

LETTER

Variable prey development time suppresses predator–prey cycles and enhances stability

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Abstract

Although theoretical models have demonstrated that predator–prey population dynamics can depend critically on age (stage) structure and the duration and variability in development times of different life stages, experimental support for this theory is non-existent. We conducted an experiment with a host–parasitoid system to test the prediction that increased variability in the development time of the vulnerable host stage can promote interaction stability. Host–parasitoid microcosms were subjected to two treatments: Normal and High variance in the duration of the vulnerable host stage. In control and Normal-variance microcosms, hosts and parasitoids exhibited distinct population cycles. In contrast, insect abundances were 18–24% less variable in High- than Normal-variance microcosms. More significantly, periodicity in host–parasitoid population dynamics disappeared in the High-variance microcosms. Simulation models confirmed that stability in High-variance microcosms was sufficient to prevent extinction. We conclude that developmental variability is critical to predator–prey population dynamics and could be exploited in pest-management programs.

Keywords

Age-structured populations, developmental variability, host–parasitoid microcosms, intraspecific trait variability, population cycles, predator–prey stability.

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INTRODUCTION

Cyclical population fluctuations or outbreak dynamics of predators and prey have been a central theme in the field of ecology for more than a century (Elton 1924; Berryman 2002). Moreover, from an applied perspective, much effort has been expended to understand, predict and suppress outbreaks in cyclical pest species (e.g. Esper *et al.* 2007; Bjornstad *et al.* 2010). Dating back to the work of Lotka and Volterra (Lotka 1925; Volterra 1926), theoretical models have served as a guiding force in understanding predator–prey population dynamics (Pimm 1992; May 2001; Murdoch *et al.* 2003). In recent years, models incorporating age (or stage) structure have demonstrated that the duration of various life stages, as well as the generation time of the prey relative to the predator, can greatly influence predator–prey dynamics (Murdoch *et al.* 1987; Godfray & Hassell 1989; Reeve *et al.* 1994; Wearing *et al.* 2004; Murdoch *et al.* 2005). Depending on the duration of these various stages, the predator–prey interaction can exhibit stability, generation cycles, multi-generation consumer–resource cycles, or chaotic fluctuations. Intraspecific variability in traits associated with predator–prey interactions, e.g. prey attack rates, host vulnerability to natural enemies, and stage-specific development times, are also theoretically important to predator–prey population dynamics (Doebeli 1997; Xu *et al.* 2010; Bolnick *et al.* 2011; Gibert & Brassil 2014). Unfortunately, experimental tests of the effects

of stage structure and/or trait variability are exceedingly rare (Murdoch *et al.* 2005; Bolnick *et al.* 2011).

A simplifying feature of most age- or stage-structured models is a fixed development time for the various prey and predator life stages. However, theoretical models incorporating distributed development times often predict more stable predator–prey population dynamics (Smith & Mead 1974; Hastings 1983, 1984; Briggs *et al.* 1993; Wearing *et al.* 2004; Eurich *et al.* 2005; Nakamichi *et al.* 2008; Xu *et al.* 2010). For example both generation and longer period cycles are less likely when development times have a distribution than when they are fixed. One mechanism underlying this increased stability is heterogeneity in the risk of parasitism generated by hosts with variable development being exposed to parasitism for different times (Chesson & Murdoch 1986; Hassell *et al.* 1991). Another is the tendency for models with distributed development to be more stable than their fixed counterparts (May 1974; Hastings 1983, 1984; Eurich *et al.* 2005). Despite the importance of distributed development time as a potential stabilising mechanism for predator–prey population dynamics, there has never been an attempt to test this theory with an experiment.

We conducted a laboratory experiment with a model predator–prey system to assess whether increased variability in the development time of the vulnerable prey stage, promoted stable predator–prey temporal population dynamics. The model system, the cowpea weevil (*Callosobruchus maculatus*)

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and its parasitoid (*Anisopteromalus calandrae*), dates back to the classic work of Utida (Utida 1941, 1957) in the early 1940s. Under controlled laboratory conditions, the dynamics of these two species are characterised by long-term limit cycles (Utida 1941, 1957; Fujii 1983; Bonsall *et al.* 2002). Consequently, this is an ideal system for examining stabilising mechanisms in population ecology.

METHODS

The study system

The biology and life history of the weevil and parasitoid are described in detail in Beck & Blumer (2007). Female weevils lay eggs on the surface of beans. The larva hatches, burrows into the bean and passes through four larval instars. In the latter larval stages, the weevil burrows close to the seed coat, leaving a round 1–2 mm window through which the adult will eventually emerge. The appearance of the emergence window indicates the start of the weevil's period of vulnerability to *A. calandrae*. We divide the weevil's life cycle into four stages: H_1 is the invulnerable juvenile stage that extends from the egg to the appearance of the window; H_2 is the vulnerable host stage or the period between window appearance and the late pupal stage; H_3 is the late pupal stage to adult emergence; and H_4 is the adult stage. Adult weevils do not feed or require water. The approximate development times for these life stages are provided in Table S1 in the Supporting Information.

The pteromalid *A. calandrae* is a solitary ectoparasitoid of bruchids such as *C. maculatus* (Ji *et al.* 2004; Tuda & Shimada 2005). Following egg hatch, the parasitoid larva quickly kills the host. Parasitism of weevils post-window formation is high for the first 5 days and drops off precipitously at day six (Fig. S1). Superparasitism occurs, but only one parasitoid can develop on a single host. *A. calandrae* will host feed which can extend adult longevity (Ghani & Sweet-

man 1955). The parasitoid is divided into two life stages, a juvenile (P_1) and adult (P_2) stage (Table S1).

Experimental microcosms

The procedures used for studying the population dynamics of *C. maculatus* and *A. calandrae* are similar to those described previously (Utida 1943, 1954; Tuda & Shimada 2005). Our experimental microcosm consisted of a single 150 × 25 mm petri dish (Fig. S2). Moth beans were chosen as the food source because of their small size (4–5 mm) which has the advantage that no more than one adult weevil can emerge per bean (see SI Appendix). Beans were contained in four 60 × 15 mm petri dishes with 5 g moth beans (182 ± 0.71 beans; mean \pm SE; $n = 20$) per dish. Initiation of a colony involved adding one dish of beans plus 10 male and 10 female adult weevils to the microcosm. At 12-day intervals, another dish of beans was added until all four dishes were present in the microcosm. Every 12 days thereafter, the oldest dish of beans (48 days since initial exposure) was removed and replaced with a fresh dish of beans. After 48 day, 10 adult *A. calandrae* (50 : 50 sex ratio) were added. The experiment was conducted in growth chambers at 28 ± 2 °C, 50 ± 5 % RH and 12 : 12 day: night light cycle.

For 27 replicate microcosms, adult host and parasitoid abundances were assessed every 12 days (Tuda & Shimada 2005). Insects were anaesthetised with CO₂ (Mbata *et al.* 1996) and number of live and dead individuals per species were counted. Live insects were returned to the microcosm and cadavers were discarded. More details regarding this census procedure are provided in the SI Appendix.

Manipulation of the vulnerable host stage

Methodologically, the manipulation of development time by using hosts of different quality (Tuda 1996) or by altering

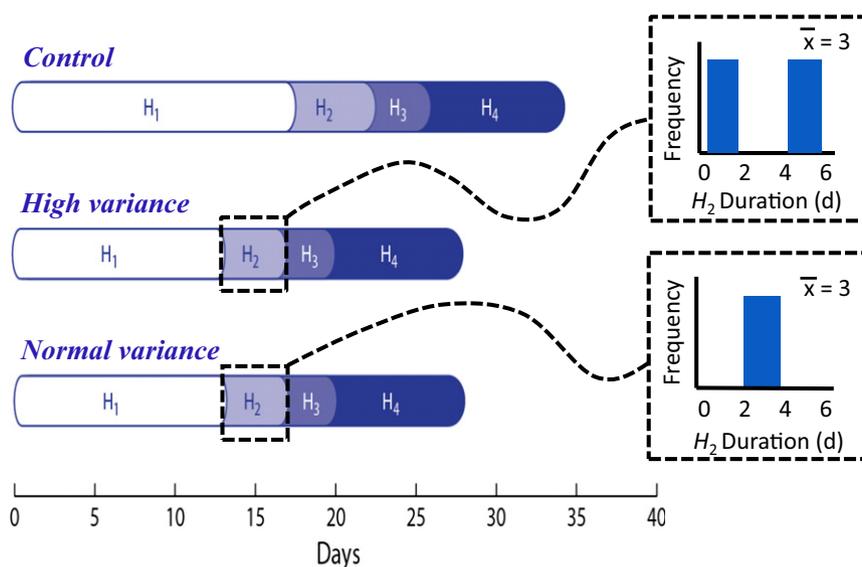


Figure 1 Diagram of the experimental treatment and its effect on the average development times of the weevil life stages. For the adult weevil life stage (H_4), the mean duration is based on the females. Inset histograms show the distribution of H_2 weevil ages used in the High- and Normal-variance treatments.

temperature regimes (Tuda & Shimada 2005) is problematic because all life stages are affected, as well as potentially other demographic parameters such as reproduction and survivorship. We opted for an artificial means of changing development time that targets a specific life stage, the vulnerable stage of the host (H_2 stage; Fig. 1), without altering any other aspects of the host's demography. This was accomplished by manually replacing beans with weevils just prior to entering the vulnerable stage with weevils that have been in the vulnerable host stage for different lengths of time. Specifically, variability in the duration of the vulnerable host stage was increased by replacing weevils entering the vulnerable stage with equal numbers of weevils at the beginning and near the end of the vulnerable stage (see below).

Critical to the experimental manipulation of the variability in the duration of the vulnerable host stage was the tracking of the age of all juvenile weevils in each microcosm. Every 3 day, weevils and parasitoids were anaesthetised with CO_2 , sieved to separate them from the beans, and aspirated into a small container. All beans were inspected and those with new eggs were separated from the rest of the beans by a small divider within the 60-mm diameter petri dish and identified with a single dot. Three days later, those beans identified with a single dot were upgraded to a second dot. The dots identified the time since appearance of the first weevil egg(s), in 3-day intervals, and continued until the weevils reached the four-dot stage (9–12 day old; median 10.5 day). Here, we assumed that one of the eggs laid in that first three-day period, when the bean was identified with a single dot, was the one to survive to the vulnerable host stage. This assumption was confirmed by the dissection of beans with eggs laid on different days – the earliest eggs laid were invariably the weevils that survived (see SI Appendix). Also, egg to vulnerable stage (H_2) survival is extremely high (0.96 ± 0.02 ; mean \pm SE; $n = 81$).

The experimental manipulation involved replacing beans infested with weevils near the end of the invulnerable juvenile stage (i.e. the 4 dot stage) with beans containing weevils that have been in the vulnerable host stage for different lengths of time (Fig. 1, see also SI Appendix). Replicate microcosms were subjected to 4 treatments: (1) high variance in the duration of the vulnerable host stage, (2) normal variance in the duration of the vulnerable host stage, (3) an experimental control, and (4) an unmanipulated control. Our *a priori* prediction was that predator–prey cycles should be evident in the unmanipulated control, experimental control and Normal-variance treatments, but should be reduced or absent in the High-variance treatment.

In this experiment, the earliest host stage (H_1) was fixed at 9–12 days (median of 10.5 day), less than the minimum duration of this stage (Table S1). The purpose of truncating the duration of the H_1 stage was to ensure that all beans removed from the microcosm had unparasitised weevils (i.e. pre-vulnerable stage hosts). Based on our model simulations, reducing the duration and fixing the length of the H_1 stage effected no qualitative change in host–parasitoid population dynamics (see SI Appendix).

For the **High-variance treatment** ($n = 6$), we established a bimodal distribution of the duration of the H_2 stage with an average duration of 3 day (Fig. 1). Every 3 day, all beans

with H_1 weevils 9–12 day old were removed from the microcosm. One half of those infested beans were replaced with beans containing weevils that had been in the vulnerable stage for 0–1 day (median 0.5 day) and the other one half were replaced with beans with weevils that had been in the vulnerable stage for 4–5 day (median 4.5 day). Because the duration of the vulnerable stage is 5.3 ± 0.1 day (Table S1), weevils from the first half took ~ 5 day and those from the second half took ~ 1 day to mature to the H_3 stage. Overall, the average duration of the vulnerable host stage was approximately 3 day. This procedure was repeated every 3-day.

In the **Normal-variance treatment** ($n = 6$), all 9–12 day old H_1 weevils were replaced with weevils that were in the vulnerable stage for 2–3 d (median of 2.5 day; Fig. 1). Consequently in those microcosms, it was expected that the replacement weevils were in the vulnerable stage for 3 day before maturing to the invulnerable H_3 stage – equivalent to the mean duration of the vulnerable stage for the High-variance treatment. Weevils in these replacement beans were expected to exhibit levels of variability in the duration of the vulnerable stage that were comparable to the variability found for unmanipulated weevils. This conclusion is based on the fact that no weevils complete their development in ≤ 3 day, and therefore the mean duration is shifted but the variation in development times remains unchanged.

Although, reducing the duration of the H_1 stage to 10.5 day was not expected to affect the population dynamics of this host–parasitoid system (see above), we included an experimental check (i.e. the **Experimental control**; $n = 5$) to test specifically whether reducing the duration of the H_1 stage by 6 days (from 16.8 to 10.5 day) affected population dynamics. In this treatment, all 9–12 day old H_1 weevils were replaced with 0–1 day old vulnerable H_2 weevils (Fig. S3). Consequently, the duration of the vulnerable host stage was equivalent to natural conditions. Finally, the **Unmanipulated control** ($n = 10$) consisted of a microcosm of weevils and parasitoids in which no manipulations were performed (Fig. 1). For both controls, insects were anaesthetised and sham manipulations were performed at 3-days intervals to mimic the handling experienced by the insects in microcosms from the experimental treatments.

Based on the distribution of development times for the vulnerable host stage (Table S1), the standard deviation in development time for this stage is 0.543 ± 0.069 (mean \pm SE; $n = 8$ samples of 100 weevils). In comparison, the High-variance treatment is estimated to have a standard deviation in development time that is 3.2 times greater than the control or Normal-variance treatment (1.714 ± 0.062).

The source of the replacement weevils for the treatments was the main weevil colony. Adult weevils from the colony were added to a large dish of moth beans (50 g), allowed to mate and oviposit for 24 h and then removed. Starting 14 day later (minimum duration of the H_1 stage), these beans were inspected daily for the appearance of windows (onset of the H_2 vulnerable stage). Those H_2 beans were then placed in a separate container and held in the environmental chamber until they reached the appropriate age for the above treatments. Using this method, we obtained weevils entering the vulnerable host stage every day for the duration of the experiment.

We note here that our bean replacement procedure required no adjustments to account for egg-larval mortality, or intraspecific competition among larvae developing within the same bean. As stated previously, egg-to-window survivorship is 96%. Also, only one larva matures to the vulnerable host stage. Therefore, each removed bean contained a single well-developed larva and was replaced with a bean containing a single vulnerable-stage weevil.

The experimental treatments were initiated June 13, 2011 and ran until July 1, 2013. Given that the generation time for the weevil is ≈ 28 day, this represented 27 generations of the host. Because parasitoids went extinct in four of the five Experimental controls within the first eight months of the experiment, we excluded this treatment from subsequent analyses.

Time-series analyses

Analyses of the time series are described in detail in the SI Appendix, so only a brief accounting is provided here. For each microcosm, we computed the mean and standard deviation (SD) of $\log_{10}(N + 1)$ transformed host and parasitoid abundances among census dates (where N is the number of adults). Differences in the mean abundance or SD in abundance among treatments (High-variance, Normal-variance, Unmanipulated control) were assessed using separate Welch's ANOVAS (Welch 1951).

We used wavelet analyses to explore the cyclical behaviour of host and parasitoid population dynamics in each microcosm. Wavelet analysis, like a Fourier analysis, is used to decompose a signal (or time series) into its different oscillatory components with different frequencies (periods) (Torrence & Compo 1998; Cazelles *et al.* 2008). However, unlike a Fourier analysis, wavelet analysis can be applied to time series where the frequency and amplitude of oscillations vary through time. Given that many time series exhibit non-stationarity (Cazelles *et al.* 2008), the ability to evaluate the spectral characteristics of a time series as a function of time, is a desirable attribute of this method. The methods for computing the wavelet transform are provided in the SI Appendix.

Because the wavelet analyses revealed no clear evidence of non-stationarity in the time series for each treatment (Fig. S6), we averaged the wavelet power values for each period across the entire time series. This yielded a global wavelet spectrum that identifies the relative oscillatory strength for each possible period. For comparisons among treatments, we computed the mean and 95% CIs of the global wavelet spectrum for all replicates within each treatment.

The host–parasitoid model

To better understand the dynamic consequences of our variance manipulations, we constructed stage-structured models for the weevil and parasitoid that allowed for gamma-distributed development times for the juvenile stages, using overall levels of variability similar to the experimental treatments (see Box 1 for details). The models were parameterised with data independent from our microcosm experiment (see Table S2). Owing to the complexity of the model, particularly regarding the pulse additions of food, stability was assessed in terms of

persistence and variability in population numbers when the system is stationary. Host–parasitoid dynamics in our experimental microcosms were compared to predictions from our models with comparable levels of variability in the duration of the vulnerable host stage. We also used the models to understand why extinctions occurred in the experimental controls.

RESULTS

Microcosms subjected to the different variance in development time treatments exhibited very different population dynamics (Fig. 2a–c; see also Fig. S5 for the time series for all replicate microcosms). Over the course of the two-year experiment, mean number of adult hosts in the High-variance microcosms was 32% higher than in the Normal-variance microcosms and 50% higher than in the unmanipulated control microcosms (Fig. 3a). Adult parasitoid numbers per microcosm showed the opposite pattern. Numbers in the High-variance microcosms were 45% and 60% lower than in the Normal-variance and Unmanipulated control microcosms respectively (Fig. 3a). As predicted by theory, increased variability in development time of the vulnerable host stage (H_2) promoted reduced variability in the abundances of the host and parasitoid. The standard deviation in population abundance was 24% lower for the host and 18% lower for the parasitoid in the High-variance microcosms as compared to the Normal-variance or control microcosms (Fig. 3b).

In addition to affecting temporal variability in host–parasitoid population dynamics, manipulation of the variability in development times also caused significant qualitative changes in the cyclicity of the system. Hosts and parasitoids in the unmanipulated control microcosms exhibited strong evidence of cyclical dynamics (Fig. 2a, d). For the host population in the representative unmanipulated control microcosm (UC-2), the global wavelet spectrum (comparable to a Fourier power spectrum that identifies the relative oscillatory strength for each possible cycle period; see Methods) revealed a very powerful signal for period-two oscillations (Fig. 2d). Averaged among the replicate Unmanipulated control microcosms, the global wavelet spectrum for the hosts consistently exhibited strong period-two oscillations (Fig. 4a; Fig. S7). In our experiments, a two-census period oscillation translates into 24 day, approximately the generation time of the host under these controlled environmental conditions. In contrast, parasitoid populations exhibited greater variability in cyclical behaviour. Although in replicate UC-2 the parasitoids exhibited very little oscillatory behaviour (Fig. 2d), the mean global wavelet spectrum for the parasitoids in all replicate control microcosms revealed relatively low-power oscillations with a period of 2–6 (Fig. 4D; see Fig. S6 and S7 for wavelet power spectrums and global wavelet spectrums, respectively, for all replicate microcosms). Figure 4 shows the mean and 95% CIs for the global wavelet spectrums for the host and parasitoid in each treatment.

Population dynamics in the Normal- and High-variance microcosms were qualitatively very different from the Unmanipulated control microcosms. In the Normal-variance microcosms, the global wavelet spectrums indicated powerful

Box 1: The Host–Parasitoid Stage-Structured Models

The stage-structured host–parasitoid model that we used was developed by Murdoch *et al.* (Murdoch *et al.* 1987) and Godfray and Hassell (Godfray & Hassell 1989). The host life cycle was divided into four stages (H_1, H_2, H_3, H_4) and the parasitoid life cycle was divided into two stages (P_1, P_2). Rather than fixed delays, however, the development time (or duration) of these stages (except the H_3 stage) are modelled using probability distributions. The model also recognises the laboratory microcosms are composed of four dishes each representing a subpopulation, whereas the adult stages are distributed throughout the microcosm. We formulated our model as a system of integral equations given below.

$$H_1^i = \int_0^t R_1^i(s) \text{prob}(t-s) e^{-\mu_{H_1}(t-s)} ds$$

$$H_2^i = \int_0^t F_1^i(s) \text{prob}(t-s) e^{-\mu_{H_2} + f(P_2)dx} ds$$

$$H_4^i = \int_0^t F_3^i(s) \text{prob}(t-s) e^{-\mu_{H_4}(t-s)} ds$$

$$P_1^i = \int_0^t f(P_2) H_2^i(s) \text{prob}(t-s) e^{-\mu_{P_1}(t-s)} ds$$

$$P_2^i = \int_0^t F_{P_1}^i(s) \text{prob}(t-s) e^{-\mu_{P_2}(t-s)} ds$$

$$B^i = B^0 - \int_0^t R_1^i(s) ds, R_1^i = \min\left(B^i, \text{lay} \frac{B^i}{\sum_{i=1}^4 B^i}\right), i = 1, 2, 3, 4$$

$$\text{lay} = \theta_1 H_4 / (1 + \theta_2 H_4)^b$$

here H_j^i is the number of H_1 hosts in the i th dish, with similar notation for other stages. The $\text{prob}(s)$ functions give the probability that an individual of age s remains within a particular stage. The functions $F(s)$ describe the maturation rate from preceding stages. The model also incorporates the dynamics of the beans within each dish, in particular the number of beans, B^i , that are available for weevil oviposition. The parameter B^0 denotes the number of beans that were added to the microcosms every 12 days, whereas R_j^i is the recruitment rate of beans to the H_1 stage. Given what is known about the oviposition behaviour of the weevils (see SI Appendix), we assumed a density-dependent rate lay for adult hosts laying viable eggs. We then modelled the recruitment rate R_j^i as the minimum of B^i and the number of viable eggs, allocated in proportion to the number of available beans in the i th dish. Note that the regular addition of beans to the microcosms make this an impulsive system, with a pulse period equal to 12 days. Other model features are standard in age-structured host–parasitoid models, such as the parasitoid attack rate $f(P_2)$ and density-independent mortality rates for each stage (μ_{H_1}, μ_{P_1} , etc.).

With the assumption of gamma distribution for development times of organisms, as seems appropriate for most stages in our system (see Table S1), the integral equations of the model can be converted to a system of differential equations that can be readily simulated (see SI Appendix). Hence, the model with this special assumption is an extension of previous works where models have incorporated the gamma distribution in a limited way (Wearing *et al.* 2004; Xu *et al.* 2010). Note that the integral model can also be used to describe population dynamics with other probability distributions, such as the Weibull distribution.

period-four oscillations (i.e. 48 day or two host generations) for the host and parasitoid (Fig. 2e). These results were consistent among the six replicate microcosms (Fig. 4b, e; Fig S7), although the signal strength was twice as great for the host than the parasitoid. In contrast, for the High-variance microcosms, the low variability in population densities (Fig. 3b) underlies the absence of periodicity in the time series (see also Fig S5). For the representative time series in Fig. 2c (*HV-1*), global wavelet spectrums for the host and parasitoid (Fig. 2f) showed no evidence of any strong periodic oscillations. Among the six replicate High-variance microcosms, the results were the same (Fig. 4c, f; see also Fig. S7).

Simulations using our stage-structured models for the host and parasitoid provided additional support that increasing variability in the duration of the vulnerable host stage, H_2 , promotes increased system stability; that is reduced amplitude fluctuations and long-term persistence of the host–parasitoid interaction (see SI Appendix, “*The Effect of Variability in the Development Time of the H_2 Stage*” and “*The Bimodal Distribution in the High-Variance Treatment*”). We first estimated the parameters in the models using the data from the Unmanipulated controls and other sources, and found that the model readily generated period 2 oscillations similar to the microcosms (Fig. 2g). We then altered the parameters to

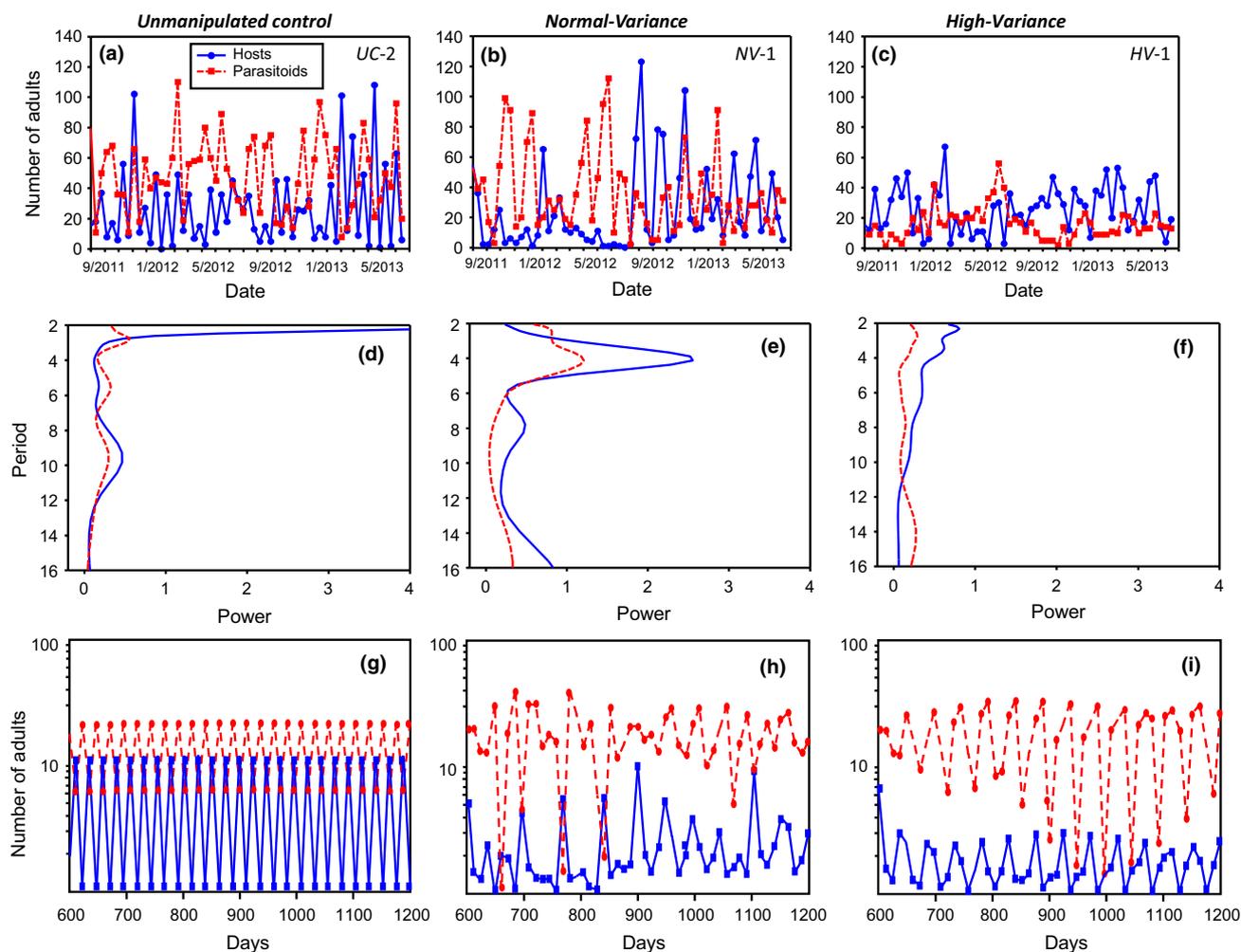


Figure 2 Host and parasitoid time series, global wavelet power spectrums and model simulations for unmanipulated control, Normal-variance and High-variance microcosms. Summary graphs for each treatment include the raw time series (a,b,c) and global wavelet spectrums (d,e,f) for the host and parasitoid for one representative time series. Summary graphs for all replicate microcosms and treatments are provided in Supplement, Fig. S5, S6, S7. Model simulations were tailored for each of the three treatment (g,h,i). In the Normal- and High-variance simulations, k (the clumping parameter from the negative-binomial model) was reduced from 0.91 to 0.61 to achieve long-term persistence in the Normal-variance treatment.

mimic the experimental treatments, and found that the Normal-variance microcosms were prone to extinction, whereas the High-variance microcosms were persistent, illustrating the stabilising effect of variability. Figure 2h–i shows the model output for these two treatments where stability was increased by adding more parasitoid aggregation, sufficient for the Normal-variance treatment to persist. The standard deviation in host population sizes was 60% higher for the Normal-variance as compared to the High-variance treatments (0.24 vs. 0.15). Standard deviation in parasitoid abundances between treatments was similar (0.37 vs. 0.33 for the Normal- and High-variance treatments respectively). Although both treatments showed some longer period oscillations, they were stronger in the Normal-variance treatment. The simulations suggest that variability in the vulnerable host stage enhances stability because it allows some hosts to escape parasitism when parasitoid densities are high, allowing additional host cohorts to arise and thereby smoothing the oscillations. Our simulation models also confirmed that whether high variabil-

ity in development time of the vulnerable host stage is brought about by reducing the shape parameter m_{H_2} of the gamma distribution or by making the distribution in bimodal as in our experimental manipulations, host–parasitoid dynamics are qualitatively the same (compare Fig S16 and S17). The models also correctly predicted extinction in the experimental control replicates, because of increased synchrony in the host and parasitoid life cycles.

DISCUSSION

Experimental manipulation of trait variability within a population and quantifying its impact on population- or community-level dynamics has been an elusive goal in the field of ecology (Bolnick *et al.* 2011). This study provided the first experimental support for the theory that variability in development time can be strongly stabilising (Briggs *et al.* 1993; Wearing *et al.* 2004; Xu *et al.* 2010). Confirming the reports of others on this classic study system (Utida 1941, 1957; Fujii

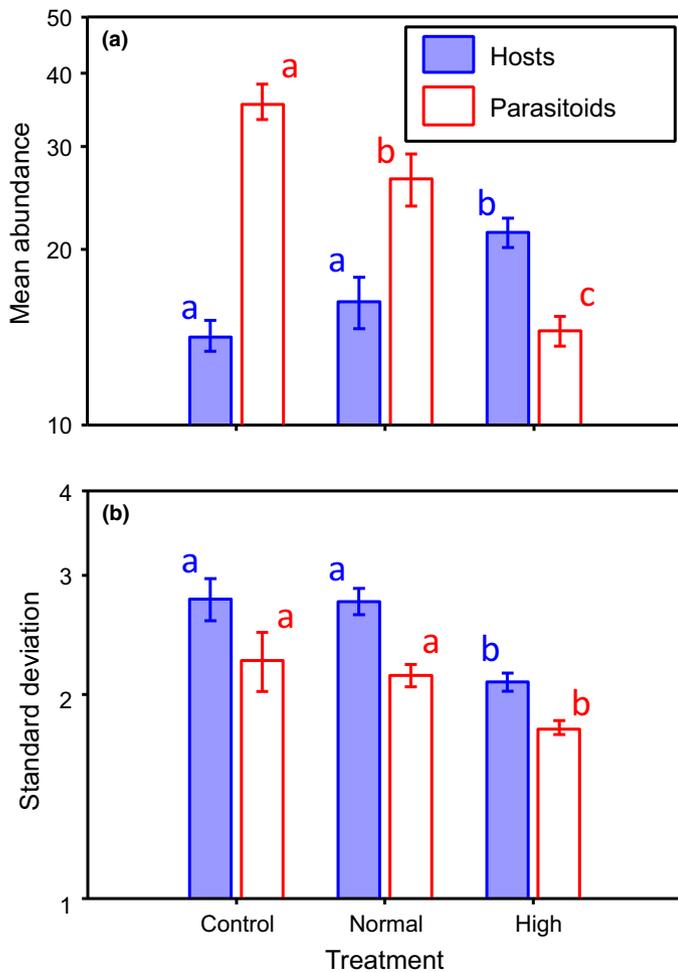


Figure 3 Mean (a) and standard deviation (b) in adult abundance for the unmanipulated control, Normal-variance, and High-variance treatments. Reported are the averages \pm SE among microcosms. Y-axes are on a log base-10 scale. Based on separate Welch's ANOVAs, mean host and parasitoid abundance ($F_{2,10.6} = 11.38$, $P = 0.0023$, $F_{2,10.8} = 45.89$, $P < 0.0001$ respectively) and mean standard deviations ($F_{2,11.5} = 14.47$, $P = 0.0007$, $F_{2,11.0} = 8.47$, $P < 0.0059$ respectively) differed significantly among treatments. Different letters denote significant differences among means based on a Tukey's HSD test ($P < 0.05$).

1983; Bonsall *et al.* 2002), the unmanipulated control microcosms exhibited strong evidence of cyclical dynamics with a period of approximately one generation (i.e. generation cycles; Begon *et al.* 1995). Increasing the variability in the development time of the vulnerable host stage not only reduced fluctuations in host and parasitoid populations, but also eliminated the periodicity in the time series.

Increased abundances of hosts and reduced abundances of parasitoids in the High-variance relative to the Normal-variance microcosms were expected because the window of vulnerability for one half of the hosts in the former treatment was quite brief. Those older vulnerable-stage hosts had a much higher probability of escaping parasitism than the younger vulnerable-stage hosts. Another consequence of the bimodal distribution of vulnerable host development times was that the majority of attacks by *A. calandreae* were likely concentrated in hosts with the greater window of vulnerability (i.e. the younger

vulnerable-stage hosts). The result was increased pseudointerference among parasitoids – an aggregated distribution of attacks and wastage of eggs on previously attacked hosts that results in a negative relationship between parasitoid density and parasitism. Pseudointerference can be strongly stabilising for a host–parasitoid interaction (Hassell *et al.* 1991; Hassell 2000; Murdoch *et al.* 2003) and *A. calandreae* is known to super-parasitise hosts (Lebreton *et al.* 2010). Variable development times also create a partial refuge from parasitism for individual hosts who pass quickly through the vulnerable stage. A partial refuge is known to contribute to system stability (Murdoch *et al.* 1987, 2003) and our study suggests that distributed development times are a potentially important mechanism for generating partial refuges from parasitism.

Development times for insect juvenile stages often resemble a gamma or Weibull distribution (Xu *et al.* 2010), not a bimodal one as was established for the vulnerable host stage in the High-variance treatment. The bimodal distribution was adopted for the simple reason that it was experimentally much more tractable to increase variability by establishing two discrete age classes than many age classes within the vulnerable stage. However, using a bimodal distribution of vulnerable host development times was unlikely to yield qualitatively different dynamic results than if we had used gamma-distributed development times. Based on our model simulations, gamma- and bimodal-distributed development times, with variability comparable to that in our High-variance microcosms, yielded similarly stable population dynamics (see SI Appendix). In addition, we do not feel that the variability in development times we created were extreme. High levels of developmental variability were found in a literature survey by Xu *et al.* (2010) of mostly laboratory studies, and even higher levels would be expected under field conditions.

For several decades, theoretical models have highlighted the dynamical complexity that can arise in predator–prey populations owing to changes in the age structure of participants. For example using a stage-structured model for the California red scale (*Aonidiella aurantii*) and parasitoid *Aphytis melinus*, Murdoch *et al.* (2005) concluded that a long invulnerable adult host stage and rapid parasitoid development greatly enhanced stability of the interaction, and these were likely the key mechanisms involved in suppressing an experimental outbreak of the scale. However, experimental tests that explicitly assess the impact that a change in the mean or variability in the development time of a particular life stage have proven to be quite challenging. Several studies have attempted to indirectly manipulate development times by changing rearing temperature or diet (Tuda & Shimada 1995; Tuda 1996), but under these circumstances, development time was unavoidably confounded with other changes in the prey and predator population. We believe that our microcosm study with *C. maculatus* and *A. calandreae* represents the first unambiguous demonstration that age (stage) structure is critical to the dynamics of host–parasitoid and prey–predator systems. Rather than representing noise in studies of development rates, as it is usually treated, variability itself has important effects on the dynamics of these systems (Xu *et al.* 2010).

Our study gives credence to the viewpoint of theorists that population models should incorporate realistic aspects of the

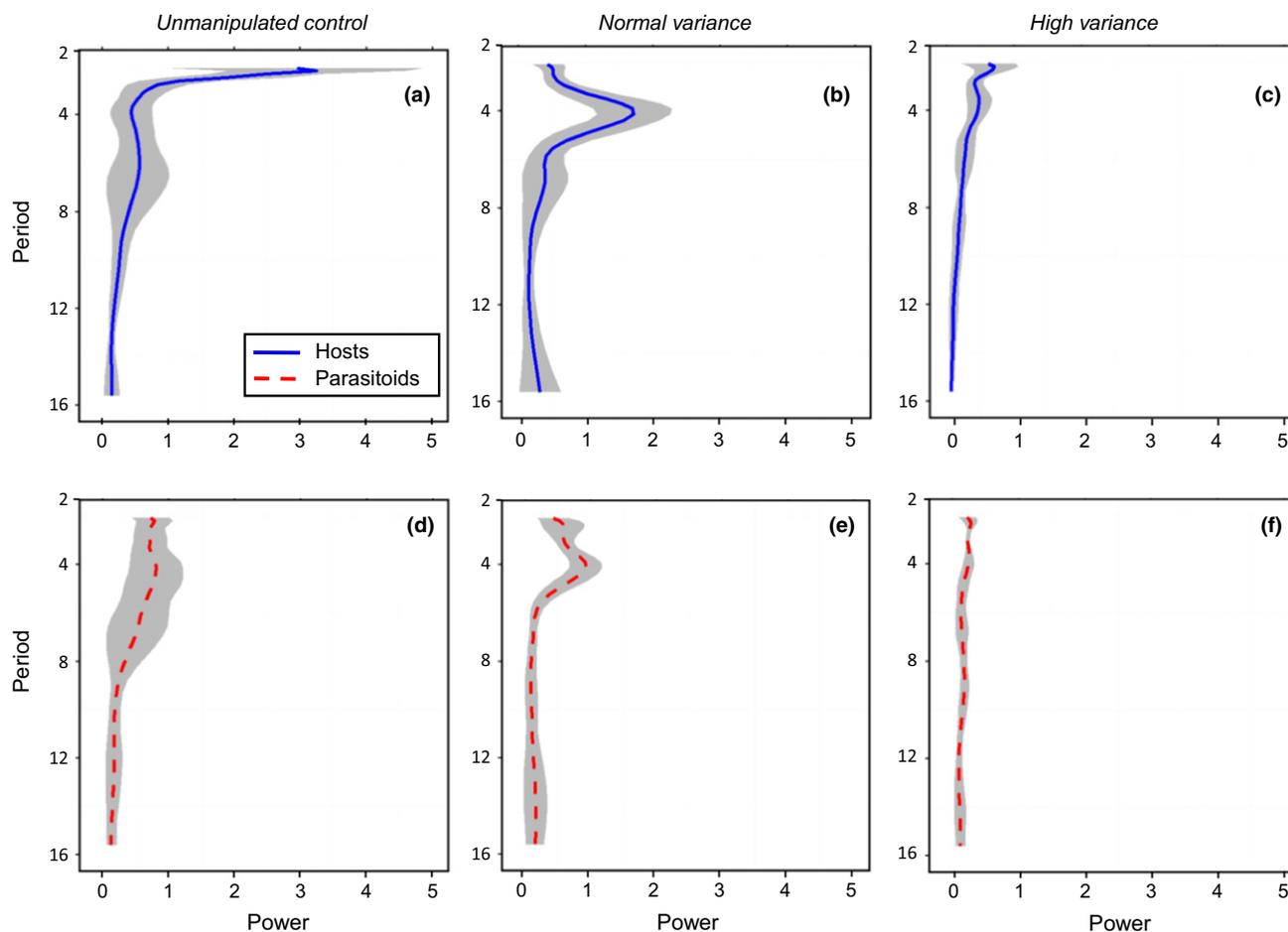


Figure 4 Mean global wavelet spectrums for replicate microcosms from the Unmanipulated control (Host, a; Parasitoid, d), Normal-variance (b; e), and High-variance (c; f) treatments. For each treatment, the global wavelet spectrums (see Fig. 2d–i, Fig. S7) were averaged among replicates. Grey bands represent 95% confidence intervals.

age (stage) structure and stage-specific development times of each species. In the burgeoning field of study of the ecological consequences of trait variability, there is now both a strong theoretical foundation for the role of development time variability on predator–prey population dynamics and empirical support for this theory. Also, because we artificially manipulated trait variability in the prey, our study represents a very rare test of the direct (phenotypic) effects of trait variability on population dynamics (Bolnick *et al.* 2011). Because heritable variation in traits may indirectly (through evolution) affect population dynamics (Bolnick *et al.* 2011), a valuable next step would be to experimentally, or through modelling, allow the evolution of development rates to occur in this system. Presumably, costs to variable development rates or covariance with other fitness-related traits may result in very different predator–prey dynamics. For example reduced risk of parasitism for individuals with fast vulnerable-stage development times may come at the cost of reduced survivorship or longevity (e.g. Lee *et al.* 2013). How developmental variability is maintained in natural populations in the face of these trade-offs is an open question.

This study has important implications for the biological control of pests. It suggests that pest-enemy stability and equi-

librium densities are strongly influenced by variability in stage-specific development times. While these quantities are known for a few pest species, variability is seldom studied for its own sake and is almost never quantified for the natural enemies (Xu *et al.* 2010). Greater emphasis needs to be placed on collecting these kinds of data, particularly if there is interest in the development of models to forecast pest populations (de Valpine *et al.* 2014). From a practical standpoint, variability in pest population development times could explain why some undergo recurring outbreaks (e.g. Esper *et al.* 2007; Haynes *et al.* 2012) whereas others appear to have stable dynamics. It could be that more stable systems have greater innate developmental variability or other factors that generate such variation. For example more stable systems could have a greater diversity of food sources (to generate heterogeneity in nutritional condition) or more structural complexity (to generate heterogeneity in microclimates) that can cause development times within the pest population to be more variable (Tuda & Shimada 1995; Tuda 1996). In fact, a common approach to pest management already involves increasing habitat complexity, through the planting of polycultures. Perhaps generating more variability in development times is one unexplored benefit of this management tactic. Even though

increased variability in the duration of vulnerable host stages may lead to higher mean pest densities, inhibition of pest outbreaks could keep pests below economic injury levels where management becomes less critical.

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AUTHORSHIP

J.T.C, J.D.R., D.X and M.X. designed the research; J.T.C and H.N.S conducted all experiments; J.T.C and J.D.R conducted the time-series analysis. J.D.R, D.X and MX developed the host–parasitoid models that included distributed development times for different life stages. J.T.C. wrote the manuscript and it was edited by J.D.R, D.X., M.X. and H.N.S.

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