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Intraspecific competition and density dependence in an *Ephestia kuehniella–Venturia canescens* laboratory system

Stephen D. Lane and Nicholas J. Mills

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A model host-parasitoid system of *Ephestia kuehniella* and *Venturia canescens* was used to examine the influence of host and parasitoid density on host and parasitoid life-history parameters via a two-way factorial experimental design (5 initial host densities × 3 parasitoid densities). In the absence of parasitoids, *E. kuehniella* experienced scramble-type competition with reduced growth, diminished adult size and a subsequent fecundity trade-off for mortality. The mortality that did occur was confined to the late larval and pupal stages. In the presence of parasitoids attacking the late larval stage, competition changed from scramble for food to contest for enemy-free space, with hosts escaping parasitism being small with low fecundity and reduced egg size, and with parasitoid adult size inversely dependent on host density. Total insect emergence (host + parasitoid), a measure of the influence of host resource competition on survivorship, exhibited a threshold effect as a function of initial host density; the threshold value was increased to a higher initial host density in the presence of parasitoids. Models of host self-limitation were fitted to the data, with the generalized Beverton-Holt model that incorporates a threshold effect providing the best fit, and the Ricker model with no threshold providing a very poor fit to the data.

S. D. Lane and N. J. Mills, Dept of Integrative Biology, and Dept of Environmental Science, Policy and Management, Univ. of California, Berkeley, CA 94720-3112, USA (nmills@nature.berkeley.edu).

The history of the study of population regulation has been characterized by a wide diversity of opinions about its presence, causes and consequences (Nicholson and Bailey 1935, Andrewartha and Birch 1954, Hanski 1990, Murdoch 1994, Turchin 1995, den Boer and Reddingius 1996, Getz 1996). While it is certainly true that density-independent mechanisms (primarily disturbance) can play a central role in the regulation of populations (Turchin 1995, Ritchie 1996, Huffaker et al. 1999), most of the focus of research on population regulation has been to elucidate density-dependent mechanisms operating on populations, internally (intraspecific competition) and/or externally (interspecific competition, density-dependent predation and/or parasitism; den Boer 1990, Latto and Bernstein 1990, Hochberg 1991, Floyd et al. 1996, Lynch et al. 1998, Bonsall et al. 1999, Huffaker et al. 1999). Since the world is populated by organisms, but not overrun by them, there must be mechanisms by which population growth rates are positive when population densities are low, and negative when they are high. The precise nature of how this occurs has been the subject of much debate.

In the realm of biological control, understanding the role of population regulation is of particular importance. In classical biological control introductions (hereafter referred to simply as biological control), potential control agent(s) are introduced at low densities, with the intention that they will establish (i.e. initially experience positive population growth rates), control the pest (i.e. cause the pest's realized population growth rate to become negative), and then settle into a stable relationship with all populations persisting at very low

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densities. Much of the focus on why biological control work has been on density-dependent processes induced by the interaction between the pest and potential control agent populations (Hassell 1978, 2000, May et al. 1981, Barlow and Wratten 1996, Mills and Getz 1996, Briggs et al. 1999, Takagi 1999, Bernstein 2000, Mills 2000).

Parasitoids are the most frequently used group of control agents in biological control projects, due largely to their high level of target-pest specificity (Greathead and Greathead 1992, Mills 2000). For this reason, some of the theory behind pest-parasitoid population dynamics is briefly considered here. Pest-parasitoid models often omit explicit density dependence in the pest population, for the purpose of focusing on density-dependent regulatory mechanisms inherent in the pest-parasitoid interaction (Briggs 1993, Getz and Mills 1996, Shea et al. 1996). It must be the case, however, that such intraspecific-competitive processes occur, because, as pest densities increase towards some threshold level, the rate of resource depletion becomes greater than the rate of resource renewal. The critical questions in this regard are: over what range of densities do such processes occur, and how are they relevant to the population dynamics of the pest-parasitoid interaction (Barlow and Wratten 1996, Getz 1996, Mills and Getz 1996, Hochberg and Holt 1999). Recently, Mills (2000), as part of a specification of minimum requirements for host-parasitoid models, included self-limitation (density dependence) for the host population as one of the fundamental features of these models if they are to be used for the purpose of examining biological control scenarios.

There has been a great deal of debate in the host-parasitoid theoretical literature over the last 30 years about the impact of variation in parasitoid density on host-parasitoid population dynamics (Barlow and Wratten 1996, Mills and Getz 1996, Briggs et al. 1999, Bernstein 2000, Hassell 2000). This work has led to the "$CV^2 > 1$" rule for the stability of the host-parasitoid interaction, where "CV" is the coefficient of variation of searching parasitoids per discrete patch of hosts (Taylor 1993). As both Mills and Getz (1996) and Hassell (2000) point out, however, the generality of this result requires that the host population not experience self-limitation, and that the functional response of the parasitoid be linear. As the generality of both these assumptions is questionable (Barlow and Wratten 1996, Getz 1996, Getz and Mills 1996, Mills and Getz 1996, Lane et al. 1999), it thus becomes even more important to understand at a mechanistic level how density dependence influences the dynamics of pest-parasitoid populations.

The Mediterranean flour moth, *Ephestia* (= *Anagasta*) *kuehniella* Zeller (Lepidoptera: Pyralidae) and its solitary larval endoparasitoid, *Venturia* (= *Nemeritis*) *canescens* Gravenhorst (Hymenoptera: Ichneumonidae) have proven to be a valuable model system for the study of host-parasitoid interactions supported by a substantial body of literature (Norris 1933, Ullyett 1945, Ullyett and van der Merwe 1947, Salt 1965, 1966, Takahashi 1968, White and Huffaker 1969a, b, Hassell 1978). Recent ecological work has addressed a wide variety of topics, from tests of ideal free distribution theory (Tregenza et al. 1996), to the examination of host suitability and the consequences of superparasitism (Harvey 1996, Harvey and Vet 1997, Sait et al. 1997), and the demonstration of apparent competition (Bonsall and Hassell 1998). In an effort to build further on this tradition, this model system was used for two experiments designed to determine the effects of variation in host and parasitoid density on both host and parasitoid life-history parameters. In the first experiment, *E. kuehniella* was reared at five initial densities to determine the effects of intraspecific competition on host survival, growth and fecundity. In the second, the same five initial host densities were exposed to two different densities of *V. canescens* to determine the effects of parasitoid density on host and parasitoid survival and growth, and on host fecundity. The data were then used to parameterize several different versions of the host self-limitation function (Getz 1996).

Materials and methods

Rearing and experimental protocols

E. kuehniella and *V. canescens* were obtained from colonies maintained by the insectary and quarantine facility at the University of California, Berkeley. The *E. kuehniella* colony was mass-reared in plastic trays ($47.0 \times 36.8 \times 3.8$ cm) on a diet of semolina. *E. kuehniella* passes through five larval instars (Harvey and Vet 1997). The *V. canescens* colony was reared on 3rd–5th instar *E. kuehniella* larvae in clear plastic boxes ($17.8 \times 12.7 \times 6.7$ cm). *E. kuehniella* and *V. canescens* used in experiments were maintained at 27.3 ± 0.1°C (mean $\pm$ SE), 72.5 ± 12.5% RH (mean $\pm$ range), and L14:D10. Naïve *V. canescens*, with no access to hosts, were provided with honey prior to exposure to hosts, and females of ages 1–3 days were used in experiments.

Experiments were conducted in 0.47 l unwaxed paper containers with clear plastic lids (obtained from AC Paper and Supply Co., Berkeley, CA), to which 5.00 ± 0.04 g semolina (mean $\pm$ range) was added. A set of 32 containers was used for each of five initial densities of *E. kuehniella* eggs (IHD $\equiv$ initial host density): 10, 45, 80, 115 or 150 eggs, as determined by direct count (IHD 10 only), or by weight (the appropriate range of IHDs for these experimental conditions was determined through pilot studies). Mean $\pm$ SE egg weight was 26.1 ± 0.5 µg for both the *E. kuehniella*-only experiment ($n = 75$) and the *E. kuehniella* + *V. canescens* ex-

periment ($n = 20$; all weights reported in this study were obtained using a Mettler Toledo UMT2 microbalance).

Egg hatch failure of *E. kuehniella* (i.e. density-independent egg mortality) was assessed by placing 100 eggs into each of eight 0.47 l unwaxed paper containers with clear plastic lids. All hatching larvae were counted. For purposes of data analysis and presentation all hatching was assumed to have occurred on day three after egg laying. It is possible that some small but significant proportion of eggs in this assessment failed to hatch due to desiccation (no evidence of cannibalism in newly-hatched larvae was observed), but newly-hatched larvae proved impossible to locate when eggs were placed in diet.

In the first experiment (parasitoids absent), populations were monitored in one of two ways. For juvenile stages, containers were destructively harvested as the experiment progressed, at the peaks of the mid-larval, late-larval and pupal stages (these peaks varied with initial host density and were determined through pilot studies). A single destructive harvest involved opening a random selection of 8 containers of each initial host density. All *E. kuehniella* larvae and/or pupae were removed, counted and then weighed to obtain a mean fresh weight for each container. A further 8 containers of each initial host density were used to monitor adult *E. kuehniella*, which were collected on a daily basis from the day of first emergence until emergence ceased. To assess host fecundity, 8 mating pairs from each initial host density (one pair per container) were removed from the containers whilst in copula and kept in 150×25 mm glass tubes capped with window screen mesh until both members of the pair died. The tubes were inverted and placed in small cups containing a thin layer of very fine-ground pastry flour, to cue female moths not to lay eggs elsewhere in the test tube. Single mating pairs were used, as Daumal and Boinel (1994) have shown that there is no effect of adult moth density on egg laying in *E. kuehniella* when an oviposition site is available for each female. Eggs from these mated pairs were collected daily via sifting the pastry flour, and the total fecundity and mean fresh egg weight was measured for each female moth. Hatching success of host eggs resulting from experimentally-obtained mated host pairs was not assessed. Additionally, twenty adults from each initial host density were fresh weighed (2 or 3 adults taken at random from each container; small per-container sample sizes were used due to low emergence rates in some treatments). Data from this experiment were used to examine host survivorship and growth in the absence of parasitoids, as well as to parameterize several different versions of the host self-limitation function.

In the second experiment, one or three female *V. canescens* were exposed to *E. kuehniella* at the peak of the late-larval stage ($n = 8$ for each initial-host-density $\times$ parasitoid-density combination.) Parasitoids were not given honey subsequent to host exposure, and typically lived for 4–6 days total after emergence (host exposure to parasitoids was thus 1–3 days). Adult *E. kuehniella* counts, fresh weights, emergence times, mated pairs and eggs were collected as in the first experiment. In addition, adult *V. canescens* were collected on a daily basis, counted, and fresh weighed as outlined above for *E. kuehniella*. The fecundity of parasitoids resulting from experimental treatments was not assessed (but see Harvey et al. 2001 for data on *V. canescens* fecundity with a different host). In some cases, host and/or parasitoid mortality prevented collection of complete replicates for some experimental combinations; these are noted in the results. Data from this experiment were used in conjunction with the data from the first experiment to examine the effects of variation in initial host density and parasitoid density on host-parasitoid life history parameters.

Statistical analysis

All data were initially subjected to two-way analysis of variance (ANOVA; Underwood 1997), to examine the effects of initial host density and host stage or parasitoid density on the response variable, using specific transformations (given with results) where necessary. The data were subsequently subjected to linear regression and/or multiple comparison, as appropriate. Analysis and presentation of emergence time data was performed on the median value for each container. Unless otherwise noted, significance was determined at the $\alpha = 0.05$ level, and means are accompanied by $\pm$ standard error of the mean.

Where ANOVA indicated a significant interaction effect, P-values for the main effects are not presented, and linear regressions on initial host density were applied separately for each of the parasitoid treatments (Sokal and Rohlf 1981). In some cases where ANOVA did not indicate a significant interaction, linear regression, while not necessary to explain main effects, was computed for the purpose of plotting graphical linear fits in figures. Linear regressions were computed using the residual sum-of-squares minimization approach for more than one value of y per value of x, the linear model being tested against the deviations from regression with no mean square pooling (Sokal and Rohlf 1981, SPlus4 1997). Where regression was not significant, the mean value of the data was plotted. When the slope of a linear regression was significantly different from zero, but there were also significant deviations from the regression, the linear model represents a significant linear trend, but some caution is noted in that these are not strictly linear relationships. Correlations were computed using Pearson's product-moment correlation (Sokal and Rohlf 1981, SPlus4 1997).

Where linear regression did not provide a significant model of the data, simultaneous mean difference 99% confidence intervals were computed via simulation (Edwards and Berry 1987, SPlus4 1997). Multiple comparisons were used both vertically, between treatments at a given host stage or initial host density; and horizontally, between host stages or initial host densities for a given treatment.

Fitting models to the data

Data from the first experiment were used to parameterize the host self-limitation or density-dependence function, $g(N)$, which models the per capita population growth rate of the host in the absence of the parasitoid. In the equation for population growth $N_{t+1} = N_t g(N_t)$, (where N_t is the density of the population at time t), it is possible to decompose the function $g(N)$ into components for survivorship and fecundity, so that $g(N) = s(N)b(N)$, where s and b are respectively the density-dependent survival and birth rates in the absence of parasitoids. This then allows models of $g(N)$ to be fitted to data which are the product of survivorship and fecundity. For each initial host density in the first experiment, the product of the mean survivorship and mean fecundity ($n = 8$) was used as a single data point for the purpose of model fitting; this data set thus contained five data points corresponding to each of the five initial host densities. Asymmetric confidence intervals (CI) for these data points were computed via the delta-method (Schervish 1997, T. Strounkov, pers. comm.):

$$\mathrm{CI}_{\mu_x\mu_y} = [\mu_x\mu_y \exp(-r),\ \mu_x\mu_y \exp(r)],$$

$$r = z\sigma_{\log(\mu_x\mu_y)},$$

$$\sigma_{\log(\mu_x\mu_y)} = \sqrt{\left[\frac{1}{n_x - 1}\left(\frac{\sigma_x}{\mu_x}\right)^2 + \frac{1}{n_y - 1}\left(\frac{\sigma_y}{\mu_y}\right)^2\right]} \qquad (1)$$

where μ_x and μ_y are the means, σ_x and σ_y the standard deviations and n_x and n_y the sample sizes of the survivorship and fecundity distributions; r is the confidence radius; z is the critical value of Student's t-distribution with infinite degrees of freedom and the desired α-level; and $\sigma_{\log(\mu x\mu y)}$ is the standard error of the log of the product of the two means.

Gauss-Newton non-linear regression (residual sum-of-squares minimization; SPlus4 1997) was used to fit four models of $g(N)$ to the per capita *E. kuehniella* population growth rate data (the linear, Ricker, generalized Ricker and generalized Beverton and Holt models; see Getz 1996 for details). The goodness-of-fit criterion for each of the four models was a modification of the method described by Hilborn and Mangel (1997, eq. 5.11). This method makes use of the formula $\mathrm{SSQ}_{\mathrm{adj}} = \mathrm{SSQ}_{\mathrm{res}}/(n - 2m)$, where $\mathrm{SSQ}_{\mathrm{res}}$ is the residual sum-of-squares of the regression, n is the number of data points, and m is the number of parameters in the model. Smaller $\mathrm{SSQ}_{\mathrm{adj}}$ indicates a better fit of the model to the data. Because $\mathrm{SSQ}_{\mathrm{adj}}$ is intended only as a relative measure of the goodness-of-fit of these models (no P-values were calculated for these regression coefficients), m was modified to be the number of parameters in the fitted model less two (since $n = 5$). Thus, for the linear and Ricker models, $m = 0$; for the generalized Ricker and generalized Beverton and Holt models, $m = 1$.

As an additional goodness-of-fit criterion for the four models, 5000 data sets of $n = 5$ were generated via bootstrapping from the original data, using the same product-of-means method described above (Davison and Hinkley 1997, Hilborn and Mangel 1997). Again, for comparison purposes only, $\mathrm{SSQ}_{\mathrm{adj}}$ were calculated for each of the models for each of these 5000 data sets, and the proportion of data sets for which each model provided the best fit (i.e. smallest $\mathrm{SSQ}_{\mathrm{adj}}$) was assessed (after Hilborn and Mangel 1997, Ch. 6).

Results

Effect of initial host density on age-specific host survivorship and growth in the absence of parasitoids

Egg hatch-success of *E. kuehniella* was 0.85 ± 0.09, independent of initial host density (Fig. 1A, Hatch). A pronounced effect of initial host density on host development rate required destructive sampling of higher initial host densities at increasingly later dates after experiment initiation in order to obtain samples from the appropriate juvenile stages (Fig. 1). A significant interaction was found between initial host density and host stage in their effects on host survivorship in the absence of parasitoids (Fig. 1A; ANOVA, $F_{16,\ 175} = 14.19$, $P < 0.001$; survivorship data were arcsin transformed). A multiple comparison test (approximated critical point 3.78) indicated no difference in survivorship at the mid-larval stage, and that survivorship in all of the treatments except for IHD 10 was the same at the late-larval and pupal stages. Only at the adult stage was survivorship significantly less in the IHD 80, 115 and 150 treatments than in the IHD 10 and 45 treatments.

There was also a significant interaction between initial host density and host stage in their effects on host weight in the absence of parasitoids (Fig. 1B; ANOVA, $F_{12,\ 140} = 7.32$, $P < 0.001$; weights for the egg stage were not included in this analysis). A multiple comparison test (approximated critical point 3.72) indicated no difference between the IHD 10 and IHD 45 treatments at any host stage, nor between the IHD 115 and IHD

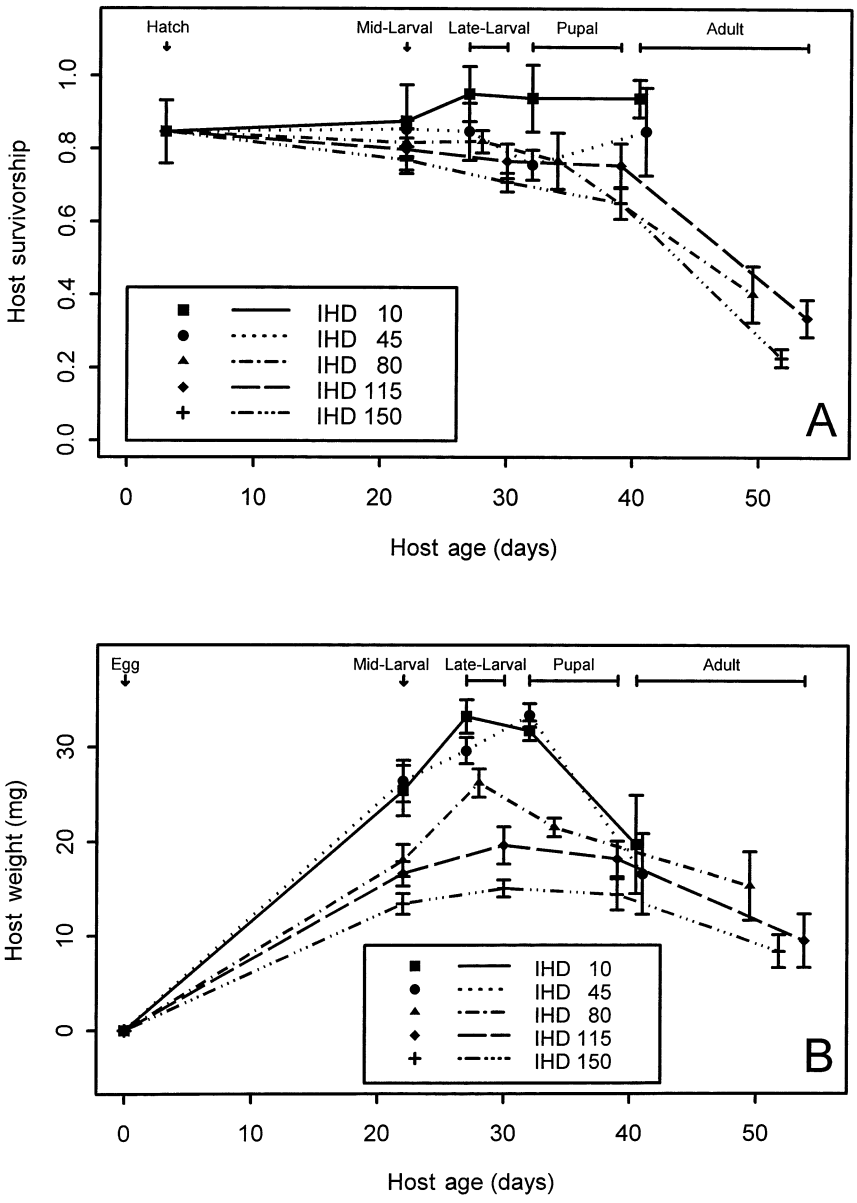

Fig. 1. Effects of initial host density and host age (parasitoids absent) on: A – host survivorship (the data point for Hatch, Host Age 3 days, applies to all five IHDs); B – host weight (the Host Age 0 datum is the mean egg weight, 26.1 ± 0.5 μg).

150 treatments, except at the late-larval stage. This test further indicated that the IHD 80 treatment is intermediate between these two extremes, grouping with the IHD 115 treatment at the mid-larval stage, with the IHD 45 treatment at the late-larval stage, with the IHD 115 treatment at the pupal stage, and with the IHD 45 treatment at the adult stage.

In the absence of parasitoids, increasing initial host density beyond a threshold for resource competition (IHD 45) resulted in decreasing host weight and reduced survivorship. The influence of competition on growth in *E. kuehniella* started early in the life cycle, as there was already a significant effect by the mid-larval stage, but did not translate into severe mortality until the pupal stage, when undernourished individuals died prior to emerging as adults.

Effect of initial host density and parasitoid density on host life-history parameters

Median host maturation time (time from start of experiment to adult emergence) was strongly non-linearly influenced by initial host density, and by parasitoid density; there was, however, no interaction effect (Fig. 2A; ANOVA: initial host density, $F_{4,\ 86} = 103.2$, $P < 0.001$; parasitoid density, $F_{2,\ 86} = 7.1$, $P = 0.001$; interaction, $F_{6,\ 86} = 2.1$, $P = 0.062$; interaction and error df

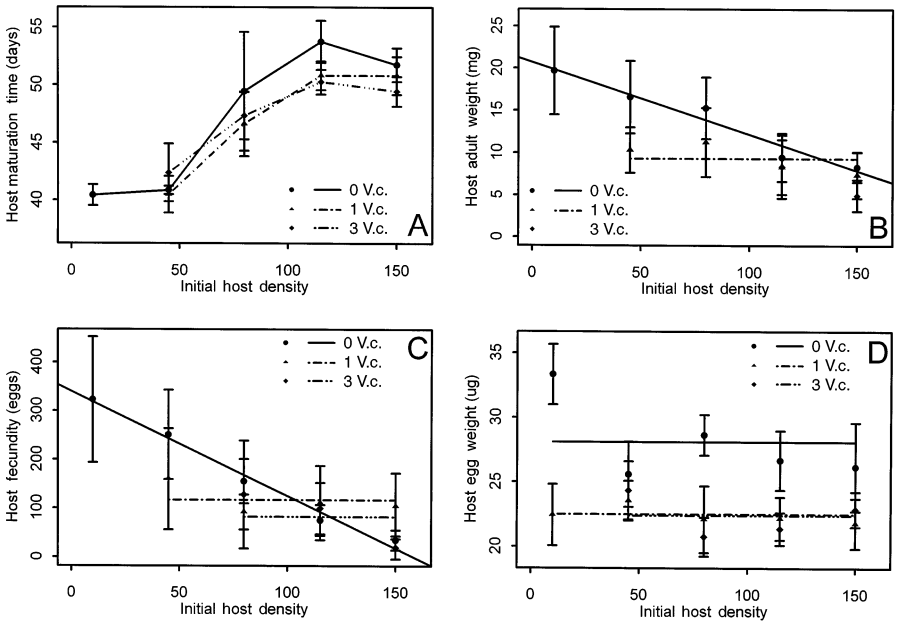

Fig. 2. Effects of initial host density and parasitoid density on host: A – median maturation time; B – adult weight (1-parasitoid treatment line is the data mean; no line was fitted to the 3-parasitoid data due to insufficient degrees of freedom for regression analysis); C – fecundity (≡ lifetime per capita eggs laid; 1- and 3-parasitoid treatment lines are the data means); D – egg weight (all lines are data means).

were reduced due to inadequate host emergence from the 10×3 treatment, and reduced sample size for the 10×1 treatment [$n = 3$], the 45×3 treatment [$n = 5$] and the 80×3 treatment [$n = 6$]). At densities beyond a threshold of IHD 45, an increase in initial host density resulted in increased host maturation time, an effect that plateaued at the highest initial host density. A multiple comparison test does not support a downturn in host maturation time with initial host density increasing from 115 to 150 for any parasitoid density (approximated critical point 3.58). In spite of a significant effect of parasitoid density indicated by the ANOVA, and the suggestion from Fig. 2A that the presence of parasitoids may depress the development time of unattacked hosts with increasing initial host density, a multiple comparison test revealed no significant differences between the 0-, 1- and 3-parasitoid density treatments (approximated critical point 3.46). This is not particularly surprising, because parasitism took place late in host larval development, after the majority of competition for resources had occurred.

There were significant effects of initial host density and parasitoid density on host adult weight; the interaction was not significant (Fig. 2B; ANOVA: initial host density, $F_{4,\ 77} = 27.8$, $P < 0.001$; parasitoid density, $F_{2,\ 77} = 7.82$, $P < 0.001$; interaction, $F_{4,\ 77} = 1.96$, $P = 0.109$; interaction and error df were reduced due to inadequate host emergence in the 10×1, 10×3, 45×3 and 80×3 treatments). Increasing host density resulted in decreased adult weight in the absence of parasitoids (linear regression, $F_{1,\ 3} = 62.0$, $P = 0.004$, error df = 35). The presence of one parasitoid served to reduce adult weight for hosts surviving parasitism evenly across host densities (linear regression, $F_{1,\ 2} = 5.41$, $P = 0.15$, error df = 28). Low host emergence from the 3-parasitoid containers precluded linear analysis of the influence of initial host density, but multiple comparison revealed no significant differences between the 1- and 3-parasitoid treatments at IHDs 115 and 150 (approximated critical point 3.34). This suggests that only the smallest hosts escaped parasitism to produce adults in each of the initial host density treatments.

Host fecundity was significantly affected by the interaction between initial host density and parasitoid density, in a manner similar to that seen for host adult weight (Fig. 2C; ANOVA, $F_{5,\ 77} = 3.94$, $P = 0.003$; fecundity data were square-root transformed; interaction and error df were reduced due to inadequate host emergence from the 10×1, 10×3, and 45×3 treatments, and reduced sample size for the 80×3 treatment [$n = 4$] and the 80×1, 115×1 and 115×3 treatments [$n = 7$]). Egg production was highly correlated with host adult weight for all treatments for which data were available ($n = 11$, $t_9 = 6.69$, $P < 0.001$, $r = 0.91$). In the absence of parasitoids, increasing initial host density led to a linear decrease in egg production by surviving host females (linear regression, $F_{1,\ 3} = 310.2$, $P < 0.001$, error df = 35). In the presence of parasitoids there was no significant influence of initial host density on the fecundity of surviving females (linear regression, 1-parasitoid treatment: $F_{1,\ 2} = 2.37$, $P = 0.26$, error df = 26; 3-parasitoid treatment: $F_{1,\ 1} = 5.5$, $P = 0.26$, error df = 16), and no difference in the level of fecundity reduction between the 1- and 3-parasitoid treatments except at IHD 150 (multiple comparison, approximated critical point 3.43). It is important to recognize that any con-

clusions about the effects of parasitoid density on host adult weight and/or fecundity in this study are somewhat tentative, because the combination of high parasitoid density and low host density resulted in almost complete suppression of the host and an absence of data for these treatment combinations.

E. kuehniella egg weight was significantly influenced by the interaction between initial host density and parasitoid density (Fig. 2D; ANOVA, $F_{7,\ 98} = 7.52$, $P < 0.001$; error df were reduced due to inadequate host emergence from the 10×1 treatment). Linear regression, on the other hand, did not reveal a significant linear relationship between host egg weight and initial host density for any parasitoid density (linear regression, 0-parasitoid treatment: $F_{1,\ 3} = 2.39$, $P = 0.220$, deviations $F_{3,\ 35} = 9.38$, $P < 0.001$; 1-parasitoid treatment, $F_{1,\ 3} = 1.91$, $P = 0.261$, error df = 35; 3-parasitoid treatment, $F_{1,\ 2} = 0.20$, $P = 0.698$, deviations $F_{2,\ 28} = 10.42$, $P < 0.001$). Host egg weight at IHD 10 in the absence of parasitoids was significantly greater than at higher initial host densities (multiple comparison, approximated critical point 3.61). Host egg weight was also significantly depressed to an equal extent in the 1- and 3-parasitoid treatments (multiple comparison, approximated critical point 3.45). This suggests an effect similar to that observed for host adult weight and fecundity. In the presence of parasitoids, smaller larvae avoid parasitism and survive to adulthood, resulting in smaller adults with smaller eggs.

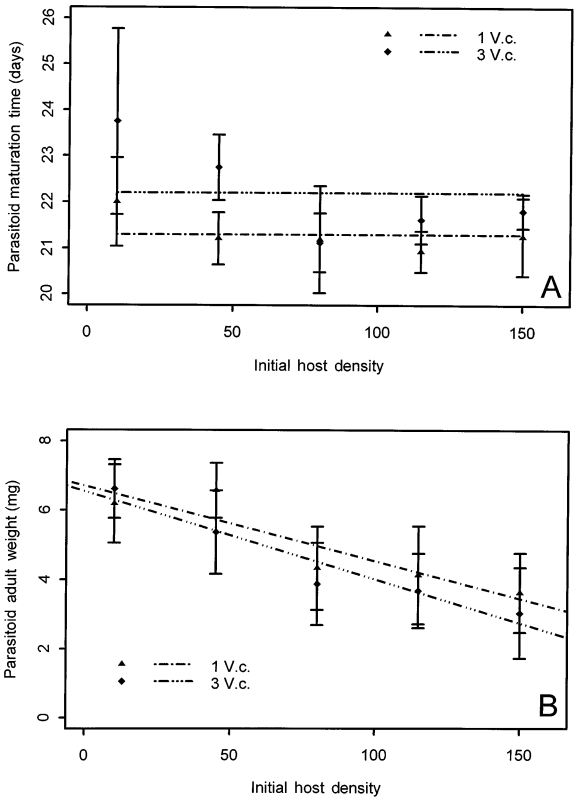

Fig. 3. Effects of initial host density and parasitoid density on parasitoid: A – median maturation time (from initial parasitoid introduction into containers; both lines are the data means); B – adult weight.

Effect of initial host density and parasitoid density on parasitoid life-history parameters

ANOVA indicated significant effects of initial host and parasitoid density on median parasitoid maturation time (defined as the time from parasitoid introduction into the containers to adult emergence), and no significant interaction (Fig. 3A; ANOVA: initial host density, $F_{4,\ 67} = 8.70$, $P < 0.001$; parasitoid density, $F_{1,\ 67} = 16.32$, $P < 0.001$; interaction, $F_{4,\ 67} = 2.34$, $P = 0.064$; error df were reduced due to reduced sample size [$n = 7$] in the 10×1, 45×1 and 115×1 treatments). Regression analyses, however, indicate that parasitoid maturation time is not linearly related to initial host density at either parasitoid density (linear regression, 1-parasitoid treatment: $F_{1,\ 3} = 2.69$, $P = 0.20$, error df = 32; 3-parasitoid treatment: $F_{1,\ 3} = 4.08$, $P = 0.14$, error df = 35). Multiple comparison tests indicate that only the 10×3 treatment is different from any of the others (between parasitoid densities approximated critical point 3.21; between initial host densities approximated critical point 3.61). Thus, no significant effect of initial host or parasitoid density on parasitoid maturation time appears to exist.

ANOVA indicated a strong effect of initial host density on parasitoid adult weight, no effect of parasitoid density, and no significant interaction (Fig. 3B; ANOVA: initial host density, $F_{4,\ 70} = 22.4$, $P < 0.001$; parasitoid density, $F_{1,\ 70} = 3.13$, $P = 0.081$; interaction, $F_{4,\ 70} = 1.04$, $P = 0.391$). For both parasitoid densities a significant linear decrease in parasitoid adult weight occurred with increasing initial host density (linear regression, 1-parasitoid treatment: $F_{1,\ 3} = 13.78$, $P = 0.034$, error df = 35; 3-parasitoid treatment: $F_{1,\ 3} = 38.8$, $P = 0.008$, error df = 35). A multiple comparison test between parasitoid densities at each initial host density indicated no differences (approximated critical point 3.20). In addition, parasitoid adult weight was highly correlated with host adult weight in the absence of parasitoids ($n = 10$, $t_8 = 4.892$, $P = 0.001$, $r = 0.87$). Thus, no effect of parasitoid density on parasitoid adult weight was detected, but interestingly, in contrast to host adult weight in the presence of parasitoids (Fig. 2B), parasitoid adult weight decreased with initial host density, suggesting that parasitized hosts may have reduced competitive ability relative to unparasitized hosts.

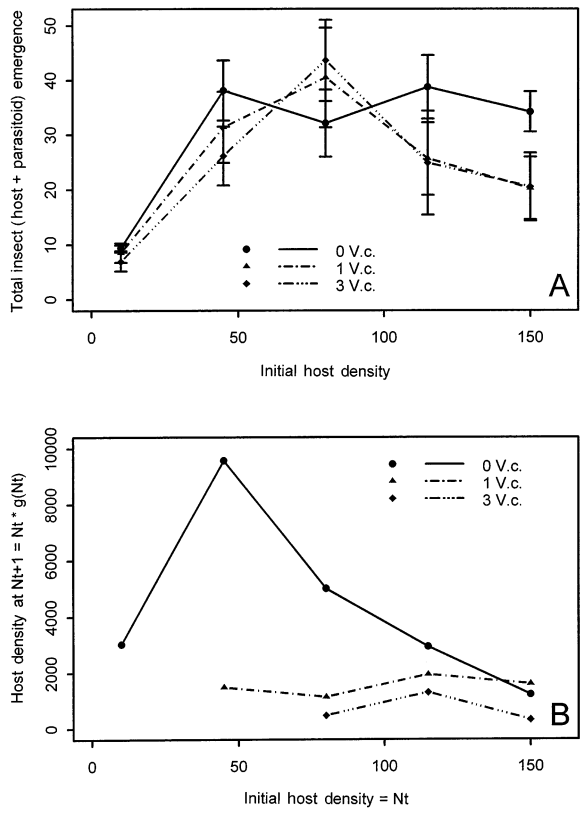

Fig. 4. A: Effects of initial host density and parasitoid density on total adult emergence (host + parasitoid). B: Host population growth rate (initial host density × survivorship × fecundity) as a function of initial host density.

Form of host intraspecific competition

The influence of resource competition on the survivorship of both parasitized and unparasitized hosts was determined from total insect emergence (host + parasitoid). A significant interaction effect of initial host density and parasitoid density on total insect emergence was evident (Fig. 4A; ANOVA, $F_{8,\ 105} = 6.96$, $P < 0.001$; emergence data were square-root transformed). In the absence of parasitoids, a saturating effect of initial host density on host emergence was detected, such that adult emergence increased from the IHD 10 treatment to the IHD 45 treatment and remained constant as initial host density increased thereafter (multiple comparison, approximated critical point 3.67). In the presence of 1 or 3 parasitoids, total emergence increased from IHD 10 to IHD 80, representing an increase in the density threshold for survivorship, suggesting that parasitized hosts have a lower demand on resources than unparasitized hosts. In addition, total emergence in the presence of parasitoids declined rather than saturated above IHD 80 (Fig. 4A).

Although the presence of parasitoids had no effect on total insect emergence in the IHD 10 treatment, there were significant effects at other initial host densities (Table 1). At lower initial host densities parasitism resulted in a replacement of hosts by parasitoids, but at the same time there was a trend toward reduced total emergence as parasitoid density increased (significant at IHD 45; Table 1). Over-stinging by parasitoids at low initial host densities may have resulted in some additional mortality of parasitized hosts. In contrast, at higher initial host densities an equivalent level of additional mortality was evident at both parasitoid densities (Table 1), suggesting that survivorship of parasitized hosts relative to healthy hosts was compromised under conditions of intense competition, either through reduced competitive ability relative to unparasitized hosts or via greater susceptibility to cannibalism.

The influence of resource competition on host population growth rate was determined by combining data for host survivorship and fecundity (Fig. 4B). In the absence of parasitoids the host's population growth rate rose sharply as initial host density increased from 10 to 45, and thereafter dropped, clearly illustrating a density threshold for intraspecific competition in *E. kuehniella*. In the presence of parasitoids this effect was completely removed – host recruitment into the next generation was independent of initial host density and relatively uninfluenced by parasitoid density, maintaining approximately the level of recruitment of the IHD 150 treatment in the absence of parasitoids.

Table 1. Simultaneous mean difference 99% confidence intervals† for total insect emergence vs *V. canescens* density at each initial *E. kuehniella* density.

V. canescens densities	Initial *E. Kuehniella* density				
	10	45	80	115	150
0 vs 1	−0.83, 1.16	−0.41, 1.58	−1.68, 0.31	**0.19, 2.18***	**0.39, 2.38***
0 vs 3	−0.54, 1.45	**0.08, 2.07***	−1.93, 0.06	**0.31, 2.30***	**0.37, 2.36***
1 vs 3	−0.68, 1.29	−0.51, 1.48	−1.25, 0.74	−0.88, 1.11	−1.01, 0.98

† Approximated critical point 3.49. Absolute differences significantly different from zero are indicated in **bold** and by an asterisk (*).

Table 2. Linear and three non-linear models of the host per capita population growth rate function $g(N)$, the fitted values of each model's parameters to the original dataset, the adjusted residual sums-of-squares describing each model's relative fit to the original dataset, and the proportion of bootstraped datasets for which each model provides the best relative fit to each bootstrapped dataset (linear model provided for comparison purposes).

Model	Form of $g(N)$	m	b	$r = ln(\lambda)$	λ	$K^{\dagger}$	γ	SSQadj	Proportion of Best Fits
Linear	$m\ N+b$	-2.22	299.9	–	–	–	–	1285.5	0.000
Ricker	$exp[r(1-N/K)]$	–	–	5.95	383.0‡	317.7	–	725.6	0.004
Generalized Ricker	$exp\{r[1-(N/K)^{\gamma}]\}$	–	–	5.73	308.3‡	145.8	2.28	124.9	0.085
Generalized Beverton-Holt	$\lambda/[1+(N/K)^{\gamma}]$	–	–	5.72‡	303.7	56.42	3.69	12.2	0.911

† In the Ricker and generalized Ricker models, $K > 0$ is the carrying capacity, the value of N for which $g(N) = 1$. In the generalized Beverton-Holt model, $K > 0$ is the value of N for which $g(N) = \lambda/2$. See Getz (1996) for details.
‡ $exp(r)$ or $ln(\lambda)$ of the corresponding estimated parameter value (λ or r), as appropriate; provided for comparison purposes.

Fitting host self-limitation models to experimental data

Non-linear regression analyses of four different models of $g(N)$, the density-dependent per capita population growth rate of the host in the absence of parasitoids, were conducted against data for host survivorship and fecundity in the absence of parasitoids. These analyses indicate that the best fit to the data is provided by the generalized Beverton and Holt model (Table 2; Fig. 5). This result further demonstrates the presence of a density threshold in *E. kuehniella* intraspecific competition, such that below this threshold there is little or no effect of density on survivorship or fecundity. The fact that the Ricker function occasionally provides the best fit to bootstrapped data sets is due to this model being the best fit to extreme "outlier" data sets generated by the bootstrap process. It is worth noting that the values obtained by regression for r, the host's intrinsic rate of increase, are in reasonable agreement among the three non-linear models (Table 2), although r for the Ricker model is somewhat larger than for the others. Similarly, the values obtained by regression for γ, the threshold or abruptness parameter, are in reasonable agreement for both the generalized Ricker and generalized Beverton and Holt models.

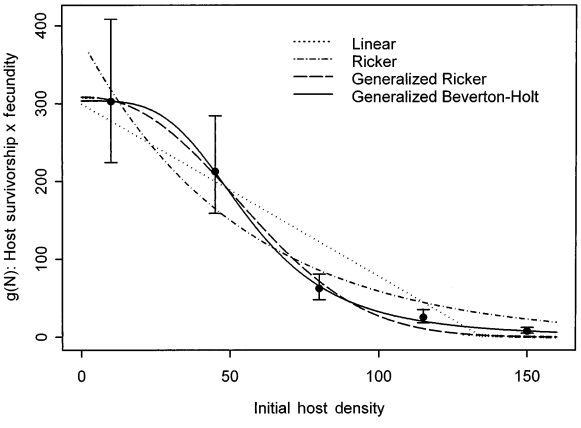

Fig. 5. Comparison of linear and three non-linear regression models of host per capita population growth rate (survivorship × fecundity) as a function of initial host density. Error bars represent 95% confidence intervals for the regression data set (i.e. $\alpha = 0.05$, so that $z = 1.96$ in eq. (1)).

Discussion

Effect of competition and parasitism on host performance

In the absence of parasitoids, an increase in initial host density led to a reduction in host growth and development rate, resulting in increased mortality in the pupal stage and reduced adult weight and fecundity (which were highly correlated). Thus, a notable delay is evident between the timing of competition for limiting resources (which occurs during the larval stages), and its consequences for the dynamics of *E. kuehniella* populations (i.e. mortality during the pupal stage and reduced fecundity). This combination of factors has determining consequences for the host's per capita population growth rate (Fig. 5) – a threshold effect exists such that below an initial host density of approximately 25 eggs/5 g semolina little to no effect of density on per capita population growth occurs, while above the threshold a significant reduction in population growth is evident, approaching a lower asymptote beyond 100 eggs/5 g semolina.

As found in the current study, Bernstein et al. (2002) have also recently demonstrated that a threshold effect in the per capita population growth rate of *E. kuehniella* occurs due to the combined influences of competition on growth (reproduction) and survivorship. Similar consequences of density (e.g. crowding, resource limitation) on individual life-history parameters for *E. kuehniella* (e.g. growth and maturation rates, survivorship and fecundity) have been found by other workers (Norris 1933, Ullyett 1945, Ullyett and van der Merwe 1947, Daumal and Boinel 1994, Anderson and Löfqvist 1996). For example, Norris (1933) found similar levels of egg mortality and that fecundity was reduced rather than mortality increased under conditions of moderate larval competition. Ullyett (1945)

also found that *E. kuehniella* fecundity decreased sharply once host density reached a value approximately equivalent to IHD 45 in this study, and Ullyett and van der Merwe (1947) found a precipitous increase in larval and pupal mortality once food availability fell below 0.1 g diet per larva. Similarly, a threshold model has been found to provide the best fit for population growth rates of a wide variety of insect species under both laboratory and field conditions, including *E. kuehniella* (Bellows 1981).

In the presence of parasitoids, although host maturation time was not significantly altered, host adult weight and fecundity were consistently reduced, independent of initial host density, to levels equivalent to those of hosts reared at higher initial host densities in the absence of parasitoids (Fig. 2B and 2C). Egg size in the presence of parasitoids was also consistently reduced (Fig. 2D). These effects could conceivably occur for at least two separate reasons, operating either singly or in conjunction. First, the suggestion (not supported statistically) that the presence of parasitoids can depress the development time of unattacked hosts (Fig. 2A) may indicate that only the smaller host larvae that matured earlier than larger individuals escaped parasitism. Small individuals may escape due to the parasitoids' inability to successfully parasitize small hosts, and/or because parasitoids preferentially attack larger hosts (Sait et al. 1997). Alternatively, some host species have the potential to survive parasitism by *V. canescens* via encapsulation (Salt 1975), but parasitism survivors can incur fitness costs resulting, for example, from reduced competitive ability (Harvey et al. 1996). Various *Drosophila* studies indicate that host-fitness consequences are associated with the encapsulation of parasitoid eggs, namely reduced competitive ability, growth and fecundity (Fellowes and Godfray 2000). Although Salt (1964, 1965) has shown that *E. kuehniella* never encapsulates the eggs or young larvae of *V. canescens*, more recent evidence suggests that parasitized 5th instar *E. kuehniella* may encapsulate up to 15% of *V. canescens* eggs or larvae (J. Harvey, pers. comm.). Thus either or both mechanisms may have been operating in the current *E. kuehniella–V. canescens* system.

With the addition of parasitoids, the density threshold for survivorship of hosts plus parasitoids increased from IHD 45 to IHD 80 (Fig. 4A) indicating that parasitized host larvae have lower demands for resources than unattacked larvae (Harvey 1996). At initial host densities above this threshold, survivorship to adulthood decreased as a consequence of increasingly intense intraspecific competition. This may reflect that parasitized larvae are poor competitors in relation to unattacked hosts, and as a consequence are often unable to support complete parasitoid development to adulthood. In addition, the present study revealed that cannibalism was common in *E. kuehniella*, and was most prevalent in the late larval stage, when resources were exhausted (pers. obs.). Chapman et al. (1999) found that cannibalism in *Spodoptera frugiperda* (Lepidoptera: Noctuidae) conferred a fitness benefit only under conditions of low food availability. Furthermore, parasitized larvae may also be more susceptible to cannibalism than unattacked larvae, increasing the rate of cannibalism for the experiment with parasitoids present as resource competition becomes more intense. Reed et al. (1996) found that *Plodia interpunctella* (Lepidoptera: Pyralidae) parasitized by *V. canescens* were more likely to be cannibalized in individual encounters between parasitized and unattacked larvae.

Effect of competition on parasitoid life-history parameters

Little to no effect of either host or parasitoid density on parasitoid maturation time was evident, and no effect of parasitoid density on parasitoid adult weight was observed. Parasitoid adult weight, however, was strongly influenced by initial host density, and was strongly correlated with host adult weight in the absence of parasitoids. Similar effects of initial host density on parasitoid size, egg load and adult survival time were found by Bernstein et al. (2002) for the same system. Harvey et al. (1995) also found that 5th instar larvae of *P. interpunctella* reared at high density produced smaller *V. canescens* adults, and Harvey and Thompson (1995) showed that the number of ovulated eggs in *V. canescens* was positively correlated with adult wasp size, with consequent implications for the fecundity of parasitoids in this study.

Essentially no effect of parasitoid competition on parasitoid life history parameters was evident in this system. Trudeau and Gordon (1989) showed that *V. canescens* reared on *Cadra cautella* (Lepidoptera: Phycitidae) has a fecundity of over 250, and that the daily rate of host attack was on the order of 16–22. Furthermore, Harvey et al. (2001) showed that *V. canescens* reared on *P. interpunctella* has a fecundity of over 400, and convincingly demonstrate that fecundity measurements based on oviduct dissections (the method used by Trudeau and Gordon 1989) underestimate actual progeny production in *V. canescens*. Thus, for purely numerical reasons, it is highly likely that superparasitism took place in all parasitoid treatments, though this was not measured directly. On the other hand, Harvey et al. (1993) found that superparasitism by *V. canescens* resulted in increased parasitoid development time, and also resulted in smaller parasitoids emerging from later-instar *P. interpunctella* larvae. No evidence of either of these effects was detected in this study, indicating either that superparasitism did not occur, or that it does not have the same effect in this system, with *E. kuehniella* being a larger host.

Host intraspecific competition: scramble vs contest

E. kuehniella life-history exhibits little mortality early in the life cycle, and trades off growth (weight) versus mortality in response to crowding. Thus, a separation occurs in the host's life-cycle between the timing of the action of density dependence and the effects of density dependence. Determining the form of competition (scramble or contest; Nicholson 1954, Hassell 1975, Łomnicki 1988, Toquenaga and Fujii 1991, Parker 2000) is often based on examining plots of the number of individuals surviving to reproduction, or to the next generation, as a function of the number of individuals in the current generation (Łomnicki 1988, Toquenaga and Fujii 1991, Reeve et al. 1998); or, more generally, by examining the population density before and after the mortality effects of density-dependent competition (i.e. *k*-factor analysis; Hassell 1975). A significant problem with this approach, however, is that scramble competition may be mistaken for contest, if the nature of the resource and the competitive mechanisms in question are not fully understood, or if the population densities over which competition is examined are sufficiently small to avoid demonstrating scramble competition.

Consider, for example, survivorship alone (Fig. 4A). In the absence of parasitoids, the number of host adults emerging as a function of initial host density remains constant for IHDs above 45, the threshold for resource limitation. This would seem to indicate that host adult emergence becomes independent of initial host density, and that competition is of contest type (Łomnicki 1988, Parker 2000). At densities above those used in this study, Bernstein et al. (2002) found adult emergence to decline, suggesting scramble competition. Furthermore, plotting N_{t+1} vs N_t for this system (Fig. 4B) leads to the conclusion that competition in the absence of parasitoids is of the scramble form, as this plot takes into account not only survivorship but also loss of fecundity due to reduced growth. In this study, *E. kuehniella* completely exhausted its resource (semolina) at higher initial host densities (pers. obs.). The importance of whether the resource was "shareable" or "unshareable" (Nicholson 1954, Parker 2000) cannot be overemphasized. "Gains to competitors in contests are all or nothing (individuals are either 'winners' or 'losers'), whereas in scrambles, all individuals achieve some gains, if sometimes less than enough to survive and/or reproduce" (Parker 2000).

The presence of parasitoids causes host recruitment into the next generation to be independent of initial host density (Fig. 4B), and the form of host intraspecific competition to shift from scramble to contest (Łomnicki 1988). In this system parasitism functions as a selective force (Tuda and Iwasa 1998), changing the competitive regime for hosts from one in which scramble competition for food is most successful, to one in which contest competition for enemy-free space becomes more successful. This effect does not result from parasitoids reducing host densities below the threshold for scramble competition (Holt and Lawton 1993), since the effects of parasitism do not occur until late in the window of competition for food resources. Scramble competition still occurs prior to the action of parasitoids in the host's life-cycle, but, in the presence of parasitoids, contest competition occurs later in the host's life-cycle, and dominates the overall competitive regime (see Reeve et al. 1998 for a system in which scramble dominates contest competition).

Host intraspecific competition: modeling the data

Although the effect of initial host density on fecundity provides much of the density-dependent reduction in per capita population growth rate, the non-linearity of the mortality response to initial host density contributes to the reverse sigmoid shape of the survivorship × fecundity data set used for non-linear regression (Fig. 5). Models of density dependence incorporating sigmoid thresholds provide the best explanation of the data, as was also found by Bernstein et al. (2002) using a different model goodness-of-fit criterion for the same host-parasitoid system. Of the four models considered here, the generalized Beverton and Holt provided the best fit to this data set. The Ricker model, with fewer free parameters, provided a poor fit to the data. Clearly, representing density dependence in this population with a model for which no threshold is present (e.g. the Ricker model) would be inappropriate. A threshold effect has been shown for a variety of insect populations (Bellows 1981), and has been reasonably argued on both theoretical (Getz 1996) and empirical grounds (Hassell 1975).

The inadequacy of the Ricker model as a description of density dependence in this population is noteworthy for two reasons. Due to its simplicity it has been widely used in host-parasitoid models (Barlow and Wratten 1996, Mills and Getz 1996, Mills 2001), yet it exhibits the phenomenologically unrealistic property that the effect (i.e. the strength) of density dependence is greatest at *low* densities (Getz 1996; but see Jarosik and Dixon 1999). Thus we recommend use of the generalized Beverton and Holt model to capture the essence of host self-limitation in host-parasitoid models.

Ordering of events in models of host-parasitoid population dynamics

In the *E. kuehniella–V. canescens* system examined here, the key mechanism of host self-limitation (reduced growth) occurs in the host's life-cycle prior to the action of the parasitoid, though the effects (reduced

fecundity) occur after parasitism. May et al. (1981) emphasize the need for, and illustrate the dynamical consequences of, stage and/or age structure and the ordering of events in even simple host-parasitoid models. While some models of host-parasitoid dynamics incorporate these features (Hochberg and Holt 1995, 1999, Tuda and Iwasa 1998, Hochberg and Ives 1999, Kean and Barlow 2000, Mills 2001), the results of the present study re-emphasize the need to account for any specific biological features that may influence either the generality or specificity of predictions made on the basis of these models, particularly in the context of biological control (after Hassell 1980). Certain advantages exist in keeping these models simple. But when they are too simple, they loose any relevance to real systems (natural, biological control or laboratory; Hochberg and Holt 1999), and become mere exercises in analysis.

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