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Origin of an insect outbreak: escape in space or time from natural enemies?

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Abstract Initiation of insect outbreaks is poorly understood, and may involve sporadic events that temporarily release insect populations from predation or parasitism. While studying a declining outbreak of the western tussock moth (*Orgyia vetusta*) on bush lupine (*Lupinus arboreus*), we witnessed the onset of a new tussock moth outbreak, separated by 1,000 m in space and 2 months in phenological timing from the original population. This new population underwent explosive growth for 2 years and then collapsed because of a massive die-off of lupines. We tested whether during its growth phase, this new outbreak benefited by escaping in either space or time from the natural enemies attacking the original population. In experimental populations on single bushes, we compared predation and parasitism at the sites of the new and the old outbreak. At the site of the old outbreak, we compared predation and parasitism early and late in the season. Parasitism was significantly lower and population growth significantly higher at the new outbreak site than the old one. Neither seasonal timing, predator exclusion, nor their interaction significantly affected survival at either site. Thus the new outbreak appeared to escape in space from parasitism. These results corroborate our previous experimental findings, which suggest that as predicted by theory, the interaction between the tussock moth and its parasitoids can produce large-scale spatial patterning in population densities.

Keywords Altered caterpillar phenology · Escape from parasitoids · Insect outbreak · Rapid population growth · Tussock moth caterpillar

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Introduction

Despite considerable study, the factors involved in the initiation of insect outbreaks remain enigmatic. Multiple explanations have been advanced, including changes in food-plant quality (Schultz and Baldwin 1982; White 1984; Mattson and Haack 1987; Rossiter 1992), favorable weather (Martinat 1987), and reductions in predation, parasitism or disease (Strong et al. 1984; Price 1987; Myers 1988; Walsh 1990), but satisfactory tests are elusive. One approach has been to examine long time-series and correlate changes in insect abundance with environmental or biotic variables (Martinat 1987; Miller et al. 1989; Hunter 1993; Hunter and Dwyer 1998). While this has the advantage of allowing large-scale processes to be considered, its limitations include the requirement for many years of data, as well as the inability to examine detailed mechanisms. An alternative approach is to manipulate such factors as insect density, predators or food quality (Faeth and Simberloff 1981; Liebhold and Elkinton 1989; Gould et al. 1990; Myers 1990; Myers and Rothman 1995). While potentially powerful, such experiments often reveal that the factors causing outbreaks are nearly impossible to reproduce during non-outbreak conditions. A third approach, relatively seldom used, is to perform experiments during the initiation phase of a natural outbreak (Turchin et al. 1999).

In this study we performed experiments during the onset of an outbreak of the western tussock moth (*Orgyia vetusta*) on bush lupines (*Lupinus arboreus*). While studying a declining outbreak of this insect in 1997, we observed a new outbreak beginning at a site about 1,000 m from the original one. For reasons we still do not fully understand, the new outbreak was about 2 months later than the original one in phenological timing. After 2 years of explosive growth, the new outbreak collapsed in 1998 because of a massive die-off of lupines. This series of events provided us with the opportunity to test two hypotheses about outbreak initiation, which we refer to as the escape in space and the escape in time hypotheses.

Our escape in space hypothesis is based on results from our study system, suggesting that the spatial spread of tussock moth outbreaks is inhibited by parasitoids (Brodmann et al. 1997; Maron and Harrison 1997). We found that at distances less than 250 m from the edge of the original outbreak population, experimentally created populations of tussock moths were suppressed by parasitoids (mainly *Tachinomyia similis* and other tachinid flies) emanating from the natural population. At distances greater than 250 m, parasitism was much lower and survival was substantially higher. These results supported mathematical theory predicting that forceful predator-prey interactions, coupled with differences in dispersal between predators and prey, can generate strong spatial patterns in population abundance within a uniform habitat (Brodmann et al. 1997; Maron and Harrison 1997). This also suggests that the new natural outbreak may have benefited substantially by being 1,000 m from the previous population, where parasitism would be expected to be low.

Our escape in time hypothesis is that the delayed timing of the second outbreak produced substantially lower predation or parasitism because of the asynchrony between tussock moths and their natural enemies. Asynchrony in the phenology of parasitoids and their hosts was first discussed by Varley and Gradwell (1958), and more recent modeling has examined the effects of such asynchrony on the stability of host-parasitoid interactions (Godfray et al. 1994). Here we simply asked whether predation, parasitism and/or mortality were lower for the later-hatching moths of the new outbreak than the earlier-hatching ones of the original outbreak, at a single location. We also asked whether the difference in tussock moth phenology between the sites had a simple environmental cause, by rearing egg masses in common environments.

We did not test a more extensively studied phenological explanation for insect outbreaks, namely that they occur when insect hatch is well synchronized with budburst so that larvae develop at the time when host plant quality is highest (Holliday 1977; Witter and Waisanen 1978; Watt and McFarlane 1991; but see Hunter et al. 1991). Although it is evidently important for many insects that defoliate deciduous trees, this mechanism is unlikely to be important in our system. Lupine bushes are evergreen and produce new leaves over a period of many months. Egg hatch by tussock moths is also prolonged, often lasting for 5–6 weeks, and the nutritional performance of tussock moths on lupines in the field does not appear to vary greatly in space or time (Harrison 1994).

We tested the above hypotheses by creating experimental populations of tussock moths (started with 1,000 neonate larvae) on bushes protected and unprotected from terrestrial predators. To test the escape in time hypothesis, in 1997 we compared the effects of predators and parasitoids on the survival of tussock moths in experimental populations created at the old outbreak site early (i.e. the timing of the old outbreak) and late (i.e. the timing of the new outbreak) in the season. To test the escape in space hypothesis, in 1997 we compared the effects of predators and parasitoids on tussock moth survival at the original outbreak site and the new outbreak site, late in the season. We repeated this second comparison in 1998, but we could not repeat the first comparison because too few early-hatching tussock moths were available.

Materials and methods

Study system

This study took place at the University of California's Bodega Marine Reserve, a 147-ha site located along the central California coast in Sonoma County (see Barbour et al. 1973 for a description of the study site). Tussock moth larvae at Bodega feed almost exclusively on bush lupine, a short-lived woody evergreen shrub that forms dense stands within coastal prairie grasslands. Tussock moths hatch in spring from masses of 100–300 eggs, develop for 6–8 weeks through 5–6 instars, and pupate in the summer. They eclose, mate and oviposit 2–3 weeks later, and egg masses remain dormant through fall and winter. Female tussock moths are flightless, and lay their egg masses on the same bush where they pupate and eclose.

Since about 1983, a dense population of tussock moths has existed within a 1–1.5 ha lupine stand at a location called Mussel Point (MP). Adjacent areas supported few tussock moths despite the occurrence of dense stands of bush lupine. Variable host-plant quality did not appear to be a reason for the patchy spatial pattern of moth population abundance. Larvae raised on bushes inside the outbreak area and at nearby sites where larvae occurred at low density showed no differences in survival, pupal weight or development time (Harrison 1994). Similar results were found for three other tussock moth populations along the California coast (Harrison 1997). Rather, spatial spread of the outbreak appeared to be inhibited by two factors: poor dispersal by the moths and heavy parasitism just outside the boundaries of the outbreak (Brodmann et al. 1997; Maron and Harrison 1997). Lupine bushes at MP were found to be highly resilient to annual defoliation (Harrison and Maron 1995).

Natural enemies of the tussock moth at Bodega include generalist predators such as ants and other invertebrates, which have the strongest effect on early-instar larvae (Harrison 1994, 1997; Harrison and Wilcox 1995). Later instars are attacked by parasitoids, the most common of which are the tachinid flies *Tachinomyia similis* and *Patelloa pleuriseriata*; others include the tachinid *Protodejeania echinata* and various Hymenopterans (Brodmann et al. 1997). *T. similis* parasitism, in particular, can be very high. This parasitoid is univoltine and it parasitizes several species of Lepidoptera, although at our site it specializes on *O. vetusta*. Parasitism on later larvae and pupae is so heavy that the population-level effects of predation on early instars are obscured (Maron and Harrison 1997). That is, parasitism is so intense late in the season that any early-season impact of predators on caterpillar numbers can no longer be detected by the time larvae pupate. Tussock moth eggs are parasitized by the scelionid wasp *Telenomus californicus*.

The tussock moth outbreak at MP has been steadily declining in density since 1992 (S. Harrison, unpublished data). In 1996, a small population of tussock moths on lupine bushes 1 km to the south of MP was observed, at a site we termed Lab Road (LR). By 1997 this population had exploded into an extensive new outbreak, with the number of late-instar caterpillars per lupine bush averaging 504±322 (mean ± SD; n=15). This new outbreak occurred 2 months later than the one at MP; egg masses began hatching in June, at the same time that larvae in the original outbreak were pupating. Observations clearly indicated that the new outbreak was not a second generation; both populations were univoltine. The reasons for its later phenology remain unknown. However, tussock moth phenology is variable from site to site

along the California coast (Harrison 1997), and the nearest known population to Bodega (at Point Reyes, about 30 km away) has the same late phenology as the new outbreak at Bodega. By August 1997, the new outbreak had spread over an area of about 10 ha, and all lupine bushes within this area were completely defoliated. Nearly all of these defoliated bushes died by spring 1998.

Escape in space experiment

The escape in space hypothesis is that mortality from predators and/or parasitoids should be lower at the site of the new outbreak than the old one, regardless of seasonal timing. To test this, in mid-June 1997 and late May 1998, we compared survival of experimental tussock moth populations on lupine bushes at the original outbreak site (MP), and at the new outbreak site 1,000 m to the south (LR). At each location, 1,000 neonate larvae were added to each of 10 (1997) or 16 (1998) pairs of similarly sized lupine bushes, a density that is within the naturally observed range (Harrison 1994 and unpublished data). Within a location, we chose pairs of lupine bushes that were widely spaced, to increase the likelihood that predation and parasitism would be independent between bushes. Larvae were laboratory-reared from eggs collected the previous fall in the new outbreak. We surrounded one bush of each pair with 30-cm-high circular fences of sheet metal, dug 30 cm into the ground and topped with Tanglefoot (Tanglefoot, Grand Rapids, Michigan, USA), to exclude terrestrial predators such as ants, beetles and mice. One ant stake (Grant Laboratories, San Leandro, Calif., USA) was placed inside of each fence. Control bushes had similar fences, but with four 10-cm-wide gates and no Tanglefoot or ant stakes.

Starting on 1 July (1997) or 1 June (1998) we censused larvae on each bush every week until all had pupated (1997=5 weeks, 1998=8 weeks). We recorded the number of larvae with the conspicuous white eggs of *Tachinomyia similis* on their cuticles. Although this does not provide an estimate of total *T. similis* parasitism, since larvae appear unparasitized after their next molt or when *T. similis* eggs hatch, it does provide an index of *T. similis* parasitism that can be compared among treatments. We also estimated parasitoid abundance by counting the number of *T. similis* flies that were caught in Tanglefoot-coated predator exclusion fences at the old and new outbreak sites. At the end of the experiment, another index of parasitism was obtained by counting parasitized larval and pupal corpses under the experimental bushes.

To obtain a rough estimate of the relationship between observed and total parasitism at MP, we made weekly collections from a pool of 800 larvae that had been placed on two lupine bushes at MP starting on 4 June 1998. Collections started on 10 June and ran for 7 weeks. We reared collected larvae in the laboratory, and each week recorded the number of previously collected larvae that died from parasitism. Our field estimates of parasitism were then adjusted by multiplying the number of apparently healthy larvae from field counts (that is, those lacking *T. similis* eggs) by the fraction of larvae collected in any week that later died from parasitism. Larvae with eggs of *T. similis* were found to have the same probability of being attacked by other parasitoids as did larvae lacking *T. similis* eggs (M. Greaves et al, unpublished data).

By the first week in July (1997), larvae in the new natural outbreak at LR were severely defoliating lupine bushes and crawling off defoliated bushes. To prevent these larvae from colonizing our control bushes at LR, on 4 July we closed the gates on all control plants (at both LR and MP) for the duration of the experiment. In 1998 we closed the gates on control bushes in the last week of June to prevent their colonization by larvae from the new outbreak that were searching for the few remaining live host plants at the site. When most experimental larvae had either died or pupated, we counted surviving pupae under our experimental bushes (on 8 August 1997, or 11 August 1998). By late August all pupae had eclosed; on 28 August (1997) or 8 September (1998) we counted the number of egg masses on each bush as well as the number of parasitized corpses under each bush.

Analysis

To test for an effect of site and predation in 1997 and 1998, we used ANOVAs with numbers of larvae at the second week of the experiment as the dependent variable, and site (MP or LR), treatment (predator exclusion or control), and their interaction as independent variables. Second-week census numbers were used because previous results revealed that predators had their greatest impacts on small larvae (Maron and Harrison 1997). Escape from predators in space would be indicated if the difference in larval numbers between the predator-exclusion (fenced) and control (gated) treatments were greater at MP than at LR.

To compare how levels of parasitism by *T. similis* varied by location in 1997 and 1998, we performed separate repeated measures ANOVAs on each year's data. In 1998, some bushes died mid-way through the experiment (1 at MP, 3 at LR), thereby creating unequal sample sizes between sites. To equalize the sample sizes for this analysis, two additional data points at MP were selected at random and deleted from the data set.

To test for an effect of site on parasitism and on overall survival in 1997, we used a MANOVA with dependent variables that included the number of corpses that had been parasitized by *T. similis*, the number of surviving pupae (log transformed), and the number of egg masses on experimental bushes. Independent variables were site, predator exclusion treatment and their interaction. In 1998, this analysis could not be repeated because of unequal sample sizes for the different dependent variables, which makes the use of MANOVA problematic (Scheiner 1993). Therefore, separate ANOVAs were used to test the effects of site, predator exclusion, and their interaction on the number of surviving pupae per bush. We found almost no corpses under control (gated) bushes, so we used a one-way ANOVA to test the effect of site on the number of parasitized corpses found under the predator-exclusion (fenced) bushes. SYSTAT 8.0 (SYSTAT 1998) was used to analyze all data.

The above analyses assume that bushes within a site are independent with respect to predator and parasitoid foraging. For ant predators, this is certainly the case. We do not fully understand, however, the details of Tachinid fly parasitoid movement patterns. Preliminary mark and recapture data suggest that *T. similis* parasitoids tend to concentrate their activities around single lupine bushes that contain high densities of caterpillars (J. Umbanhower, unpublished data). However, these parasitoids undoubtedly search for hosts over some broader spatial scale as well. As such, a caveat about our analysis is that we do not know for certain the degree to which parasitism rates of tussock moths on separate experimental bushes are truly independent.

Escape in time experiment

The escape in time hypothesis predicts that mortality from predators and/or parasitoids should be lower during the time of the new outbreak, i.e. later in the season, regardless of spatial location. To test this, we compared larval survival and parasitism in the experimental populations established at MP late in the season (June 1997; described above) with survival during a similar experiment that was initiated early in the season (April 1997; see Maron and Harrison 1997 for details). This earlier experiment was set up identically to the one just described, and 10 pairs of fenced or gated bushes were inoculated with 1,000 tussock moth larvae in late March 1997. Starting on 19 April, we censused larvae on bushes every week, and at the end of the season we counted the number of pupae and the number of egg masses on the bushes. To prevent wandering by late-instar larvae, gates were closed on control bushes on 8 May.

Analysis

To test the effect of seasonal timing on predation, we used a two-way ANOVA, with the number of larvae surviving to the second

week of the experiment as the dependent variable, and time (early or late season), treatment (predator exclusion or control), and their interaction as independent variables. To test the effect of seasonal timing on survival, we used a MANOVA with the number of surviving pupae, number of parasitized corpses and egg masses as the dependent variables, and with the same independent variables as used in the ANOVA. To determine how levels of parasitism changed depending on timing of caterpillar hatch, early- and late-season parasitism at MP was compared using a repeated measures ANOVA.

To determine whether the difference in phenology between populations was environmentally caused, in 1997 we pinned 20 egg masses from the new outbreak at LR to bushes within the site of the old outbreak at MP, and we also pinned 20 control egg masses from LR to bushes at LR. Several hundred egg masses from MP were also reared in an outdoor cabinet near the laboratory, and the hatching phenology of these egg masses were compared with that of natural egg masses at MP.

Results

Escape in space

In 1997, predation appeared to affect the survival of early-instar larvae, as shown by significantly higher larval numbers during the first few censuses on predator-exclusion than control bushes (Table 1; Fig. 1). However, this effect did not differ between the site of the old and new outbreaks, as shown by the lack of a treatment by site interaction (Table 1). This initial effect of predator exclusion did not affect the numbers of tussock moths surviving to pupation (MANOVA, Wilks' Lambda =0.97, $F_{3,14}$=0.33, P=0.93).

Parasitism, in contrast, was sharply different between the two sites. Observed parasitism was lower (rmANOVA, $F_{1,18}$=9.8, P<0.01; Fig. 2), and final survival to pupation higher by about fivefold, in the new outbreak site (Table 2). There were significantly more parasitized corpses under experimental bushes at the old outbreak site than the new outbreak site (Table 2). More parasitoid flies were caught on Tanglefoot-coated predator-exclusion fences at the old outbreak site than the new outbreak site (Table 2).

In turn, there were significantly more progeny egg masses per experimental bush at the new outbreak site than the old outbreak site (Table 2). Even with protection from ground-based predators, initial densities of 1,000 larvae (or 7–10 egg masses) yielded an average of less than one egg mass per bush at the site of the old outbreak. In contrast, the yield at the new outbreak site averaged 15.8 egg masses per bush.

In 1998, there was no effect of predator exclusion on the number of larvae per bush (Table 1). The percentage of larvae that were visibly parasitized by *T. similis* was again significantly greater at the old outbreak compared to the new outbreak (rmANOVA, $F_{1,24}$=22.6, P<0.0001; Fig. 2). In an observational study, significantly more *T. similis* were counted on bushes at the old than the new outbreak site (J. Umbanhowar, unpublished data). In turn, numbers of surviving pupae were significantly higher at the new outbreak site (Table 3). Including all

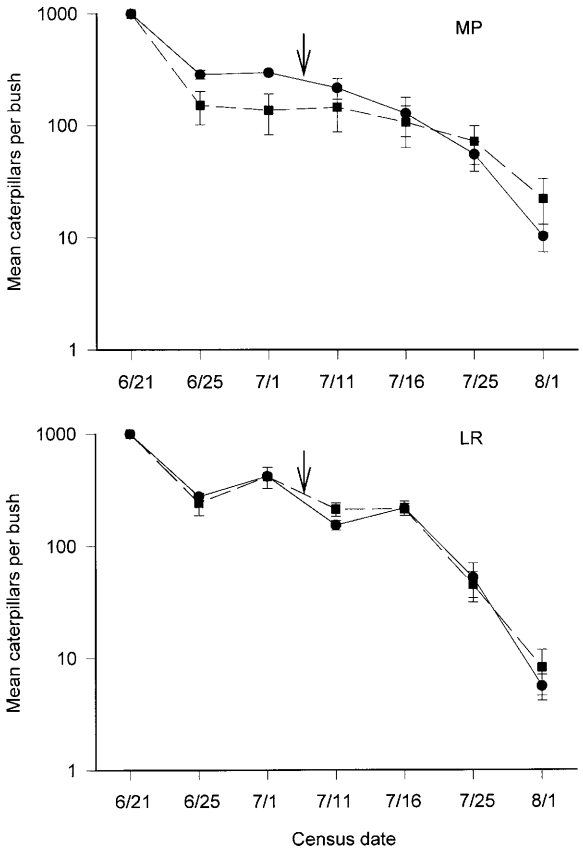

Fig. 1 Mean (± SE) abundance of tussock moth larvae from experimental populations placed on single lupine bushes at Mussel Point (MP) and Lab Road (LR) late in the season in 1997, at the time of the second outbreak. *Circles* Bushes protected from ground-based predators, *squares* bushes exposed to predators. *Arrows* indicate when gates on control fences were closed. Experimental populations started with 1,000 caterpillars per bush, n=5 populations per treatment. *MP* is site of historic outbreak, *LR* is the site of the new tussock moth outbreak

Table 1 Effect of site (1997and 1998) or timing (early or late in season) and predator exclusion on mean number of larvae per bush

Source	*df*	MS	*F*	*P*
1997				
Site	1	7,566	0.96	0.34
Predator exclusion	1	35,364	4.5	0.05
Site × Exclusion	1	12,751	1.6	0.22
Error	16	7,841		
1998				
Site	1	12,474	1.98	0.17
Predator exclusion	1	2,249	0.35	0.55
Site × Exclusion	1	1,394	0.22	0.64
Error	16	6,274		
Timing				
Time	1	7,411	0.65	0.43
Predator exclusion	1	93,982	8.2	0.01
Time × Exclusion	1	3,354	0.3	0.6
Error	16	11,488		

parasitoids, larval parasitism rates at MP were approximately twice that of rates of parasitism by *T. similis* alone. However, at both sites pupal mortality from parasitism was so high that there were almost no progeny egg masses at either site (old outbreak site, 1 mass under each of 2 bushes, out of 15 bushes; new outbreak site, 2 egg masses under 1 of 16 bushes).

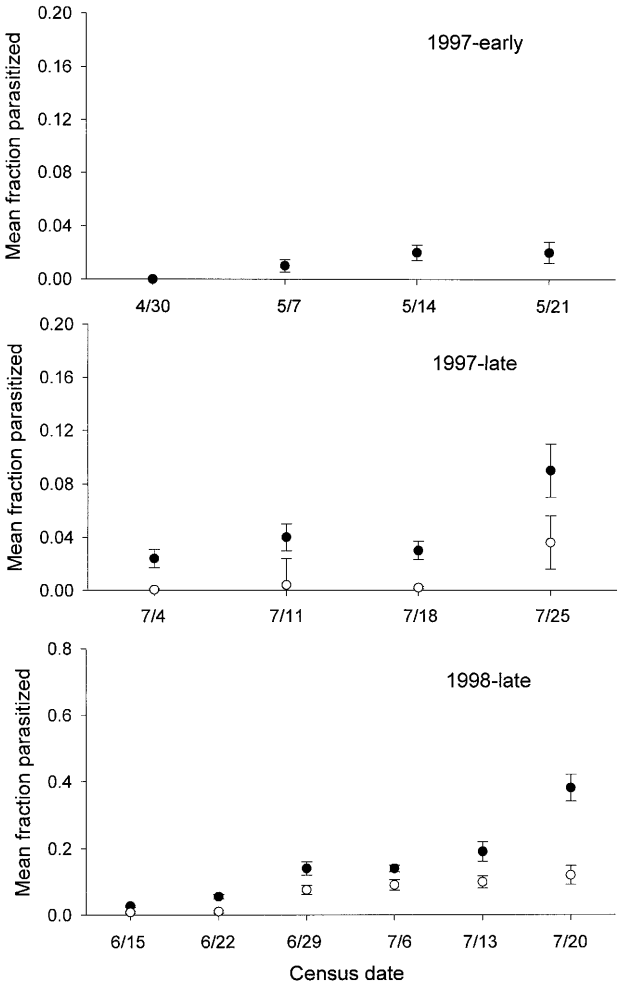

Fig. 2 Mean (± SE) fraction of larvae in experimental populations carrying an egg of the tachinid parasitoid, *Tachinomyia similis. Closed circles* MP site, *open circles* LR site. *Top and middle panels*, *n*=10 populations; *bottom panel*, *n*=13–15

Escape in time

Predator exclusion significantly increased the survival of early-instar larvae, both early and late in the season in 1997 (Table 1, Fig. 3). However, the magnitude of predation was not significantly different early versus late in the season, as evidenced by the fact that there was no significant time by treatment interaction (Table 1). There was no effect of predation or time on the number of surviving pupae or the number of egg masses (MANOVA, $P>0.3$ for time, treatment and their interaction). There was also no effect of time on the percentage of larvae that became parasitized (rmANOVA, $F_{1,18}=0.12$, $P=0.74$).

All egg masses collected from the new outbreak site in 1997 hatched late in 1998, at the same time as the natural population at the new outbreak site, regardless of whether they were field-reared at the old or the new site. All egg masses collected from the old outbreak site in 1997 hatched early in 1998, at the same time as the natural population at the old site, whether they overwintered at the old site or in the outdoor closet near our laboratory.

Discussion

Our results support the hypothesis that a spatial escape from parasitoids facilitates the growth of incipient local outbreaks. In 1997, the first year of a large new out-

Table 3 Two-way ANOVA testing effect of site (near old or new outbreak) and predator exclusion on mean number of pupae per bush

Source	*df*	MS	*F*	*P*
Site	1	295.4	7.6	0.01
Predator exclusion	1	127.5	3.3	0.08
Site × Exclusion	1	0.56	0.01	0.91
Error	23	39.1		

Table 2 Effect of site (near old or new outbreak) on parasitism and survival of tussock moths in experimental populations in 1997. Univariate analyses performed after significant MANOVA (Wilks' Lambda =0.27, $F_{3,14}=12.6$, $P<0.001$) with all variables combined

Source	*df*	MS	*F*	*P*
Parasitized corpses/bush	1	551	12.5	0.003
Error	16	43.9		
Pupae/bush	1	16.9	20.5	0.001
Error	16	0.82		
Egg masses/bush	1	1140	9.4	0.007
Error	16	121		
	Old outbreak site	New outbreak site		
Mean (± SE) parasitized corpses per bush	11.3±2.8	0.8±0.36		
Mean (± SE) parasitoid flies caught	13.1±2.8	5.4±1.6		
Mean (± SE) egg masses per experimental bush	0.7±0.47	15.8±4.7		

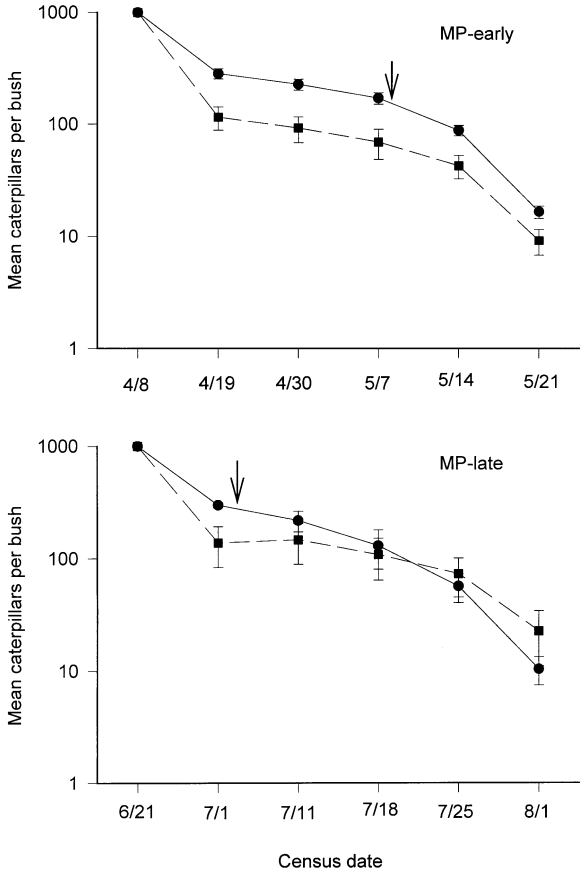

Fig. 3 Mean (± SEM) abundance of tussock moth larvae from experimental populations placed on single lupine bushes. *Circles* Bushes protected from ground-based predators, *squares* bushes exposed to predators. *Arrows* indicate when gates on control fences were closed. For each treatment, *n*=5 populations

break, our experimental populations survived substantially better at the site of the new outbreak than at the site of the declining previous outbreak. Several lines of evidence implicated a spatial difference in parasitism as the reason for this effect. Visibly parasitized larvae, parasitized larval and pupal corpses under bushes, and observed parasitoids all were more numerous at the site of the old outbreak than the new one. In contrast, rates of predation (as estimated by the difference in larval survival on protected and exposed bushes) did not differ between the two sites. All of these results were consistent in 1997 and 1998. Phenology, in contrast, did not appear to affect either parasitism, predation or survival. At the site of the old outbreak in 1997, none of these vital rates were affected by the seasonal timing of the old outbreak (April–May) versus the new one (June–July).

These results are consistent with our earlier work showing that experimental propagules of tussock moths experienced substantially lower parasitism and higher survival when they were placed greater than 250 m from the existing outbreak population (Maron and Harrison 1997). We concluded that the observed "sharp edges" and spatially localized distribution of tussock moths may be caused by parasitoids, as predicted by theories of spatial pattern formation (Deutschmann et al. 1993; Holmes et al. 1994; Tilman and Kareiva 1997). We further hypothesized that new tussock moth outbreaks might begin when their propagules were transported further away from an existing population than their parasitoids could disperse. Our present results from a more natural situation corroborate some aspects of this scenario. However, since we could not replicate new and old outbreak sites, it is possible that some unknown difference between sites (besides parasitism) contributed to the results we observed. Furthermore, we cannot say with certainty that the new outbreak we observed came from a transported propagule, rather than from the growth of an existing low-density population. Nor can we say that reduced parasitism was the reason for the initial growth of the new outbreak; it may have begun to grow for some other reason, with reduced parasitism following as a consequence of its suddenly increased size. However, we can say with some confidence that rates of parasitism were highly heterogeneous in space, at a scale that is highly consistent with our earlier results, and that the new outbreak benefited greatly from this heterogeneity. We speculate that parasitism would have caught up with and ended the new outbreak after several generations of parasitoid population growth, had the outbreak not been terminated sooner by mass mortality of the host plant.

We still have no definitive explanation for the difference in hatching phenology between the two sites. However, our experiments indicated that environmental differences were unlikely to be the cause, and we are currently investigating a possible genetic basis. Whatever its explanation, the shift in phenology is unlikely to have aided the initiation of the outbreak, since our results showed it was not associated with increased survival. In the longer term, the phenological shift apparently doomed the new outbreak to collapse by starvation. At the new outbreak site, heavy late defoliation in 1997 plus flooding in winter 1998 killed the vast majority of lupine bushes. As a result, most newly hatched tussock moths in 1998 starved; as such, few egg masses were produced at the new outbreak site in 1998. In contrast, at the old outbreak site, even very heavy defoliation earlier in the season rarely killed lupine bushes (Harrison and Maron 1995; Strong et al. 1995). In our experiments in 1997, plants defoliated late in the season died with equal frequency at the old and new sites. Hence the deaths of lupines at the new site were probably caused by the late timing of defoliation, rather than by the plants at this site being less vigorous. In turn, the massive die-off of lupine at the new site may explain why parasitism rates at this site were much higher in 1998 than 1997. In 1998, most naturally occurring larvae starved early in the season because all the host plants were dead. Therefore our experimental larvae became virtually the sole hosts for a large, late-emerging parasitoid population, which virtually annihilated the moths just around the time of pupation.

Like other outbreaking insects, tussock moths occur in many sites at low densities, and in a few sites at very high

densities (Harrison 1997). Our results indicate that parasitoids play a strong role in maintaining this patchy distribution, in that they can effectively suppress incipient outbreaks and limit the spatial spread of existing outbreaks (Maron and Harrison 1997). More generally, our results support the proposition that the interplay of species interactions, limited dispersal, and habitat geometry can generate a wide array of patterns in nature (Tilman and Kareiva 1997; Cronin et al. 2000). Such spatial theory has still been relatively little applied to the understanding of real ecological phenomena such as insect outbreaks. However, Roland and Taylor (1997) recently showed that forest fragmentation leads to lower parasitism and longer-lasting outbreaks of tent caterpillars in Canadian forests, possibly through altering the movement behavior of tachinid parasitoids. Cappuccino and her coworkers (Cappuccino and Martin 1997; Cappuccino et al. 1998) have likewise shown that forest structure may affect the parasitism rates and survival of forest defoliators. Also, Sharov and Liebhold (1998) have used spatial modeling to examine alternative strategies for slowing the spread of the gypsy moth. Thus we suggest it may be worth broadening the traditional set of questions asked about insect outbreaks, to include more exploration of how spatial processes can affect outbreak growth, spread and collapse.

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