Dynamics of predator-prey cycles and the effects of dispersal and the Moran effect

Here we describe in more detail the dynamics of predator-prey limit cycles in our model, and the manner in which dispersal and the Moran effect affect the synchrony of these cycles. Similar dynamics occur when the model exhibits damped oscillations to equilibrium rather than a stable limit cycle in the absence of stochasticity.

We first consider deterministic dynamics in the absence of any stochasticity. In the absence of dispersal, the angular velocity of the system (i.e. the rate of change in predator and prey densities along the cycle) varies at different points on the cycle (Fig. S1). Angular velocity is slowest at the nadir of the cycle, when prey and predator densities are both low, implying low population growth rates. The system accelerates as it exits the nadir and prey density increases, in part because increasing prey density increases total prey growth rate, and in part because increased prey density reduces the per-prey risk of predation and so increases prey per-capita growth rate. The system decelerates slightly as prey growth slows due to intraspecific competition and increasing predator density, then accelerates again as increasing predator density drives prey to low levels. This acceleration occurs in part because decreasing prey densities increase the per-capita predation rate experienced by prey, so that prey decline more rapidly as their density decreases, and in part because of increasing predator densities which increase total predator growth rate. When prey density drops sufficiently low, predators decline, and the system decelerates as both prey and predator densities drop to low levels.

In the accelerating regions of the cycle, two patches initially at nearby points will separate (e.g., Fig S1, time 1). In these regions of the cycle the system’s intrinsic dynamics promote desynchronization; density differences between initially-similar patches increase. In the decelerating regions of the cycle, two patches initially at nearby points will be brought closer together by the intrinsic deterministic dynamics (e.g., Fig. S1, time 2). Thus, in the absence of dispersal, the phase difference between the two out-of-sync patches fluctuates over time in a characteristic pattern, increasing and decreasing twice during a single cycle.

Dispersal reduces and eventually eliminates these fluctuations in phase difference by creating diffusion of individuals from patches of high density to patches of low density over the entire cycle. Dispersal accelerates the ‘trailing’ patch (patch A in Fig. S1), while decelerating the ‘leading’ patch (patch B in Fig. S1), eventually forcing the two patches into phase.

In contrast, the Moran effect is ineffective at the nadir of the cycle. Since environmental variation affects population growth in a multiplicative fashion, periods of low prey and predator densities which occur with predator-prey cycles imply periods when environmental noise has very little effect in absolute terms, allowing cycles to easily decohere due to additive spatially-independent stochasticity. Predator-prey oscillations weaken the Moran effect in our model.
because prey densities do not make extended excursions to low density in the absence of predators.

We now illustrate the behavior of the full stochastic model and the operation of the dynamical mechanisms described above. The mechanisms described above imply that changes in parameter values that decrease minimum prey and predator densities and slow system dynamics at the nadir of the cycle should weaken the Moran effect but not alter the synchronizing effect of dispersal. Fig. S1 illustrates this effect by varying the predator attack rate $a$. The predator attack rate is low in Fig. S1a, leading to oscillations in which prey and predators spend only brief periods of time at low density and never crash to extremely low density. The Moran effect maintains synchrony relatively effectively, and cycles only drift out of phase occasionally and relatively briefly. Increasing the predator’s attack rate generates cycles with larger amplitude in which both prey and predator spend long periods at very low density; such cycles quickly decohere in the absence of dispersal (Fig. S2b). Inspection of the time series indicates that cycle phase drifts primarily during the cycle nadir; this reflects the operation of additive, spatially-independent noise which the Moran effect cannot counter when densities are low. Adding a small amount of dispersal ($d = 0.01$; Fig. S2c) ensures that cycles remain coherent, despite spending long periods with both prey and predator at low density.
Directly testing the mechanisms by which dispersal affects cycle synchrony

We conducted a second independent experiment to test for the predicted acceleration and deceleration along the predator-prey cycle, and associated fluctuations in the phase difference between two out-of-sync patches. We also tested the predicted ability of dispersal to reduce or eliminate these phase fluctuations and bring initially out-of-sync patches into synchrony (as opposed to the first experiment, which tested the ability of dispersal to maintain synchrony in initially-synchronous patches). This second experiment tests whether the model reproduces the detailed dynamical mechanisms by which dispersal generates the patterns of synchrony observed in the first experiment described in the main text.

Twelve pairs of cultures were initiated with densities of $10^{3}$ *Tetrahymena* ml$^{-1}$ and $3.33 \times 10^{3}$ *Euplotes* ml$^{-1}$. These low densities were near the detection threshold of our sampling procedure and were sufficiently low to ensure low initial population growth rates. Cultures were otherwise identical to those in the first experiment.

We used a ‘time-for-space’ substitution to achieve a small difference in initial (day 0) densities between ‘leading’ and ‘trailing’ patches. The ‘leading’ culture in each pair was initiated on day -1, 24 h before the ‘trailing’ culture. Leading and trailing patches both were initiated using medium inoculated with bacteria 24 h in advance, so that there was no difference in medium age between leading and trailing patches and no detectable difference in bacterial density at the time of protist inoculation. The 24 h time interval was chosen on the basis of pilot experiments so that on day 0 the ‘leading’ patch initiated on day -1 would be slightly ahead of the ‘trailing’ patch initiated on day 0, but with densities remaining low. Sampling on day 0 found mean densities ±SE of $36.7 \pm 3.5$ *Tetrahymena* and $1.2 \pm 0.8$ *Euplotes* in the leading patches. We used a time-for-space substitution in order to ensure that the initial densities in the leading and
trailing patches fell on the same dynamical trajectory in phase space. While we cannot determine with certainty whether this trajectory falls on the system’s asymptotic attractor, it is not necessary that it do so for our purposes. Transient oscillations starting from low density are expected to exhibit the same pattern of acceleration and deceleration as oscillations on a stable limit cycle, and our results confirmed this.

The cultures were maintained at a constant 20 °C; environmental fluctuations would have obscured detection of the dynamics of interest. Half the pairs experienced daily dispersal (10% per day) and the cultures were sampled daily, including on weekends. We used daily dispersal rather than thrice-weekly as in the first experiment in order to ensure sufficient dispersal to generate an effect during a short-term experiment. Medium replacement was unnecessary due to the short term nature of the experiment.

This second experiment matched important features of model dynamics. Panels a and b of Fig. S3 show the dynamics of the predator-prey model where each patch is initiated at slightly different densities on the attractor. In the absence of dispersal (panel a) the remain out of phase until the end of the cycle. However, in the presence of dispersal the leading patch (red lines) grows relatively slower and peaks later due to emigration and the lagging patch grows relatively

![Figure S3](image-url)

**Figure S3.** First-order differences of the predator-prey model (Box 1; panels a and b) and follow-up experiment (panels c and d) showing the impact of dispersal during the emergence of the system from the cycle nadir. In the upper panels prey dynamics ($N_{t+1} - N_t$; solid lines) are shown for one complete cycle, beginning at the nadir and separated by an initial phase shift approximately 1/5 the duration of the cycle. Leading (red) and lagging (black) prey (solid lines) and predator (dashed lines) densities are shown. In the lower panels *Tetrahymena* density in leading (red) and lagging (black) patches are shown for each experimental replicate. Paired patches are coded with a common symbol.
quicker and peaks earlier due to net immigration. Differences between patches are essentially eliminated by the time the cycle reaches the apex in prey density. The experimental results closely mimic the model behavior (Fig S3 panels c and d). In the absence of dispersal the initially-small difference in prey density between patches grows until the patches approach the prey apex, and the prey remain separated in phase up to and slightly past the prey apex. Dispersal eliminates the phase separation between patches before prey densities in the leading patch reach the cycle apex. One difference between the model and experimental dynamics occurs during the decreasing portion of the cycle. Experimental prey densities decay more slowly than their model counterparts, obscuring the phase separation between the leading and trailing patches and suggesting a slight mismatch between the dynamics of our model predator and *Euplotes*. Despite this mismatch, these results demonstrate that the cycle nadir is a sensitive region in which dispersal can quickly eliminate patch-differences and ensure phase-locked spatial synchrony.

**Alternative dispersal scenarios**

We simulated our model of predator-prey dynamics using two additional dispersal scenarios: dispersal of prey only and dispersal of predators only. Despite causing a reduction of overall synchrony in prey and predators, both dispersal scenarios demonstrated the same interaction found in the main paper between dispersal and the presence of predators (Fig. S4). Regardless of which population disperses, dispersal only increases synchrony in the presence of predators. Dispersal of either prey or predators is somewhat less synchronizing than dispersal of both because in some regions of the predator-prey cycle dispersal of one of the species cannot generate synchrony. For instance, patches A and B at time 1 in Fig S1 can only be synchronized by prey dispersal, because they have similar predator densities. If only one species disperses, the system will desynchronize during those periods when that species, but not the other, is at the approximately the same density in both patches. Synchronization of predator-prey cycles previously has been demonstrated in other models in which either the prey or the predator

**Figure S4.** A three way interaction plot of the impact of dispersal, the Moran effect, and predators, on prey (a and b) and predatory (c) synchrony in our theoretical model (Box 1). Points represent the mean $z$-transformed Pearson correlation from 10 model replicates. Here two more dispersal levels have been added to the model presented in the main text, dispersal of prey only (red lines; $d = 0.15$) and dispersal of predators only (blue lines; $d = 0.15$). Any combination of dispersal of prey and/or predators is sufficient to generate the dispersal by predators interaction observed in the main text.
disperses, but not both\textsuperscript{27,32}.

**Sensitivity of treatment effects to model parameterization**

The sensitivity of the treatment effects found in our model were determined by replicated simulation of the model using 10,000 Monte Carlo trials where each of five parameters were drawn randomly on a uniform distribution: attack rate (2.33,5); dispersal rate (0.05,0.25); process noise SD $\sigma_z$ (0.05,0.25); environment noise SD $\sigma_e$ (0.3,0.9); and the correlation of the predator’s and prey’s environment (-1,1). These parameters are each described in detail in the methods section, except for the last, which provides a means to determine how differences between the predator’s and prey’s sensitivity to environmental conditions affect the model outcome\textsuperscript{15}. We calculated main treatment effect size for each of the random parameter sets as (for example):

$$\tau(d) = \left( \sum \bar{z}_{(d+)} \right) - \left( \sum \bar{z}_{(d-)} \right)$$

where $\bar{z}_{(d+)}$ is the mean Fischer’s $z$ transformed cross correlation (synchrony) for all prey pairs with dispersal and $\bar{z}_{(d-)}$ is the mean synchrony for all prey pairs without dispersal. Other main effects were quantified in the same manner. Similarly, the two way interactions effects are (for example):

$$\tau(d,M) = \left( \sum \bar{z}_{(d+M)} \right) + \left( \sum \bar{z}_{(d-M)} \right) - \left( \sum \bar{z}_{(d-M+)} \right) - \left( \sum \bar{z}_{(d+M-)} \right)$$

In the main text (Fig. 3) the distribution of treatment effects is shown and in Fig. S5 this distribution is broken down by plotting the five varied parameters against the effect size. The scatter of points in each plot is the variation generated by random variation in the four remaining parameters and the red line represents the effect of changing a single parameter while holding the others at fixed values (see methods section). Sensitivity of treatment effects to different parameters generate trends in the scatter and/or line plots.

**Does more variability increase the effect of dispersal?**

It seems plausible to hypothesize that any process that increases the absolute density differences of prey across patches could amplify the treatment effect of dispersal, since large absolute density differences result in a larger net flow of individuals. To investigate how variability among patch densities alters the impact of dispersal we added another source of random mortality to the prey (independent across patches) by removing a fraction $f_i$ from the population at the beginning of each time step, where $f_i$ was drawn randomly from the uniform distribution (0,0.5). Despite the additional source of variability, the effect of dispersal is unaltered (Fig. S6) further demonstrating that in our experiment predators affected synchrony by generating cycles, not by increasing the ‘random’ variability of prey.

**Match of the experimental system to the model assumptions**

The experimental system matches key model assumptions. The prey *Tetrahymena* grows approximately logistically\textsuperscript{33}. The predator *Euplotes* spp. has a type II functional response\textsuperscript{34}. The
Figure S5. Sensitivity of the main treatment effects (a) and two-way interaction effects (b) are shown across a range of each parameter used in the Monte-carlo simulation. Plotting the results in this fashion demonstrates the sensitivity of each effect to each parameter, given variability in the other four parameters. Red lines indicate the sensitivity of each effect, given fixed values of the remaining four parameters used in the main paper (Box 1).
strain of *Euplotes patella* used here cannot survive on bacteria alone and declines approximately log-linearly in the absence of protist prey, consistent with a density-independent background per-capita mortality rate\(^{35,36}\). Per-capita mortality rates of protists are known to vary with temperature\(^{35}\). Protists are subject to demographic stochasticity\(^{37}\), and our sampling regime acts as a source of spatially-independent environmental stochasticity. The existence of sources of spatially-independent stochasticity also is implied by the fact that initially-synchronous pairs of patches desynchronized over time in the absence of dispersal, even when subject to identical environmental fluctuations (Fig. 2b). The experimental methods imposed diffusive dispersal at equal per-capita rates for predators and prey.

**Figure S6.** A three way interaction plot of the impact of dispersal, the Moran effect, and predators, including random prey mortality as another level in the predator treatment. Points represent the mean \(z\)-transformed Pearson correlation from 10 model replicates. Adding random mortality does not alter the effect of dispersal from the ‘predator absent’ treatment despite adding variability to prey densities.

**Figure S7.** Results of a spectral analysis quantifying the proportion of variability (spectral power) in within-patch prey dynamics at periods >10 d. In bottles without predators, prey exhibit significantly greater variability at longer periods, indicating that predators generated long-period, high-amplitude cycles. Previous studies\(^{38,39}\) and inspection of our time series suggest that *Euplotes*-generated cycles in this system have periods ≥~15 d, so the choice of a 10 d threshold is conservative. Our time series are too short to allow precise estimation of cycle period.
**Figure S8.** Incubation temperature had no significant effect on the growth rates of *Tetrahymena* in our experiment (measured as the change in density after 1 day at incubation temperature). Boxes show the median 25th/75th and 10th/90th percentiles for Tetrahymena growth at 20°C and 30°C from all treatment combinations.

**Supplementary References**


