.

In addition to parasitoids, mantid oothecae can also be attacked during the winter by vertebrate

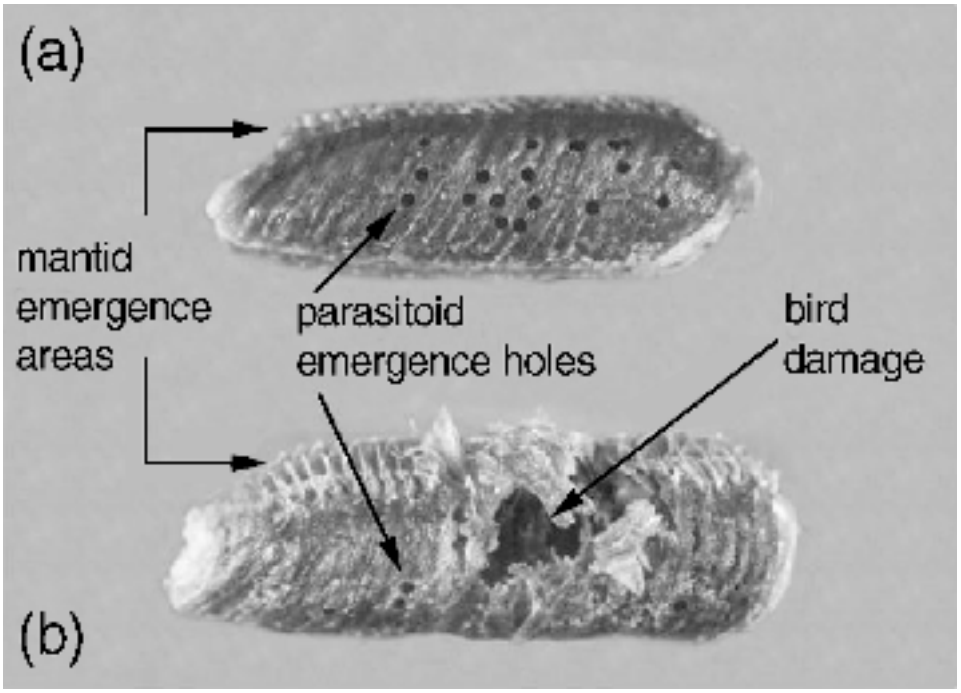

Fig. 1. Oothecae of *Stagmomantis limbata* showing exit holes of parasitoid *Podagrion* wasps and bird damage attributed to resident Mexican jays. (a) Undamaged ootheca; (b) bird-damaged ootheca.

predators, especially birds. The principal bird predators of *S. limbata* oothecae in this study, Mexican jays (*Aphelocoma ultramarina* Bonaparte) are permanent residents in southern Arizona. These birds are fairly generalized in their foraging, feeding on such items as acorns, arthropods and piñon pine nuts (Brown 1983; Brown & Brown 1985). Bird damage to oothecae appears as a large, ragged-edged hole, typically in excess of 1 cm across, with the underlying egg compartments partially hollowed out. Such damage is readily distinguished from the small circular holes produced by *Podagrion* parasites (Fig. 1b). In addition, bird damage typically affects the dorsal surface of the oothecae, whereas the parasite emergence holes generally occur laterally and, to a lesser extent, ventrally. Because bird damage typically involves peeling back rather than completely removing the outer layers of oothecae, it is often possible to identify pre-existing parasite emergence holes from portions of bird-damaged oothecae. Nevertheless, bird damage, especially when severe, has the potential to destroy evidence of past parasite emergence. In such cases, a previous inspection of the ootheca can provide a best guess of parasitism, but is probably an underestimate.

Studies took place in desert grassland habitat of the Chiricahua Mountains, Arizona, USA, at roughly 1500 m elevation. This research site, on the privately owned El Coronado Ranch in the West Turkey Creek drainage, is on the west slope of the Chiricahua range. Patchily distributed woody plants [principally mesquite (*Prosopis glandulosa* Torrey) and whitethorn acacia (*Acacia constricta* Bentham)] emerging through a grassland understory characterized the field site. Trees of these species range from 0·5 to 5 m tall at this site. The parasitic wasps at this site were predominantly *Podagrion crassiclava* Gahan, although some *P. mantis* Ashmead were also present (E. Grissell, personal communication).

FIELD STUDIES AND OOTHECAL COLLECTIONS

On 7 November 1998, field assistants and I located and marked the position of 65 *S. limbata* oothecae. On 15 February 1999, we revisited the initial 65 oothecae and, by re-searching and then increasing the size of the initial search areas, located 65 additional oothecae. I re-censused these 130 oothecae on 12 March 1999 and 9 April 1999, locating an additional 12 oothecae inside my census areas during the latter visit. By my last census (8 July 1999), nine of the 142 marked oothecae had been lost due to storm damage. On each visit, I examined each ootheca for bird damage and counted the number of parasite emergence holes. All marked oothecae (minus those lost during storms) were collected on 8 July 1999, by which time all mantis and parasite emergence had ceased.

On 15 February 1999, I also made surveys well outside my primary study areas, collecting 159 additional oothecae, with an intentional emphasis on collection of bird-damaged oothecae (the 'winter collection'). In the laboratory, I measured length, width and height of these oothecae and counted the number parasite emergence holes present. These 159 oothecae were then distributed to individual rearing containers at 26 °C, and examined daily over the next several weeks for

mantid and parasite emergence. After all mantid and wasp emergence had ceased, oothecae were reinspected to count the number of parasite emergence holes.

Opportunistic observations of foraging and roosting birds during my surveys and additional trips to the area allowed identification of resident Mexican jays as the chief (and perhaps only) bird species responsible for inflicting damage to the oothecae.

DATA ANALYSES

Volume of each ootheca was calculated using the dimensional measurements with the assumption that egg cases were perfectly ellipsoidal in shape. Using emergence data from the 159 winter-collected oothecae, a regression of the number of mantids emerging as a function of oothecal volume was developed (Fagan & Folarin 2001). Data for a subset of these oothecae for which all parasite emergence occurred in the laboratory reveal a relationship between the number of parasite exit holes and the number of emerging wasps ($1{\cdot}26 \pm 0{\cdot}06$ SE wasps for every hole, because some later-emerging wasps dig tunnels that connect with tunnels of earlier emerging wasps) that did not vary significantly as a function of oothecal size. I combined this measure with subsequent mantid emergence data to estimate for each ootheca: (1) its total emergence (mantids + wasps) and (2) its extent of parasitism (wasps/total emergence). I relied upon final counts of parasite exit holes rather than counts of emerging wasps at this step because some wasps had already emerged from oothecae prior to collection. For the 90 bird-damaged oothecae and the 69 undamaged oothecae collected in February and for the 56 bird-damaged and 77 undamaged collected post-emergence in July, I conducted linear regressions of total emergence on oothecal volume to quantify the reduction in overall emergence potential caused by bird damage. Because wasp larvae develop in a 1 : 1 relationship with mantid eggs inside oothecae (Breland 1941), using total emergence in my regressions allows me to examine the overall potential of bird damage as a mortality source for mantid eggs while still compensating for the fact that comparably sized oothecae in the two data sets may have suffered vastly different levels of parasitism. Severe bird damage may have destroyed evidence of parasitism on 13 oothecae from the winter collection and 8 oothecae from the season end collection.

Pacala & Hassell (1991) provide a maximum likelihood approach for estimating the CV^2 of the density of searching parasitoids per host from field data that detail the distribution of percentage parasitism among host patches. Whether or not this approach characterizes aggregation adequately of risk among hosts has been questioned (e.g. Taylor 1993; Gross & Ives 1999). Nevertheless, because I wished to quantify the impact of bird predation on the *Stagmomantis–Podagrion* interaction, I adopted the methodology outlined by Pacala & Hassell (1991) as a convenient, if incomplete, framework. For the July collection, I treated each ootheca as a patch and contrasted the CV^2 of parasitism for undamaged oothecae only with the CV^2 of all oothecae together. This approach utilized the CV^2 of the subset of oothecae that were undamaged as an estimate of what the CV^2 of the entire set of oothecae would have been in the absence of bird damage. Thus, these analyses assume that the undamaged oothecae are a random sample of all oothecae as far as the risk of parasitism is concerned. Results presented later substantiate the validity of these assumptions. Note that parasitism here refers to the cumulative effects of several generations of winter-active parasitoids. Maximum likelihood estimates of the parameters used to calculate the CV^2 were obtained by programming in Maple V. Because the winter-collected oothecae represent a data set intentionally biased towards bird-damaged oothecae, a comparable analysis with the winter data was not appropriate.

Results

Most bird damage to oothecae occurred during winter (December–March), affecting 30% of marked oothecae by April (Fig. 2). All 65 oothecae inspected on 7 November lacked bird damage, but 14% of these oothecae already exhibited parasite emergence holes. Birds did not appear to have consumed any oothecae entirely during the course of the study. Several marked oothecae missing on the last visit appear to have been lost due to a windstorm several weeks earlier (post-emergence), as judged by the recovery of several 'lost' oothecae intact, but fallen to the ground in the vicinity of their oviposition sites.

Birds exhibited a strong preference for elevated oothecae (Fig. 3). Mean ($\pm$ SE) height of damaged oothecae was 2·08 m ($\pm$ 0·06) vs. 1·24 m ($\pm$ 0·08) for undamaged oothecae ($t_{107} = 8{\cdot}2$, $P < 0{\cdot}001$). More specifically, oothecae deposited at least 2·5 m off the ground (8% of the oothecae censused) were almost always attacked by birds, whereas those deposited within 1 m of the ground (25% of those censused) were never attacked. The 35% of all oothecae that were deposited at least 1·75 m above the ground faced a greater than 50% chance of being attacked by birds.

Generally speaking, undamaged oothecae were a random sample of all oothecae as far as the risk of parasitism was concerned. Linear regressions indicated a weak tendency for oothecae higher off the ground to be smaller [volume (cm^3) = 2·177 – 0·174*height (m); $r^2 = 0{\cdot}08$], but this does not appear to have influenced the risk of parasitism. The mean percent parasitism for oothecae below 1 m in height (i.e. those never attacked by birds) did not differ from those above 1 m in height ($t_{61} = 0{\cdot}06$, $P = 0{\cdot}96$). Neither did the overall distributions of percentage of parasitism differ (Kolmogorov–Smirnov $D_{Max} = 0{\cdot}25$, $P = 0{\cdot}24$). Equivalent results occurred using other breakpoints.

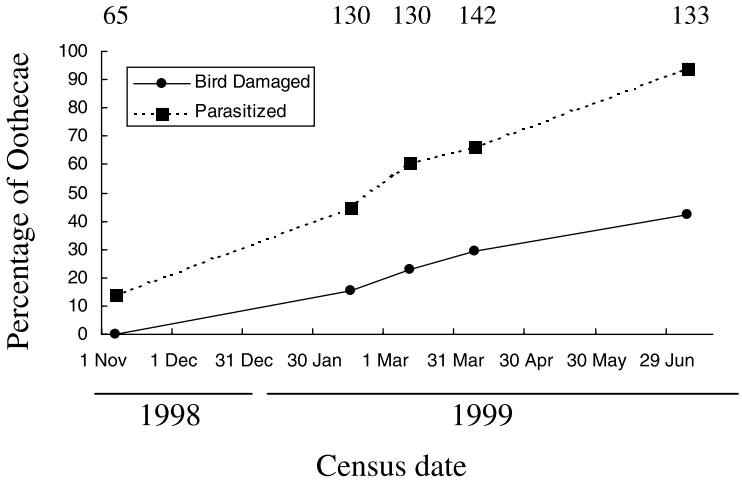

Fig. 2. Prevalence of bird damage and parasitism for oothecae of *Stagmomantis limbata* during winter 1998–99. Parasites of the genus *Podagrion* are multivoltine and winter-active. A core group of oothecae, whose membership increased over time, was monitored on repeat field visits and then collected in July after all emergence had ceased. Numbers at top are sample sizes for each date.

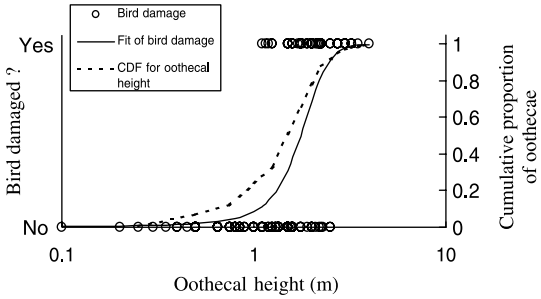

Fig. 3. Occurrence of bird damage as a function of oviposition height. Fitted curve from logistic regression. Overlay *y*-axis plots the cumulative distribution of oothecal height, which is left-shifted relative to the regression of bird damage, indicating a preference by birds for higher oothecae.

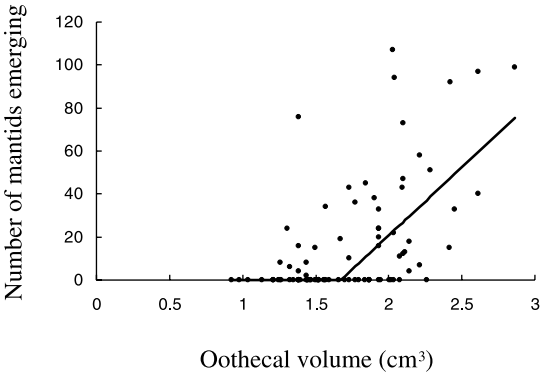

Fig. 4. Conditional linear regression of mantid emergence on oothecal volume for 90 bird-damaged oothecae collected in February 1999. Fitted function was (0, $a + bx + Error$).

Overall, contingency table analysis of the winter collection revealed no statistical association between parasitism and bird damage at the oothecal scale (Pearson $\chi^2_1 = 1{\cdot}2$, $P = 0{\cdot}28$). In contrast, the end of season collection revealed a significant relationship between bird damage and the absence of parasitism (Pearson $\chi^2_1 = 11{\cdot}7$, $P < 0{\cdot}001$). Specifically, all 77 undamaged oothecae from the summer collection eventually suffered some degree of parasitism, but eight of the 56 damaged oothecae escaped parasitism. These eight were neither unusually high nor unusually large (*t*-tests, $P = 0{\cdot}42$ and $P = 0{\cdot}17$, respectively). However, six of them suffered extensive physical damage by birds that may have obliterated evidence of past parasitism.

I also tested whether parasitism and bird damage were independent across time or whether parasitism led to bird damage or vice versa. Examining the transition from 15 February to 12 March, I found that bird-damaged but not yet parasitized oothecae and undamaged-unparasitized were equally likely to suffer new parasitism (92% vs. 90%, Pearson $\chi^2_1 = 0{\cdot}2$, $P = 0{\cdot}87$). Similarly, a history of parasitism did not influence the likelihood of undamaged oothecae suffering bird damage during this time period (Pearson $\chi^2_1 = 1{\cdot}2$, $P = 0{\cdot}27$). Sample sizes and the onset of mantid emergence prevented me from conducting similar tests with other transition periods.

Direct observations of parasitoid behaviour reinforce these results, suggesting relatively little bias of parasites with respect to bird-damaged oothecae. Of 82 female wasps observed attacking oothecae during the study, 27 (33%) were on oothecae that had previously suffered bird damage. Of all oothecae examined across census dates (except July, which is outside the wasps' activity period), 25% had previously suffered bird damage. Thus, the presence of bird damage did not influence the likelihood of wasp attack (Pearson $\chi^2_1 = 0{\cdot}8$, $P = 0{\cdot}38$).

Bird damage to small oothecae frequently resulted in the consumption (or consumption coupled with subsequent dessication) of all mantid eggs developing inside, as evidenced by the fact that nymphs rarely emerged from such oothecae (Fig. 4). In contrast, larger bird-damaged oothecae frequently yielded nymphs. To quantify mantid emergence as a function of oothecal

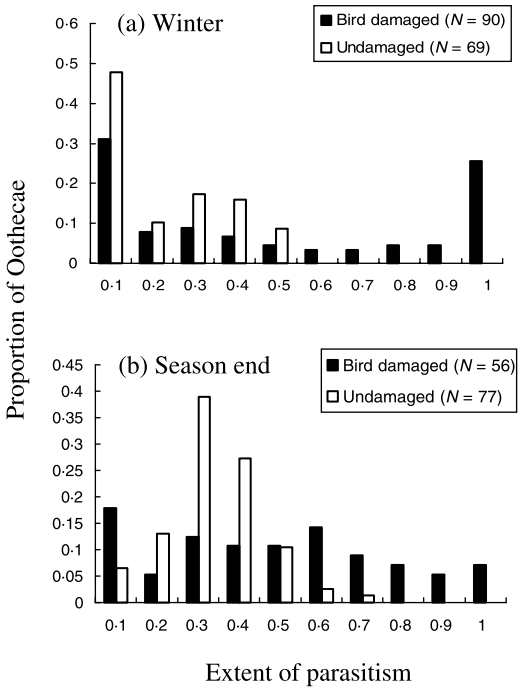

Fig. 5. Frequency distributions for the extent of parasitism (i.e. *Podagrion* wasps/(mantids emerging + *Podagrion* wasps)). Data for winter-collected oothecae (a) were based on actual emergence attained through lab rearing. Data for oothecae marked but left in the field until after emergence (b; see Fig. 1) were estimated from regression relationships (Fagan & Folarin 2001).

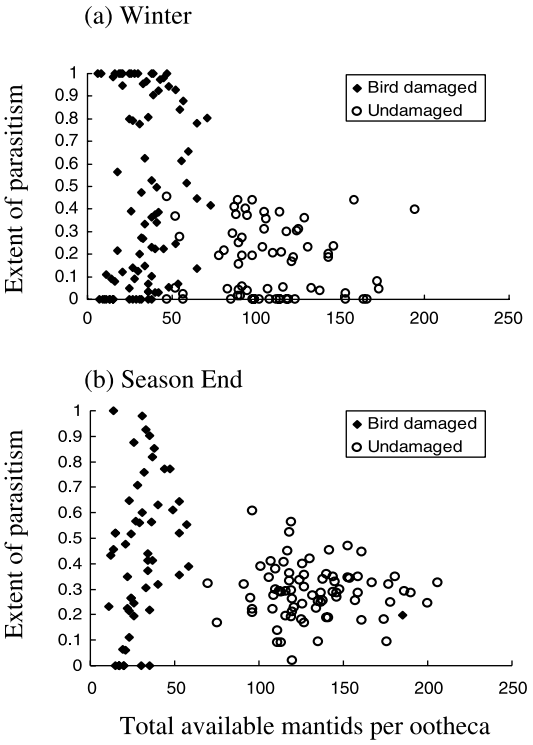

Fig. 6. Extent of parasitism as a function of total available mantids per ootheca from the winter collection (a) and season end collection (b). 'Total available mantids' based upon regression relationships from lab rearings and necessarily includes parasitoids that emerged prior to bird damage.

volume while taking the above size dichotomy into account, I fitted my emergence data using a regression of the form $y = \max(0, a + bx)$. The breakpoint was fitted to the data, yielding 1·68 cm^3 (Fig. 4). This regression explained 64% of the observed variance of mantid emergence from bird-damaged oothecae. Number of wasp holes (or its log-transform) was not a significant predictor of mantis emergence for this data set, and a multiple regression approach was abandoned in favour of the single predictor (volumetric) approach.

After all wasp and mantis emergence from the winter collection of oothecae had ceased, the effective extent of parasitism (Fig. 5) averaged 44% for bird-damaged oothecae, but exhibited substantial variation (95% CI = 8%). Bird-damaged oothecae collected at season's end showed equivalent cumulative parasitism (43 ± 8%). These levels of parasitism in bird-damaged oothecae are significantly higher than the 16% (± 3%) parasitism of undamaged oothecae in the winter collection (Kolmogorov–Smirnov $D_{Max} = 0{\cdot}54$, $P < 0{\cdot}001$) and the 29% (± 3%) from oothecae collected at season's end (Kolmogorov–Smirnov $D_{Max} = 0{\cdot}70$, $P < 0{\cdot}001$). Parasites comprised at least 50% of total emergents from nearly half of bird-damaged oothecae (winter collection: 41%, end of season: 43%), compared with less than 4% of undamaged oothecae collected at season's end. No undamaged ootheca in the winter collection exceeded 46% parasitism.

In the winter collection, 78% of total wasp emergence holes formed on bird-damaged oothecae were completed in the laboratory. This implies that < 20% of parasite eggs successfully deposited in the fall and early winter had completed their development and emerged prior to the occurrence of bird damage. However, because exposure of the winter-collected oothecae to parasitoids was cut short, this 20% figure overestimates the relative frequency of parasitism pre vs. post-bird damage. For those oothecae remaining in the field until season's end, less than 15% of wasp holes formed in oothecae that were ultimately damaged were cut prior to the discovery of bird damage. However, because bird damage reduced the total emergence potential of damaged oothecae to levels far below the distribution typifying undamaged oothecae (Fig. 6), wasps emerging before bird damage often constituted a large fraction of all successful emergents, especially for small damaged oothecae from which few mantids would eventually hatch (Fig. 3).

One might interpret Figs 5 and 6 as indicating that bird-damaged oothecae suffered, on average, little additional parasitism following bird predation (44 vs. 43%). However, parasitism levels in the winter and season's end collections of bird-damaged oothecae are not directly comparable because two-thirds of the oothecae in the latter collection suffered bird damage after 15 February when the winter collected oothecae were removed from the field. In addition, roughly 25% of the

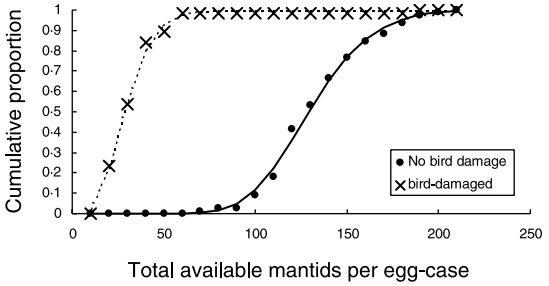

Fig. 7. Cumulative distributions of total available mantids per ootheca for summer collections with and without bird damage. Curves from best fit gamma distributions are also plotted.

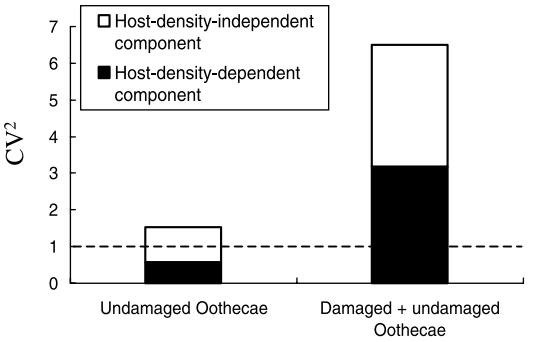

Fig. 8. Coefficient of variation across hosts in the risk of parasitism, with host-density-independent and host-density-dependent components indicated. Data are for end of season collections of undamaged oothecae and all oothecae (undamaged + bird damaged).

winter-collected oothecae suffered unusually extensive bird predation. For these oothecae, the few wasps that emerged prior to collection (most probably prior to bird damage as well) were the only successful emergents. Thus such oothecae had 100% parasitism but few total available mantids (Fig. 6a).

Based upon linear regressions of total emergents against oothecal volume for undamaged oothecae, total emergents = 49·0*volume (cm^3) + 29·7, and for damaged oothecae, total emergents = 32·0*volume (cm^3) – 23·8. Although each of the two regressions had relatively low explanatory power ($r^2 = 0{\cdot}20$ for bird-damaged vs. 0·17 for undamaged oothecae), both were significant. Slopes of the relationships for bird-damaged and undamaged oothecae did not differ significantly ($F_{1,174} = 1{\cdot}32$, $P > 0{\cdot}25$). Based on estimates provided by the regressions, the average bird-damaged egg case lost 74% of its total emergence potential to birds. On average, bird damage reduced the emergence potential of small oothecae (those 0·5–1·35 cm^3 in size) by 82% (74·5 emergents) but that of large oothecae (2·4–3·1 cm^3) by only 48% (72·6 emergents) compared to similarly sized undamaged oothecae. The fact that these proportions differ significantly (Pearson $\chi^2_1 = 13{\cdot}6$, $P < 0{\cdot}001$), coupled with the similarity in the regression slopes, indicates that the relative mortality caused by bird damage decreased with increasing egg case volume. These results assume implicitly that bird-damaged and undamaged oothecae, if left in the field, would not have suffered differential mortality of potential emergents from the time of their collection in February to the time of mantid emergence in mid-March–mid-May.

In addition to its effect on overall emergence potential, bird predation also altered the nature of the distribution of mantid eggs among oothecae. Although the availability of 'hosts among patches' in both damaged and undamaged oothecae were approximately gamma distributed (Fig. 7) (where the gamma distribution is $\gamma(\alpha, \beta) = \frac{1}{\beta^{\alpha}\Gamma(\alpha)} x^{\alpha-1} e^{\frac{-x}{\beta}}$), the underlying parameters were different. Specifically, for undamaged oothecae, eggs were distributed as γ (23·4, 5·6) and for bird-damaged oothecae as γ (5·7, 5·3), indicating that the majority of the birds' effects were concentrated on the 'scale' parameter of the gamma distribution, with less impact on its shape (Hastings & Peacock 1975)

Evaluated at season's end, bird damage increased the CV^2 of (cumulative) parasitism risk among hosts from 1·5 (considering undamaged oothecae alone) to 6·5 (considering undamaged + damaged oothecae together). Absolute increases in both the host-density independent and host density dependent components contributed to this fourfold shift away from the theoretical $CV^2 = 1$ boundary separating stability from instability. On a relative basis, bird damage increased the contribution of host-density-dependent parasitism to the total CV^2, although host-density-independent parasitism predominated for both the complete data set and the undamaged subset (Fig. 8).

Discussion

Bird damage to oothecae represents a formidable source of mortality for *Stagmomantis limbata*. Birds eventually damaged more than one-third of all oothecae censused (Fig. 2), and ate or otherwise killed an estimated 72% of the summed oothecal emergence potential of the oothecae they attacked. Summing across oothecae that completely escaped bird predation from oviposition to emergence, roughly 35% of mantid eggs were parasitized by *Podagrion* wasps. Hence some of the potential emergents lost to bird predation in this study would undoubtedly have been *Podagrion*, either because some mantid eggs had already been parasitized prior to the bird predation, or because they would eventually have been parasitized by later emerging wasps. Consequently, among bird-damaged oothecae, a better estimate for the loss of mantid eggs to bird predation is about 47% [= 72% * (1–35%)]. Because 42% of oothecae were attacked, birds inflicted roughly a 20% overall loss. This compares with an estimated 31% loss of mantid eggs to the cumulative effects of several generations of parasitoids.

In addition to direct mortality of both *Stagmomantis* and *Podagrion*, bird predation may also modify the nature of the interaction between mantids and their

parasites. Specifically, bird predation leads both to an increase in the overall extent of parasitism and to a significant shift in the distribution of parasitism among oothecae (Figs 5, 6). In fact, after accounting for (parasitized and unparasitized) mantid nymphs lost to bird predation, bird-damaged oothecae collected in February suffered half again as much realized parasitism as did undamaged oothecae exposed to parasitism for the entire season.

This distributional shift in the severity of parasitism appears to occur via two mechanisms. First, emergence by initial generations of the multivoltine wasps early in the winter affords a way for some wasps (but no mantids) to escape bird predation, increasing the effective fraction of mantid eggs parasitized for some oothecae. Secondly, bird predation does not appear to alter the distribution of parasitoids among oothecae, but instead alters radically the distribution of available hosts (Figs 6, 7).

The relative timing of bird predation and parasitoid attack may prove critical to the host–parasitoid interaction in this system, acting as a kind of switch that, depending on the year, shifts impacts of bird predation toward or away from parasitized mantid eggs. It is important to distinguish between bird impacts on the population size of available parasites and impacts on the distribution of parasite attacks. For example, a low degree of random bird predation early in the winter is unlikely to influence the parasite population greatly because relatively few developing mantid nymphs would have been parasitized yet. However, early season bird attacks could have more profound consequences for the distribution of parasitoid attacks among hosts. Existing data suggest that, generally speaking, parasitoids neither avoid nor aggregate toward bird-damaged oothecae. However, bird damage, especially damage occurring early in the season, shifts the distribution of available hosts among patches: Damaged oothecae are both smaller and less variable in terms of the number of available mantids than are undamaged ones (Figs 6,7). In contrast, later in the winter when substantially larger fractions of mantids may have been parasitized, the same degree of random bird predation might involve substantial mortality of parasitized hosts. Other kinds of behavioural preferences by birds may, of course, be at work, but it is hard to imagine a bird investing the time to break open a hardened ootheca as preferring parasitized or unparasitized eggs. This discussion suggests sensitive dependence of the dynamics of the mantid host–parasitoid system on the timing of the interaction modification by birds, a feature not easily incorporated into standard host-parasitoid models. Models explicitly representing temporal accumulation of parasitoid attacks over several generations or addressing multivoltinism in other ways (e.g. Rohani & Miramontes 1995; Briggs & Godfray 1996; Takagi 1999) and experimental manipulations of bird access to oothecae would provide alternative avenues for examining these ideas.

Variation in the degree of parasitism, especially the extent to which parasitism differs spatially among hosts (as a function of, and/or independent of, host density per patch), is crucial to the stability of host-parasite dynamics in a variety of theoretical models (e.g. Chesson & Murdoch 1986; Hassell *et al*. 1991; Pacala & Hassell 1991). In this study, bird predation shifted the distribution of parasitism risk among hosts away from the stability–instability boundary by producing a cohort of small oothecae that varied little in the number of mantid eggs they contained. These relatively similar patches were then subjected to extensive variation in the extent of parasitism suffered. Although the host–parasitoid interaction was above the $CV^2 = 1$ threshold prior to the impacts of bird damage, it was not far above the threshold. Adding bird predation to the mix modified the spatial character of the mantid–wasp interaction, resulting in a fourfold increase in the CV^2 metric to well above the stability boundary and an increase in the relative importance of host-density-dependent parasitism (Fig. 8).

This mantid-parasitoid system is one in which impacts from other members of the ecological community impinged upon an otherwise highly specialized interaction. Tscharntke (1992) reported a similar case involving gall-forming midges on reeds. In that system, blue tits disproportionately attacked parasitized galls, resulting in subadditive impacts of birds and parasitoids on midges. For these kinds of systems, ignoring the potential for interaction modifications and studying host–parasitoid interactions in isolation provides an incomplete picture of the range of dynamics possible and has the potential to 'give the wrong answer' (Kareiva 1994).

This host–parasitoid study system is somewhat unusual in that the host species is itself a predator. Indeed, of nearly 100 host species whose spatial patterns of parasitoid attack were reviewed in Lessells (1985), Stiling (1987) and Walde & Murdoch (1988), only six were predatory, and all but one of these were vespid or sphecid wasps. Although mantids are strictly predatory (and thus one might expect a decrease in mantid densities to yield increases in numbers or biomass of other arthropods), the consequences of bird predation on mantid oothecae for the rest of the desert grassland arthropod community are not clear. That is, bird predation leading to lowered densities of hatching mantids will not necessarily translate into less suppression of herbivorous arthropods. Ironically, on a per capita basis, mantid nymphs surviving to emerge from bird-damaged oothecae may enjoy a substantially increased chance of reaching adulthood. This is because mantid survivorship, especially among first instar nymphs, is generally very poor, and decreases strongly with increasing density due to starvation, intraguild predation, and size-dependent cannibalism (e.g. Hurd & Eisenberg 1984; Fagan & Hurd 1994). Because mantid emergence from an individual ootheca is sometimes highly synchronous (Eisenberg & Hurd

1977; Hurd & Eisenberg 1988) and because first instar nymphs may disperse little following emergence (Hurd & Eisenberg 1984), immediately after hatching in the spring, local densities of mantid nymphs may be high at a time when prey are scarce (Hurd & Eisenberg 1984; Fagan & Odell 1996, W.F. Fagan unpublished data).

Overwinter mortality from bird predation greatly reduces local hatch densities for affected oothecae, and may therefore indirectly facilitate per capita survivorship (and ultimately reproduction) of nymphs from damaged oothecae. As a result, untangling the consequences of vertebrate predation for the dynamics of this or other systems in which generalized consumers impinge upon the dynamics of specialized host–parasitoid interactions will require novel theory in addition to increased experimentation and field study.

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