Abstract Recent population-dynamic theory suggests that mechanisms of lethal interference competition can have profound effects on parasitoid coexistence and pest suppression in biological control systems. We investigated lethal intraspecific and interspecific interference competition in Eretmocerus eremicus and Encarsia sophia, parasitoids of the whitefly pest, Bemisia tabaci. Our first experiments evaluated whether one or both species could suppress the progeny production of the other species through two mechanisms of lethal interference competition: (1) usurpation of hosts in cases of multiparasitism, and (2) host feeding on and killing parasitised hosts. We found that both species could suppress the progeny production of the other. E. eremicus’ effect on E. sophia appeared to reflect multiparasitism. E. sophia’s effect on E. eremicus appeared to reflect a combination of multiparasitism and host feeding on parasitised hosts. Second, we investigated the effects of lethal intraspecific interference on conspecific progeny production in both species. E. sophia interfered intraspecifically by host feeding on parasitised hosts. E. eremicus also apparently host fed on parasitised hosts, however the effect of host feeding on conspecific progeny production was slight. Third, host dissections and behavioral observations confirmed the mechanisms inferred from the progeny production experiments. Our results suggest a need to consider mechanisms of lethal interference competition in theoretical and empirical research on parasitoid competition.

Keywords Biological control · Host feeding · Interspecific competition · Intraspecific competition · Multiparasitism

Introduction

Biological control systems exemplify a diversity of ecological processes. In the classic idealised case, a single introduced natural enemy controls an exotic pest. In these situations, “control” implies strong “top-down regulation” (Hawkins 1992) and tightly coupled predator-prey or parasitoid-host dynamics (Murdoch 1990). Ecologists have long sought to identify the processes that promote pest suppression and/or long-term persistence of pest-enemy interactions (Hassell 1976; Beddington et al. 1978; Murdoch 1990; Murdoch et al. 1985, 1998).

In most cases of biological control, however, more than a single natural enemy species is introduced to control a pest (Myers et al. 1989). In these situations, both competitive exclusion and coexistence are possible outcomes, and the resulting natural enemy complex can be made up of a few or many species. Here, the question is whether interspecific interactions among natural enemies can disrupt effective biological control. This issue has, in fact, been one of the most important and longest-standing controversies in classic biological control (Pemberton and Willard 1918; Smith 1929; Turnbull and Chant 1961; DeBach 1966; May and Hassell 1981; Kakehashi et al. 1984; Briggs 1993; Rosenheim et al. 1995; Murdoch et al. 1998). One of the key challenges has therefore been to identify the processes that promote pest suppression and long-term persistence of multiple natural enemy species in biological control systems.

Recent theory sheds some new light on this issue by highlighting the role of lethal interference competition among natural enemies, specifically facultative hyper-parasitism (Briggs 1993; see also Mills and Gutiérrez 1996), multiparasitism (Briggs 1993), and intraguild predation (Rosenheim et al. 1995; Holt and Polis 1997). Each of these interactions may be thought of as mechanisms of lethal interference competition: direct negative interactions that lead to the death of heterospecific competitors (Murdoch et al. 1998). Intraguild predators kill other predators in addition to killing their prey (Holt and Polis 1997), facultative hyperparasitoids parasite and
kill other parasitoid species and “multiparasitoids” (cf., May and Hassell 1981) kill heterospecific competitors within the host. Although the trophic relationships are not identical for each of these interactions (Rosenheim et al. 1995), the consequences for individuals are the same – one natural enemy kills another.

The population consequences of these mechanisms of lethal interference competition can also be quite similar. Recent population-dynamic models suggest that interference competition can yield a strong advantage to the intraguild predator, facultative hyperparasitoid or multiparasitoid. This may promote coexistence of natural enemies through “counter-balanced competition” (Zwolfer 1971) but may also lead to the disruption of biological control (Briggs 1993; Rosenheim et al. 1995). In the models, two parasitoid species may coexist through a balance between the competitive advantage afforded by interference competition and the primary predator or parasitoid’s greater efficiency at attacking the pest (Briggs 1993; Holt and Polis 1997). However, coexistence can be associated with lower densities of the more efficient parasitoid. Because more efficient natural enemies are essentially replaced by their less efficient counterparts, pest densities can be higher than what they would be with the more efficient natural enemy species alone (Briggs 1993).

Thus, recent population-dynamic theory underscores the need to understand mechanisms of lethal interference competition in biological control systems. Here, we investigate lethal interference competition in *Eretmocerus eremicus* (Rose and Zolnerowich) (= *E. nr. californicus*) and *Encarsia sophia* (Girault) (= *E. transvena*), two parasitoids of the whitefly pest, *Bemisia tabaci* (Gennadius) (Strain B). One potential mechanism of lethal interference competition in these species is “multiparasitism” (MacKauer 1990). Multiparasitism occurs when heterospecific parasitoids lay eggs in the same host individual. Typically, the eggs or larvae of one species are killed by the other through either physiological suppression or physical attack (Mackauer 1990). Another potential mechanism of lethal interference competition in our study is “host feeding on parasitised hosts.” Host-feeding parasitoids (including *E. sophia* and *E. eremicus*) probe and feed on the hemolymph of some of the hosts they encounter (Jervis and Kidd 1986). In “destructively host feeding” species (c.f., Jervis and Kidd 1986), the act of probing and feeding causes the death of the host and may cause the death of parasitoid eggs or larvae within. Host feeding thus introduces the possibility of both interspecific and intraspecific interference competition.

Note that *E. sophia* may also interfere with *E. eremicus* via “autoparasitism” (or “heteronomous hyperparasitism”; Hunter and Wooley 2001). As in other autoparasitoids, female *E. sophia* develop as primary parasitoids of Homoptera (in this case, whiteflies) and males develop as hyperparasites – feeding on and killing the immature stages of conspecific and heterospecific parasitoids (Hunter and Wooley 2001; Hunter and Kelly 1998). We did not study this mechanism of interference, however (see Hunter and Kelly 1998). Instead, we focused our attention on recently parasitised hosts, which are unsuitable for the development of male *E. sophia*.

Our primary goal was to document that these parasitoids could interact via one or more mechanisms of lethal interference competition. Specifically, we addressed the following. Can *E. sophia* reduce the progeny production of *E. eremicus* through multiparasitism and/or host feeding on parasitised hosts? Can *E. eremicus* reduce the progeny production of *E. sophia*? We also investigated the effect of host feeding on conspecific progeny production in both *E. sophia* and *E. eremicus*. Overall, we sought to identify mechanisms of lethal interference competition as a first step towards understanding the population-level interactions between these species in a field-cage experiment (M. S. Hunter et al., unpublished data) and towards understanding in a general way, the potential role of lethal interference competition in parasitoid communities.

### Materials and methods

Parasitoids were maintained in a walk-in environmental chamber at ca. 27°C and under 16 h fluorescent light. The *Encarsia sophia* culture was established using wasps obtained from the USDA/APHIS Mission Biological Control Laboratory in Texas (= *Encarsia transvena*: quarantine no. M93003). This population of *E. sophia* was introduced to the southwestern U.S.A. from Murcia, Spain in 1996 for biological control of *B. tabaci*. Like other species in the genus *Encarsia, E. sophia* are endoparasitic, i.e., their immature stages develop within whitefly nymphs. The *Eretmocerus eremicus* culture was established from wasps from Koppert Biological Systems (Romulus, Mich.). This species is native to Arizona and southwestern California where it attacks *B. tabaci* (Gerling 1966; Rose and Zolnerowich 1997). As in other *Eretmocerus* species, *E. eremicus* eggs are laid externally but the larvae enter the host and develop as endoparasitoids (Gerling 1966).

Parasitoid cultures were reared on the greenhouse whitefly, *Trialeurodes vaporariorum* (Westwood) (Aleyrodidae; Hemiptera). Whitefly cultures were maintained under greenhouse conditions (natural light and 18–32°C). *T. vaporariorum* was reared on tobacco (*Nicotiana tabacum* L.) and beans (*Phaseolus vulgaris* L.). *B. tabaci* was reared on cotton (*Gossypium hirsutum* L.) and occasionally on tobacco or honeydew melon (*Cucumis melo* L.).

Experimental cotton plants were grown individually under greenhouse conditions in “cup cages”, which were constructed as follows. The bottom section of a cup cage consisted of a 473-ml clear plastic cup with its bottom end cut off and glued to the rim of a plastic pot (6.2 cm in diameter). The cup-cage lid consisted of an inverted 266-ml clear plastic cup with the bottom end cut off and covered with fine nylon mesh. The cotton plant grew up into the clear part of the cup cage from soil in the pot.

Once a plant early third-instar *B. tabaci* nymphs were removed from the area inside the callous cushion. Experimental parasitoids were obtained by exposing *T. vaporariorum*-infested bean plants to 20–30 female wasps. Infested plants were maintained under experimental conditions: 25°C and 14 h of light. After 12–18 days, wasps were isolated as pupae in
0.9-ml (quarter dram) vials with a drop of honey. Vials were checked daily for emergence and wasps that had emerged were used 2 days later. Matings were observed for all experimental females.

Interspecific interference

The first experiments were designed to detect the effects of interspecific interference competition on heterospecific progeny production. For a focal species, there were two treatments: (1) a female of that species followed by a heterospecific competitor (Competition Treatment), or (2) a female of that species alone, i.e., no subsequent competitor (Alone Treatment). The first wasp was released into a clip-cage arena, which fit snugly over the callosus pad and leaf. After 24 h, the first female was removed and treatment assigned randomly. If the Competition Treatment was assigned, a heterospecific female was released into the clip cage for 24 h. After the wasps were removed, the plant was kept alive for 10–14 additional days. We then recorded the number of wasp pupae, dead whiteflies and unparasitised whiteflies (whitefly exuviae). “Dead” whiteflies were flattened, dried and discoloured.

Data were analyzed using Generalised Linear Interactive Modelling (GLIM) (McCullough and Nelder 1983; Crawley 1993), a statistical package that can utilise non-normal error distributions. Since the critical tests involved count data, the appropriate error distribution was Poisson. The main comparison was the number of pupae in the Alone Treatment versus the Competition Treatment. Effects of interference competition should appear as a reduction in the number of pupae of the focal species in the Competition Treatment relative to the Alone Treatment.

We used two additional analyses to infer mechanisms of interspecific interference competition. First, we compared the number of dead whiteflies in the Alone Treatment versus the Competition Treatment. If host feeding on parasitised hosts was a mechanism of interference, then there should be more dead hosts in the Competition Treatment than in the Alone Treatment. We also devised a test – based on simple accounting – to determine if more heterospecific progeny were produced in the Competition Treatment than would be expected in the absence of (successful) multiparasitism. Some heterospecific pupae should be produced even with no multiparasitism because a few hosts were left unparasitised (and not fed upon) by the first parasitoid. The number of hosts left unparasitised by the first parasitoid can be estimated as the number of emerged whiteflies in the Alone Treatment. If no multiparasitism occurred, then we would expect that the number of hosts unparasitised in the Alone Treatment should be equal to the number of heterospecific pupae in the Competition Treatment plus the number of hosts unparasitised by either parasitoid (which were very few: four of 784 hosts in all). If, however, the heterospecific species usurped some hosts from the first species through multiparasitism, then we would expect more heterospecific pupae than could have come from unparasitised hosts left by the first parasitoid.

Mechanisms of interspecific interference: host dissections

An additional experiment was conducted to determine whether multiparasitism had indeed occurred in the Competition Treatments. The Competition Treatments described above were repeated for the two-species combinations, however, all host nymphs were dissected after the second wasp was removed from the arena. Hosts were first carefully turned over with an insect pin to identify *E. eremicus* eggs, which are laid between the ventral surface of the host cuticle and the leaf. Hosts were then dissected in Insect Ringer’s solution to identify *E. sophia* eggs. Dead hosts were also turned over to look for *E. eremicus* eggs. It was impossible to dissect these dried-out hosts to look for *E. sophia* eggs. We scored the number of multiparasitised hosts, hosts parasitised by a single species, and dead hosts that had been previously parasitised by *E. eremicus*.

Intraspecific interactions

Additional experiments were conducted to detect lethal intraspecific interference competition in both *E. sophia* and *E. eremicus*. The design was similar to that of previous experiments except that the first and second females were the same species. Although we were unable to discern whether the first or second female produced a given pupa, an effect of host feeding on parasitised hosts should appear as a reduction in the total number of progeny produced, and an increase in the number of dead hosts in the Competition Treatment. These data were also analyzed in GLIM, using Poisson-distributed errors.

Behavioural observations

Finally, we conducted behavioral assays in an attempt to confirm the mechanisms of interference inferred from the experiments described above. The initial experimental set up was the same as in previous experiments, and observations of all combinations of first and second parasitoid species were conducted. Mated individual female *E. eremicus* or *E. sophia* were released into arenas with eight third instar nymphs for 24 h. After the first female was removed, a single mated conspecific or heterospecific female was introduced to the arena and observed until: (1) 40 min had elapsed, (2) the parasitoid sat motionless for 10 min, or (3) the parasitoid flew off the leaf surface. In the intraspecific assays, observations were also terminated when the parasitoid initiated host feeding (since the goal was merely to confirm host feeding on parasitised hosts). Immediately after observations, all hosts were dissected to look for parasitoid eggs.

Results

Interspecific interference: *E. eremicus’* effect on *E. sophia*

*E. eremicus* reduced *E. sophia*’s progeny production by about 50% or 0.6 offspring. The number of pupae produced in the Alone Treatment dropped from 1.24 offspring to 0.64 offspring in the Competition Treatment (Fig. 1a). This difference is statistically significant (analysis of deviance, \( \chi^2=4.87, 1 \text{ df}, P=0.027 \)). Host feeding on parasitised hosts did not appear to be the mechanism by which *E. eremicus* reduced *E. sophia*’s progeny production. The number of dead whiteflies in the alone and Competition Treatments were remarkably similar (Fig. 1b; analysis of deviance, \( \chi^2=0.049, 1 \text{ df}, P=0.83 \)). Instead, *E. eremicus’* effect on *E. sophia* appeared to reflect multiparasitism. *E. eremicus* produced about 0.5 more progeny than would be expected if they had not multiparasitised or lost in multiparasitism (Fig. 1c; analysis of deviance, \( \chi^2=5.01, 1 \text{ df}, P=0.025 \)). This difference is similar in magnitude to the 0.6 reduction in *E. sophia*’s progeny production.

Interspecific interference: *E. sophia’*s effect on *E. eremicus*

*E. sophia* reduced *E. eremicus*’ progeny production by 92% or about four offspring (Fig. 2a), a statistically significant effect (analysis of deviance, \( \chi^2=100, 1 \text{ df}, P<0.001 \)). Some of the reduction in *E. eremicus’* proge-
ny was consistent with an effect of host feeding on parasitised hosts. The number of dead whiteflies increased significantly in the Competition Treatment from about three hosts to almost six hosts (Fig. 2b; analysis of deviance, $\chi^2 = 19.96$, 1 df, $P < 0.001$). *E. sophia* also appeared to reduce *E. eremicus*’ progeny production via multiparasitism. *E. sophia* produced about 1.5 more progeny than would be expected if they had not multiparasitised or lost in multiparasitism (Fig. 2c; analysis of deviance, $\chi^2 = 21.2$, 1 df, $P < 0.001$). The increase in the number of
dead hosts (three hosts) and the greater than expected \textit{E. sophia}'s progeny production (1.5 offspring) easily explain the total reduction in \textit{E. eremicus}' progeny production (four offspring).

Mechanisms of interspecific interference: host dissections

We inferred from the results of the experiment above that both \textit{E. eremicus} and \textit{E. sophia} reduced each others' progeny production through multiparasitism. The results of the dissections were consistent with this explanation; both species multiparasitised (Table 1). In addition, many hosts appeared dead and had been previously parasitised by \textit{E. eremicus} (Table 1). These situations predominately occurred when \textit{E. eremicus} was followed by \textit{E. sophia}.

Intraspecific interference: \textit{E. sophia}

Exposure of conspecific-parasitised hosts to a second female \textit{E. sophia} reduced the total progeny production of \textit{E. sophia}. The total number of pupae of \textit{E. sophia} in the Competition Treatment was about 1.5 individuals less than in the Alone Treatment (Fig. 3a; analysis of deviance, $\chi^2=16.8$, 1 df, $P<0.001$). This reduction may have reflected host feeding on parasitised hosts. The number of dead whiteflies was greater in the Competition Treatment by an average of two individuals (Fig. 3b; analysis of deviance, $\chi^2=7.67$, 1 df, $P=0.0056$). This difference easily explains the 1.5 offspring reduction in \textit{E. sophia}'s progeny production.

Intraspecific interference: \textit{E. eremicus}

Exposure of conspecific-parasitised hosts to a second female \textit{E. eremicus} reduced the total progeny production of \textit{E. eremicus} from 3.6 pupae in the Alone Treatment to three pupae in the Competition Treatment (Fig. 4a). Variance in progeny production was high, however, and this difference was not statistically significant (analysis of deviance, $\chi^2=1.05$, 1 df, $P=0.30$). Nevertheless, the number of dead hosts in the Alone Treatment increased from about three individuals to over four individuals in the Competition Treatment (Fig. 4b). This difference is marginally statistically significant (analysis of deviance, $\chi^2=3.866$, 1 df, $P=0.05$). Taken together, these results suggest a small effect of host feeding on parasitised hosts on \textit{E. eremicus}' progeny production.

Table 1 Host dissection data from repetitions of the Competition Treatments. Shown are the mean number of hosts per arena in various categories (mean+Poisson SEs). Note that Poisson SEs may be asymmetric so both mean-negative (neg.) SEs and mean+positive (pos.) SEs are given. Note also that dead hosts with (w/) \textit{Encarsia sophia} eggs could not be evaluated in dissections. w/o Without

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Multiply parasitised hosts</th>
<th>E. sophia-parasitised hosts</th>
<th>E. eremicus-parasitised hosts</th>
<th>Dead hosts w/ E. eremicus eggs</th>
<th>Dead hosts w/o E. eremicus eggs</th>
</tr>
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<tbody>
<tr>
<td>\textit{E. sophia} followed by \textit{E. eremicus} (\textit{n}=11)</td>
<td>1.00 (0.74, 1.35)</td>
<td>0.45 (0.32, 0.78)</td>
<td>0.54 (0.36, 0.82)</td>
<td>0.27 (0.15, 0.48)</td>
<td>5.54 (4.88, 6.30)</td>
</tr>
<tr>
<td>\textit{E. eremicus} followed by \textit{E. sophia} (\textit{n}=13)</td>
<td>1.54 (1.23, 1.92)</td>
<td>0.77 (0.56, 1.05)</td>
<td>0.31 (0.19, 0.50)</td>
<td>2.69 (2.27, 3.19)</td>
<td>2.62 (2.20, 3.10)</td>
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Fig. 3 A \textit{E. sophia}'s effect on \textit{E. sophia}. Shown is the mean total number of progeny (pupae) produced when hosts exposed to a female \textit{E. sophia} are exposed to a second female \textit{E. sophia} (Competition) or to no additional \textit{E. sophia} (Alone) (w/ Poisson SEs). Sample sizes: alone, \textit{n}=22; competition, \textit{n}=24. B Effect of host feeding by \textit{E. sophia}. Shown is the mean number of dead hosts in the Competition and Alone Treatments (w/ Poisson SEs). Sample sizes: alone, \textit{n}=22; competition, \textit{n}=24
Behavioral observations

Female *E. sophia* readily multiparasitised. Of the eight female *E. sophia* observed in assays in which *E. sophia* followed *E. eremicus*, all eight multiparasitised at least once. *E. eremicus* were also observed to multiparasitise, although only three of the ten observed females did so. Because of small sample sizes and different initial conditions in these assays (number of available unparasitised and parasitised hosts), it is difficult to formally compare *E. sophia*'s and *E. eremicus*' behavior. However, *E. eremicus* appeared to have a low propensity to multiparasitise, and frequently rejected hosts after antennation; the mean acceptance-to-contact ratio (with SE) was 0.19 (0.08). *E. sophia* appeared to readily host feed on *E. eremicus*-parasitised hosts; eight of eight females were observed to host feed at least once on hosts previously parasitised by *E. eremicus*. By contrast, none of the eight female *E. eremicus* were observed to host feed on *E. sophia*-parasitised hosts (none host fed at all). Female *E. sophia* also readily host fed on conspecific parasitised hosts; six of eight fed on hosts parasitised by the first female *E. sophia* (one host fed on an unparasitised host and one host fed on a host previously parasitised by itself). Only one of seven female *E. eremicus* was observed to host feed on an *E. eremicus*-parasitised host (one fed on an unparasitised host).

**Discussion**

Our study identified two mechanisms of lethal interference competition in *E. sophia* and *E. eremicus* – multiparasitism and host feeding on parasitised hosts. We initially expected that either *E. eremicus* or *E. sophia* would have a consistent advantage in multiparasitism, and that one species would therefore have a substantially greater impact on the progeny production of the other. We found, however, that both species could win in multiparasitism when ovipositing second. There are two potential explanations for this result. First, both species may have been able to win in some cases of multiparasitism. Thus, both may have reduced the progeny production of the other by winning some fraction of contests (and losing others). A second explanation is that second-ovipositing females may have actually had an advantage over first females, regardless of species. We cannot distinguish between these possibilities, but both have bases in other studies. There are examples from other systems that show approximately equal competitive ability (e.g., Isenhour 1988; Tillman and Powell 1992; Micropletis croceipes versus Micropletis demolitor) and cases of second-female advantage (e.g., Tillman and Powell 1992; *Cotesia kazak* versus *M. croceipes*).

Neither of these scenarios has been incorporated in population dynamic models, so the potential role of multiparasitism in *E. eremicus-E. sophia* interactions is unclear. Population models in which parasitoids compete directly via multiparasitism generally assume that one species always wins in competition in the host (Pimm and Lawton 1978; May and Hassell 1981; Kakehashi et al. 1984; Briggs 1993). We note, however, that behavioural observations suggested that *E. eremicus* was much less prone to multiparasitise than *E. sophia*. We might therefore expect that the outcome of multiparasitism in these species may well approximate a consistent-advantage scenario, with *E. sophia* having a much greater effect on *E. eremicus* than the reverse.

We also found that *E. sophia* reduced *E. eremicus*’ progeny production by host feeding on and killing *E. eremicus*-parasitised hosts. In addition, *E. sophia* host fed on and killed conspecific-parasitised hosts and thereby reduced conspecific progeny production. Other examples of host feeding on parasitised hosts exist, though they are few. Yu et al. (1990) found that *Aphytis melinus* multiparasitised and host fed on scale insects previously parasitised by *Encarsia perniciosi*. In combination, host feeding and multiparasitism by *A. melinus* caused mortality of *E. perniciosi*, although as presented, the data do not separate the effects of the two phenomena. *A. melinus* has also been observed to destructively host feed on...
conspecific parasitised hosts (T. R. C., personal observation). Ueno (1999) documented that two ichneumonids, *Pimpla nipponica* and *Itoplectis naranyae*, fed on hosts parasitised by the other species. *I. naranyae* also host feeds on conspecific-parasitised hosts (Ueno 1998). Finally, we (T. R. C. and M. S. H.) have observed that other *Encarsia* spp. – *E. formosa* and *E. luteola* – feed on both conspecific- and heterospecific-parasitised hosts (unpublished data). We suggest that host feeding on parasitised hosts may be a generally important mechanism of interaction in parasitoid communities, particularly in biological control systems, where destructively host-feeding parasitoids have disproportionately established and led to successful control relative to non-host-feeding species (Jervis et al. 1996).

The potential population-dynamic consequences of host feeding on parasitised hosts are not entirely clear. No population dynamic model directly incorporates host feeding on parasitised hosts as a mechanism of interference competition. Nevertheless, recent population models consider an analogous interaction: autoparasitism (Mills and Gutierrez 1996; Briggs and Collier, in press). Autoparasitoids have hyperparasitic males that develop on and kill immature conspecific and heterospecific parasitoids (Hunter and Wooley 2001). In autoparasitoid-host models, interspecific attack yields a tremendous competitive advantage to the autoparasitoid, as do other types of lethal interspecific interference competition. Intraspécific attack, on the other hand, tends to promote coexistence by introducing self-limiting density dependence in the autoparasitoid population (Briggs and Collier, in press). We expect that by analogy to autoparasitism, host feeding on parasitised hosts may promote either coexistence or competitive displacement of the heterospecific competitor. However, we lack sufficient quantitative information to predict the net effect of host feeding on *E. eremicus*–*E. sophia* interactions.

In the end, we expect that the outcome of competition between *E. eremicus* and *E. sophia* should depend in part on the trio of mechanisms of lethal interference competition in these species, i.e., host feeding on parasitised hosts, multiparasitism, and particularly autoparasitism by *E. sophia*, the latter of which appears to strongly favour *E. sophia* over *E. eremicus* (Hunter and Kelly 1998; Briggs and Collier, in press). *E. sophia*’s apparent advantage in multiparasitism and autoparasitism may well have allowed this species to drive *E. eremicus* to near extinction in field cages over the course of a 7-week competition experiment (Hunter et al., unpublished data).

**General implications**

The *E. eremicus*–*E. sophia* system exemplifies a potentially important ecological mechanism of interaction in parasitoid communities: lethal interference competition. In the most common theoretical view of interspecific interactions in parasitoids, there is a unidirectional, asymmetric effect of a facultative hyperparasitoid or multiparasitoid on a “primary” parasitoid (Pimm and Lawton 1978; May and Hassell 1981; Kakehashi et al. 1984; Briggs 1993). Our results (and others’) suggest that parasitoid interactions can be more complex in at least two ways. First, both parasitoids had the potential to directly affect the other through multiparasitism. Second, one parasitoid species interfered intraspecifically as well as interspecifically by host feeding on parasitised hosts. Our results thus highlight the need to consider mechanisms of both intraspecific and interspecific interference competition in order to better understand coexistence and host suppression in parasitoid communities and biological control.

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