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How robust is dispersal-induced spatial synchrony?

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Many biological populations fluctuate in synchrony over large geographic regions. This behavior may increase the chance of extinction. The combination of time-scale separation between interacting species and weak spatial linear diffusive coupling is one mechanism that can generate synchrony; however, accounting for travel time between habitat patches may destabilize this synchrony. Here, we show that ubiquitous behavioral aspects of dispersal (e.g., predator avoidance), implemented as nonlinear diffusive coupling, may also destabilize synchrony. In addition, these aspects interact with travel-time delays and amplify mechanisms that destroy synchrony. Our work suggests that dispersal-induced synchrony is more rare than typically assumed. © *2015 AIP Publishing LLC*. [http://dx.doi.org/10.1063/1.4906951]

Many biological populations show cyclic behavior, for example, the famous snowshoe hare-lynx cycles in Western Canada. The surprising observation is that these cycles are often synchronized over spatial scales much larger than the movement scale of individuals. The danger of such large-scale synchrony is that extinction risk increases when population densities are low everywhere. Ecologists studied the conditions under which local cyclic predator-prey communities can be synchronized through dispersal. Somewhat surprisingly, even small amounts of random dispersal can be sufficient to synchronize these meta-communities. We include some behavioral aspects of dispersal and investigate their effect on synchrony. In particular, we model dispersal as a density-dependent process whereby the dispersal rate of the prey species increases as the perceived predation risk increases. Following previous studies, we also include a time delay to account for travel time between habitat patches. We find that each of these mechanisms can inhibit the synchronizing effects of dispersal, and their combination has a synergistic effect. Our results indicate that dispersal-induced synchrony may be more rare than previously assumed. They also lead us to speculate that behavioral aspects of dispersal may inhibit regional synchrony and thereby decrease the risk of regional extinction.

I. INTRODUCTION

One striking feature of many ecological systems is that cyclic populations can be synchronized over very large geographic regions where local population densities increase or decline simultaneously.[24] Famous examples include the Canadian hare-lynx system and the spruce-budworm system. The two mechanisms known to potentially synchronize oscillating systems over space are (i) the Moran effect, which describes entrainment by external, spatially correlated factors (e.g., weather),[14,25] and (ii) spatial coupling of local populations by even relatively small amounts of dispersal.[24] We show here that dispersal may not be a particularly robust mechanism for synchrony. Specifically, we show that including very simple, reasonable behavioral aspects of dispersal into predator-prey metacommunities may lead the system from synchrony to asynchrony or anti-synchrony.

Several mathematical models have explored how relatively simple, density-independent dispersal in multi-patch predator-prey systems may lead to spatially synchronized cyclic population dynamics.[6,15,20] Higher rates of dispersal are more likely to induce spatial synchrony as long as local dynamics are similar, but even high dispersal rates may not synchronize population dynamics that are too dissimilar.[27] Dispersal can only be considered the driving force behind spatial synchrony if it synchronizes populations relatively fast, before naturally occurring perturbations destroy synchrony. Since dispersal between ecological communities is often relatively weak, such a fast approach to synchrony requires population cycles that result from different time scales and resemble relaxation-oscillation cycles.[13] The time scale to reach synchronization when local cycles are regular sinusoidal is the inverse of the dispersal rate and therefore very long.[19] Even in the case of relaxation oscillations, local populations may synchronize only when they are already nearly synchronous. Relatively small dispersal rates may lead to stable asynchrony when initial conditions are relatively far apart. In particular, synchrony may be destroyed by sufficiently large perturbations.[13]

Most models for spatially coupled predator-prey systems implement dispersal as a simple density-independent instantaneous process. Realistically, however, dispersal takes time and occurs often as a behavioral response, so that it is density dependent. Including even small delays, representing travel times between patches, into a model may weaken or destroy the ability of dispersal to synchronize cyclic populations in space; asynchronous or anti-synchronous cycles will result.[32] We show that density-dependent dispersal may have a similar effect, and we show how density-dependent dispersal and travel-time delay interact to destroy spatial synchrony.

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Spatial synchrony of cyclic populations may refer to the (nearly) simultaneous occurrence of peaks and troughs or in addition to (almost) equal densities throughout a cycle.[6] We focus on the phase during a cycle and the phase difference between cycles in different locations. We study the evolution of phase difference by means of phase equations, a technique only recently introduced into spatial ecology.[13,32] Two cyclic populations are *phase-locked* if they have constant phase differences. Specifically, *in-phase locking* and *anti-phase locking* refers to phase differences equal to 0 or π, whereas *out-of-phase locking* refers to other values for this difference. Ecologically, it is tempting to identify in-phase locking with synchrony and anti-phase locking with anti-synchrony.

Our model system consists of MacArthur-Rosenzweig predator-prey dynamics on two identical patches, linked by dispersal of the prey. Dispersal has two components: a density-independent rate and a behavioral aspect where the dispersal rate is proportional to the perceived predation risk of the prey. We include travel-time delays between patches so that our model and results include previous work.[32] A stability analysis of our model showed that travel-time delay and density-dependent dispersal, while individually stabilizing, may synergistically act to destabilize the coexistence state of the populations.[35] Here, we focus on the effects of the two mechanisms and their interaction on synchrony. We present the model, a brief summary of its stability requirements and the transition to phase equations in Sec. II. We present the results on density-dependent dispersal (Sec. III A) and travel-time delay (Sec. III B) separately before we discuss their interaction in Sec. III C.

II. MODELS AND METHODS

A. The ecological model

The standard ecological model for studying oscillations and synchrony is the multi-patch MacArthur-Rosenzweig model.[13,14] We model dispersal between patches as a density-dependent process that captures the behavioral response of the prey species to perceived predation risk.[35] We limit the density-dependent response of prey to predation risk rather than to their total fitness.[3] Specifically, density-dependent dispersal is proportional to the per capita (of prey) predation rate. It increases as the density of predators increases but decreases as the density of prey increases since the individual predation risk decreases. We also include travel time between patches as delays.[32] In dimensional terms, the prey (H_i) and predator (P_i) densities on patch i at time $s > 0$ satisfy the equations

$$\begin{aligned}\frac{dH_i}{ds} &= rH_i\left(1-\frac{H_i}{K}\right)-\frac{acH_iP_i}{b+H_i}-D\left(\alpha\frac{\rho P_i}{b+H_i}+(1-\alpha)\right)H_i\\&\quad+D\left(\alpha\frac{\rho P_j(s-\eta)}{b+H_j(s-\eta)}+(1-\alpha)\right)H_j(s-\eta),\\\frac{dP_i}{ds} &= \frac{aH_iP_i}{b+H_i}-mP_i,\end{aligned}$$

for $i = 1$, 2 and $j \neq i$. On each of the identical patches, the prey grows logistically with growth rate r and carrying capacity K. Predation follows a Holling type-II function with maximum ingestion rate a, yield $1/c$ and half-saturation constant b. The predator grows according to the same Holling type-II response function and has linear mortality m. Only the prey species can move between patches; its movement rate is D. Fraction α of movements is density-dependent, according to the predation risk, given by the Holling type-II function, the remaining fraction $(1-\alpha)$ is density-independent. Parameter ρ is simply a scaling parameter. Travel time between patches enters the model as delay η.

We introduce non-dimensional variables via $H_i = bh_i$, $P_i = \frac{rb}{ac}p_i$ and $t = as$. These satisfy the equations

$$\begin{aligned}\frac{dh_i}{dt} &= \frac{h_i}{\epsilon}\left(1-kh_i-\frac{p_i}{1+h_i}\right)-\delta\left(\alpha\frac{gp_i}{1+h_i}+(1-\alpha)\right)h_i\\&\quad+\delta\left(\alpha\frac{gp_j(t-\tau)}{1+h_j(t-\tau)}+(1-\alpha)\right)h_j(t-\tau),\\\frac{dp_i}{dt} &= \frac{h_ip_i}{1+h_i}-\mu p_i,\end{aligned}\tag{1}$$

with the following non-dimensional parameters: $\epsilon = a/r$, $k = b/K$, $\delta = D/a$, $g = \frac{\rho r}{ac}$ $\mu = m/a$, and $\tau = a\eta$.

A detailed stability analysis of this model was carried out by the authors elsewhere.[35] For $\alpha = 0$, the model simplifies to the one studied by Wall *et al.*[32] For $\alpha = 0$ and $\tau = 0$, it is essentially the model studied by Goldwyn and Hastings.[13] In our simulations, we will fix the scaling parameter $g = 1$.

On a single patch, the behavior of the MacArthur-Rosenzweig equations is well understood. The (0, 0) equilibrium is unstable. The prey-only state $(1/k, 0)$ is globally asymptotically stable as long as $h^* = \frac{\mu}{1-\mu} \geq 1/k$. When $h^* < 1/k$, then there is a unique positive equilibrium (h^*, p^*) with $p^* = (1-kh^*)(1+h^*)$. This state is globally asymptotically stable as long as $(1/k - 1) < 2h^*$. When $2h^* < (1/k - 1)$, there is a unique stable limit cycle that is globally asymptotically stable. We are only interested in the latter case when there is a limit cycle on each patch. We denote this limit cycle as $\gamma(t)$ with period T and frequency $\Omega = 2\pi/T$.

We cast model system (1) into vector form for $X_i = (h_i, p_i)^T$ as

$$\begin{aligned}\frac{dX_1}{dt} &= F(X_1(t))+\delta W(X_1(t), X_2(t-\tau)),\\\frac{dX_2}{dt} &= F(X_2(t))+\delta W(X_2(t), X_1(t-\tau)),\end{aligned}\tag{2}$$

with

$$\begin{aligned}F(X_i) &= \left(\frac{1}{\epsilon}\left(h_i(1-kh_i)-\frac{h_ip_i}{1+h_i}\right), \frac{h_ip_i}{1+h_i}-\mu p_i\right),\\L(X_i) &= \left(\alpha g\frac{p_ih_i}{1+h_i}+(1-\alpha)h_i, 0\right),\end{aligned}\tag{3}$$

and $W(X, Y) = -L(X) + L(Y)$. In Sec. II B, we shift our point of view to study synchrony. To that end, we assume that the system is oscillating, and we describe only the dynamics of the phase of the system along the periodic orbit on each patch.

B. The phase-difference model

We denote $\theta_i \in [0, 2\pi]$ as the phase variable of model (2) along the periodic orbit $\gamma(t)$ on patch i. Following the theory for weakly connected networks,[16] Izhikevech[18] proved that for small dispersal rates δ, these phase variables satisfy the following two-dimensional model (neglecting terms of order δ^2)

$$\begin{aligned}\frac{d\theta_1(t)}{dt} &= \Omega + \delta H(\theta_2(t-\tau) - \theta_1(t)),\\ \frac{d\theta_2(t)}{dt} &= \Omega + \delta H(\theta_1(t-\tau) - \theta_2(t)).\end{aligned} \tag{4}$$

The 2π-periodic function H is given by

$$H(x) = \frac{1}{T}\int_0^T \hat{\gamma}(t) \cdot W\big(\gamma(t), \gamma(t + x/\Omega)\big)dt, \tag{5}$$

where $\hat{\gamma}(t)$ is the unique solution of

$$\frac{d\hat{\gamma}(t)}{dt} = -DF(\gamma(t))^T \hat{\gamma}(t) \tag{6}$$

with the normalization condition $\hat{\gamma}(t) \cdot \gamma'(t) = 1$.

Next, we rescale time by δ and introduce the phase deviation variables ϕ_i through $\theta_i(t) = \Omega t + \phi_i(\delta t)$. System (4) can now be rewritten as

$$\frac{d\phi_i(t)}{dt} = H\Big(\phi_j(t - \delta\tau) - \phi_i(t) - \Omega\tau\Big), \tag{7}$$

for $i, j = 1, 2$ and $j \neq i$. Since δ is small, the additional assumption that τ does not exceed unity will make the product $\delta\tau$ small so that one can remove the delay in the first argument. This simplification allows one to write the single equation for the phase difference $\phi := \phi_1 - \phi_2$ as

$$\frac{d\phi(t)}{dt} = G(\phi) := H(-\phi - \Omega\tau) - H(\phi - \Omega\tau). \tag{8}$$

Obviously, function G is also 2π-periodic. We will study this function for a number of different parameter combinations in Sec. III (Figures 2–4). A constant solution ϕ^* satisfying $G(\phi^*) = 0$ represents the phenomenon of phase-locking. We say, that the system is in-phase synchronized when it is locked at $\phi^* = 0$, and that the system is anti-phase synchronized when it is locked at $\phi^* = \pi$. The sign of $G'(\phi^*)$ determines the local stability of the phase-locked state ϕ^* in model (8). If it is positive (negative), the steady state ϕ^* is unstable (stable). The absolute value $|G'(\phi^*)|$ indicates the rate of convergence to phase locking.

We observe numerically the effect of the density-dependent dispersal and travel-time delay on phase locking. We use the software package XPPAUT[11] to first find

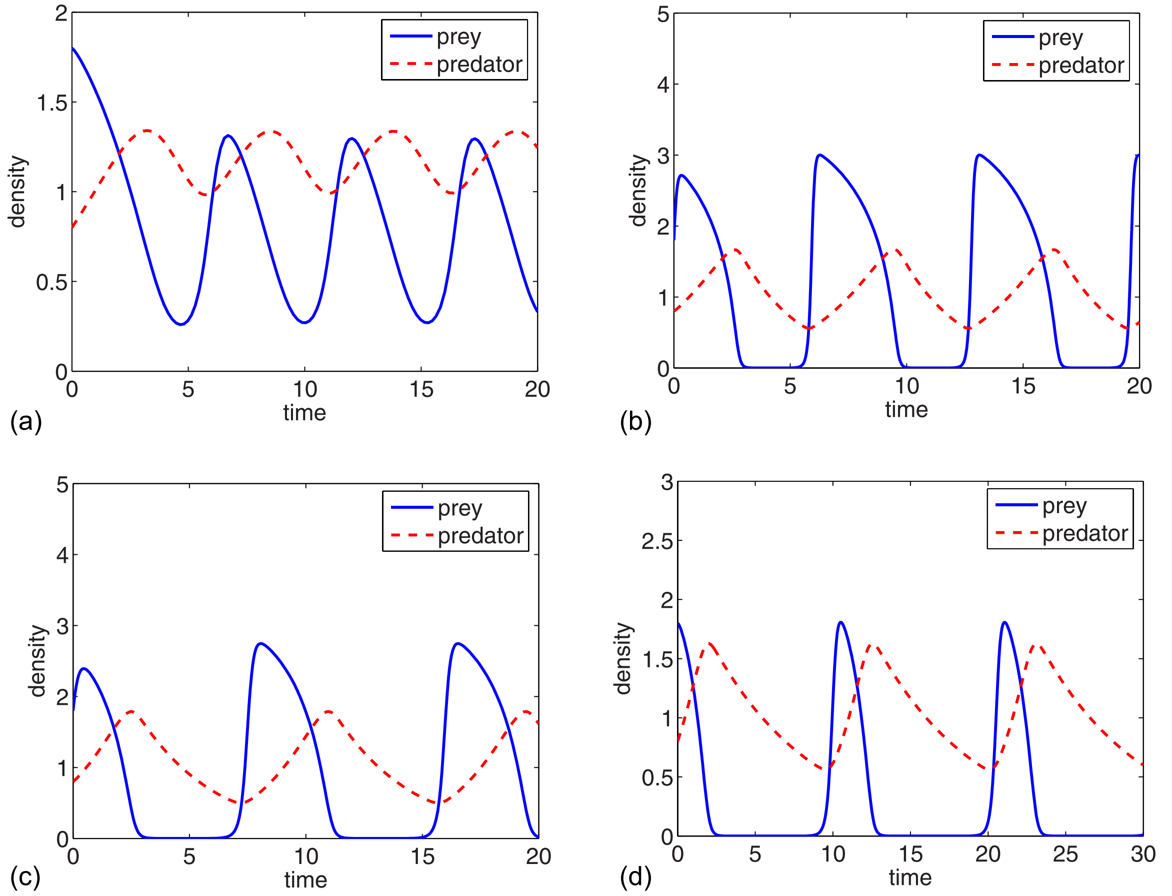

FIG. 1. The shape of the oscillations in the single-patch MacArthur-Rosenzweig model. Parameter values are chosen as in scenarios (a)–(d) from Goldwyn and Hastings.[13] (a) Regular oscillations with $\epsilon = 0.1$, $k = 0.4$, $\mu = 0.4$, (b) Relaxation oscillations with $\epsilon = 0.05$, $k = 0.28$, $\mu = 0.38$, (c) Relaxation oscillations with $\epsilon = 0.1$, $k = 0.3$, $\mu = 0.3$, (d) Relaxation oscillations with $\epsilon = 0.1$, $k = 0.4$, $\mu = 0.15$.

the periodic solution $\gamma(t)$ and its period T from the single-patch MacArthur-Rosenzweig model in (1). Then, we calculate the solution $\hat{\gamma}(t)$ and the interaction function H. Finally, we compute and plot function G that determines the dynamics of the phase differences. To compare and contrast with previous results and to observe the behavior with respect to different time scales between predator and prey, we choose the same parameter sets as Goldwyn and Hastings,[13] and refer to them as (a)–(d), accordingly. Scenario (a) represents similar time scales and corresponds to regular oscillations; scenarios (b)–(d) show differences in time scales and exhibit oscillations that are reminiscent of relaxation oscillations. Figure 1 shows parameter values and corresponding shapes of oscillations for these four scenarios. The parameters used in Ref. 32 are different, but the behavior of the system there is similar to parameter set (b) from Ref. 13.

III. RESULTS

A. The effect of density-dependent dispersal on synchronization

Density-dependent dispersal, in the absence of any travel-time delays, may destabilize in-phase locking and can introduce out-of-phase locking. We illustrate the various scenarios by plotting the graph of the function G in (8) that determines the dynamics of the phase-difference variable ϕ. Figure 2 illustrates the different responses between the four different parameter sets. In scenario (a), in-phase locking (i.e., $\phi = 0$) is stable in the absence of density-dependent dispersal (i.e., $\alpha = 0$, solid line), and anti-phase locking (i.e., $\phi = \pi$) is unstable. No other phase-locked states exist. Neither intermediate (i.e., $\alpha = 0.5$, dashed) nor strong (i.e., $\alpha = 1$, dotted) density-dependent dispersal can alter the qualitative behavior of the system, but both do affect the speed of convergence to in-phase locking.

In scenario (b), only the in-phase locked (stable) and anti-phase locked (unstable) states exist when dispersal is density-independent (solid line), but intermediate density-dependent dispersal ($\alpha = 0.5$) can induce two out-of-phase locked states, one unstable and one stable (dashed line). Complete density-dependent dispersal ($\alpha = 1$) reverses the stability of the in-phase locked (now unstable) and anti-phase locked (now unstable) states (dotted line). In scenario (c), the in-phase locked state is stable for density-independent dispersal, but with a very small basin of attraction. An intermediate, out-of-phase state near $\phi = \pi/2$ is stable. Introducing density-dependent dispersal into this scenario moves the out-of-phase state to larger values and eventually makes the in-phase solution unstable and the anti-phase solution stable. In scenario (d), the in-phase locked state is stable, but with a very small basin of attraction, and anti-phase locking is stable. Density-dependent dispersal has no visible effect on the dynamics nor on the rate of convergence.

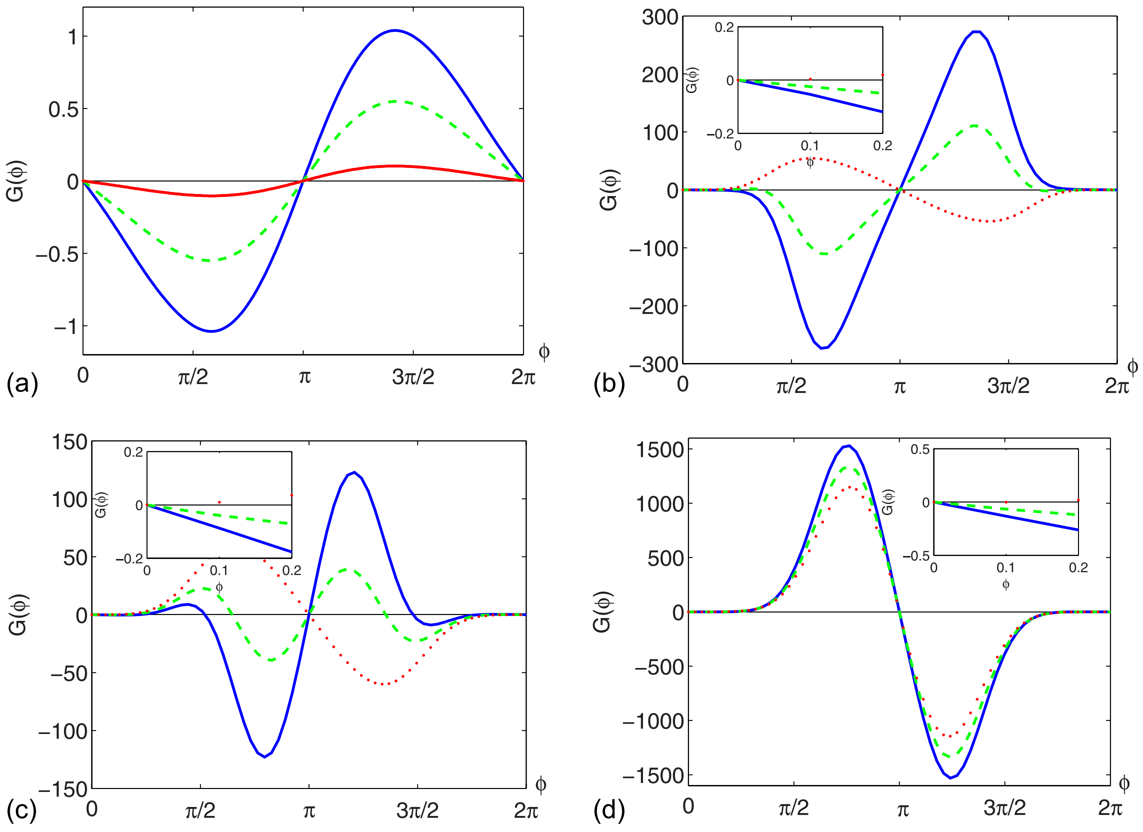

FIG. 2. The effect of density-dependent dispersal on the dynamics of the phase difference ϕ in equation (8) when $\tau = 0$. We plot function G for $\alpha = 0$ (solid), $\alpha = 0.5$ (dashed), and $\alpha = 1$ (dotted). Numerical evaluation shows that $G'(0) < 0$ in all cases, except when $\alpha = 1$ in (b) and (c). The inset magnifies the graph of G near $\phi = 0$ to demonstrate that $\phi = 0$ is stable in all cases. (a) G-function with $\epsilon = 0.1$, $k = 0.4$, $\mu = .4$, (b) G-function with $\epsilon = 0.05$, $k = 0.28$, $\mu = 0.38$, (c) G-function with $\epsilon = 0.1$, $k = 0.3$, $\mu = 0.3$, (d) G-function with $\epsilon = 0.1$, $k = 0.4$, $\mu = 0.15$.

B. The effect of travel time on synchronization

We now consider the effect of travel-time delay (τ) on the shape of the function G and hence on the dynamics of the phase difference in the absence of density-dependent dispersal. The four cases are illustrated in Figure 3; a scenario similar to (b) was originally investigated in much detail by Wall *et al.*[32]

In scenario (a), the in-phase locked state remains stable for small and intermediate delays (e.g., $\tau = 0.1$, dotted, and $\tau = 0.5$, dashed), but becomes unstable for large delays (e.g., $\tau = 1$, dash-dot), when the anti-phase locked state is stable. It turns out that in all four scenarios, $\phi = 0$ is unstable and $\phi = \pi$ is stable for large values of τ. Just as in Sec. III A, introducing a delay can induce intermediate, out-of-phase locked states in scenarios (b) and (c). In contrast to Sec. III A where intermediate to large values of α were necessary to destabilize, the in-phase locked solution ($\phi = 0$) becomes unstable already for relatively small values of τ. We refer to Wall *et al.*[32] for a more detailed bifurcation analysis of the in-phase locked state in a scenario similar to (b).

C. The joint effect of the density-dependent dispersal and travel-time delay

When $\alpha = 0$, then increasing τ increases $G(\phi)$ pointwise for all $\phi \in (0, \pi)$ and decreases $G(\phi)$ for $\phi \in (\pi, 2\pi)$ by symmetry. Accordingly, the in-phase locked state may lose stability, the anti-phase locked state will eventually be stable, and out-of-phase locked states may arise for intermediate values of τ through a saddle-node bifurcation so that one is stable and one unstable. The pointwise increase of $G(\phi)$ in $\phi \in (0, \pi)$ still occurs when $\alpha > 0$ is fixed. For example, compare Figures 2(b), 4(b)–4(d). In particular, increasing τ for any fixed value of α will (i) tend to destabilize in-phase locking, (ii) tend to stabilize anti-phase locking, and (iii) move out-of-phase locked states in $(0, \pi)$ closer to π.

The effect of α on $G(\phi)$ is not monotone when $\tau = 0$. While $G(\phi)$ increases pointwise with α for $\phi \in (0, \pi)$ in scenario (a), it decreases pointwise in the same interval in scenario (d), see Figure 2. This behavior for scenarios (a) and (d) also holds when τ is positive, e.g., Figure 4(a). Characteristically in those cases, function G is of one sign in the interval $(0, \pi)$: negative in scenario (a) and positive in scenario (d).

Figure 4(a) also demonstrates that increasing density-dependent dispersal in conjunction with positive τ can change the stability of the in-phase locked state from stable to unstable, which was impossible for $\tau = 0$.

Figures 4(b) and 4(c) show that increasing α may increase or decrease $G(\phi)$ independent of its sign. In all cases, however, the intermediate, out-of-phase locked state (if there is one), will move closer to $\phi = \pi$ as α increases.

Overall, the two effects that we studied, density-dependent dispersal and travel-time delays, seem to affect

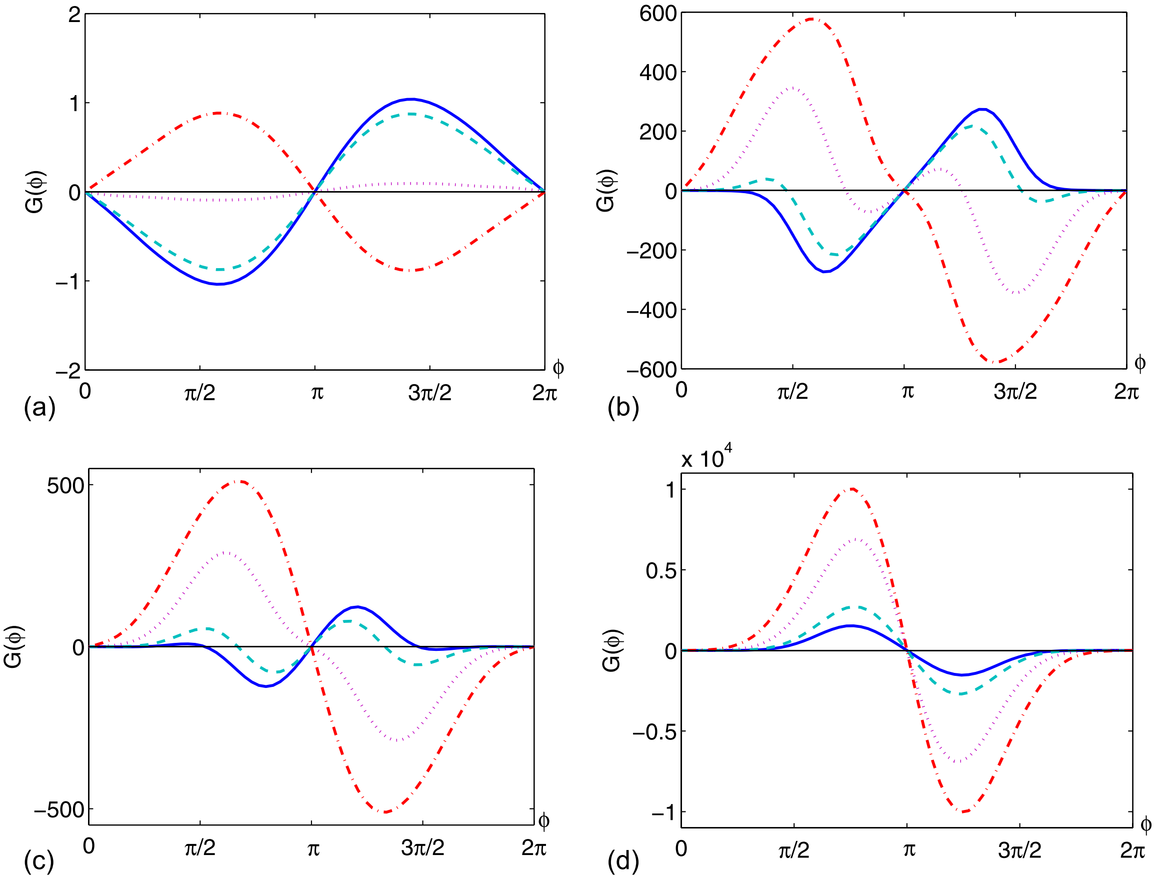

FIG. 3. The effect of travel delay τ on function G with $\alpha = 0$. The solid, dashed, dotted, dashed-dotted lines represent $\tau = 0, 0.1, 0.5, 1$, respectively. (a) G function with $\epsilon = 0.1$, $k = 0.4$, $\mu = .4$, (b) G function with $\epsilon = 0.05$, $k = 0.28$, $\mu = 0.38$, (c) G function with $\epsilon = 0.1$, $k = 0.3$, $\mu = 0.3$, (d) G function with $\epsilon = 0.1$, $k = 0.4$, $\mu = 0.15$.

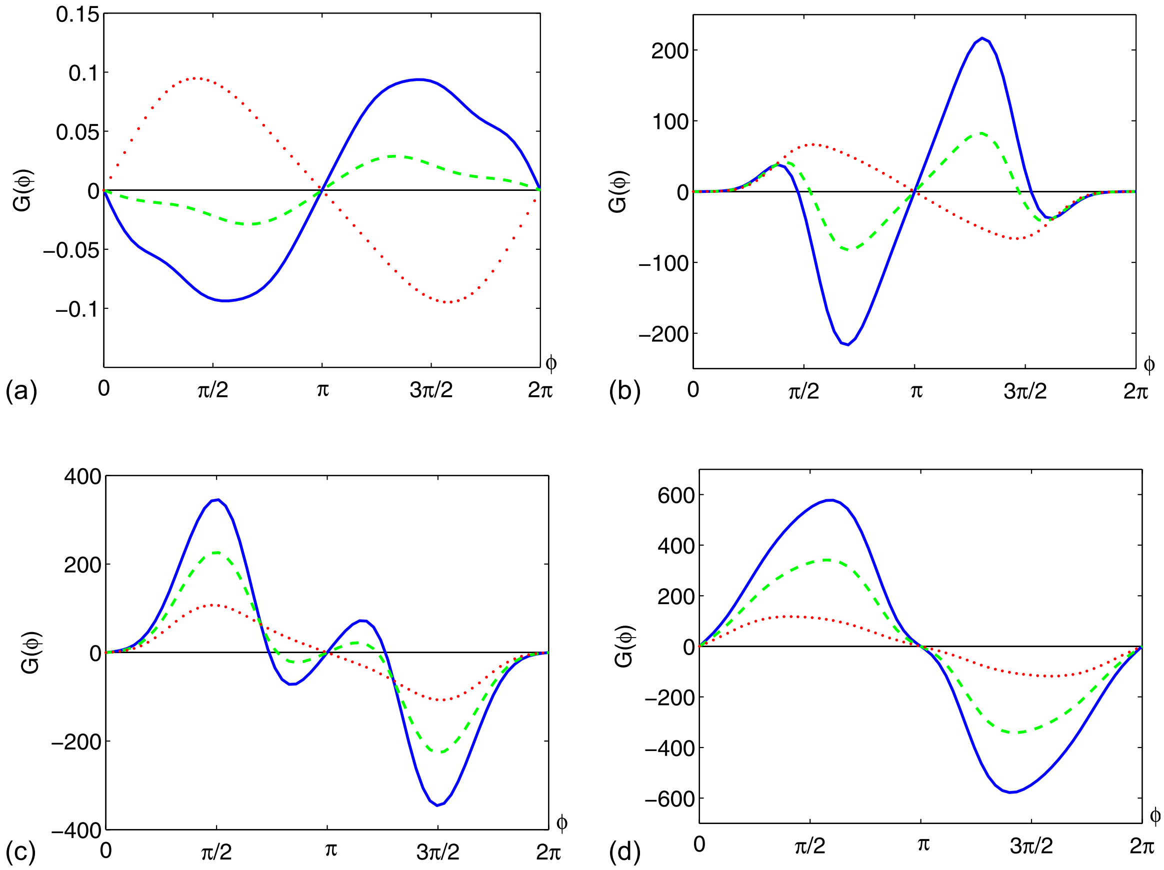

FIG. 4. Selected scenarios to illustrate the interaction between travel-time delay and density-dependent dispersal. Panel 4(a) corresponds to scenario (a), all other panels are scenario (b). In all plots, α varies between $\alpha = 0$ (solid), $\alpha = 0.5$ (dashed) and $\alpha = 1$ (dotted). (a) G function with $\tau = 0.5$ in scenario (a), (b) G function with $\tau = 0.1$ in scenario (b), (c) G function with $\tau = 0.5$ in scenario (b), (d) G function with $\tau = 1$ in scenario (b).

phase-locked states in similar ways. Both can destabilize the in-phase locked state and stabilize the anti-phase locked state. Both can give rise to intermediate out-of-phase locked states, and increasing either tends to move these states closer to anti-phase locking. While even small values of $\tau > 0$ can reverse stability of $\phi = 0$ in some scenarios and large enough values will reverse stability in all cases, the same is not true for α. Increasing α cannot change stability in scenarios (a) and (d) when $\tau = 0$, but it can do so when τ is large enough. Therefore, the value of τ seems to control the extent to which parameter α can influence the state of the system.

IV. DISCUSSION

Synchrony, or the lack thereof, is an important property of spatially extended ecological systems. For example, extinction risk can increase with increased synchrony.[10] Dispersal-induced synchrony is documented in natural systems,[24] and theories predict its effect on population stability.[1] Dispersal-induced synchrony among populations is typically predicted for large dispersal rates. However, many ecological systems are coupled through small dispersal rates. In that case, Goldwyn and Hastings[13] showed that fast convergence to in-phase synchrony is most likely within a trophic meta-community (2-patch predator-prey system) in the presence of time-scale differences between species. Under those conditions, while in-phase locking is locally stable, other locally stable states can arise and have large basins of attraction. In addition, a small travel-time delay can even make in-phase locking unstable.[32] Our results here show further that density dependence in the time to movement interacts with travel time between habitats to prevent the onset and stability of in-phase synchrony. One ecological implication of our results is that dispersal-induced synchrony could be more rare than assumed.

A number of studies have shown the stabilizing effect of dispersal delays on cyclic dynamics:[22,34] cycle amplitude can decrease to zero and render the coexistence steady-state stable so that no cycles exist. On the other hand, it is also known that stronger cycles can promote synchrony in models and experiments.[20,31] One could therefore speculate that density dependence and time delays in our model reduce cycle amplitude (but not to zero) and thereby make synchrony less likely. Since our method of studying the phase difference does not consider amplitudes, we cannot investigate this potential mechanism in our theoretical framework. Numerical simulations of the two-species, two-patch model show a minimal decrease in cycle amplitude. This question is open for future investigation. Our results are related to previous work for a single-species model.[17] The authors there considered a discrete-time model with cyclic behavior generated by a Gompertz-type growth function. Negative density-dependent dispersal would destroy spatial synchrony in their stochastic model. Our per-capita dispersal rate is also decreasing with prey density.

We assumed that population dynamics on the two patches are identical whereas they are bound to be somewhat different in reality. The question of how dispersal can synchronize non-identical oscillators has been studied in detail in the physics literature,[5,9] but has received little attention in ecology.[7] In the following, we identify key ecological processes, and their interactions within and outside of habitats, that can explain the maintenance of spatiotemporal heterogeneity, and thus the persistence of many natural ecosystems.

A. Interactions between time to movement and travel time

Simple two-patch models assume instantaneous passive movement with constant time until movement. In natural landscapes, non-zero travel time is expected between discrete patches distributed across a landscape matrix.[12] Such travel times result from the distance between habitats, but are also affected by the properties of the landscape matrix and its impact on movement speed.[29] Similarly, time until travel during the life span of an individual is an important temporal scale characterizing natural communities. It has been generally modeled as a constant life-history characteristic, but many studies have explored density-dependence in that temporal scale resulting from behavioral aspects of foraging,[26] predator avoidance,[30] or competition.[4] In particular, simulation studies show that predator avoidance by prey can prevent in-phase synchrony[23] and can have strong effects on foodweb structure and stability as trait-mediated indirect effect.[2,33] Previous studies had revealed the importance of differences in the temporal scales of demographic processes between predator and prey species,[13] which can be characterized by the time to capture of prey.[35] Our results show how density-dependent movement and travel time introduce additional key interactions between temporal scales of movement. The effect of density-dependent movement generates fluctuations in movement rate (time until movement), and its effect on synchrony depends on both travel time and time to capture. Both temporal scales of movement, namely, the time to movement and travel time, can be seen to extending the range of demographic parameter values over which travel time can destabilize in-phase synchrony. This interaction has important implications for the study of spatially structured communities. It reveals how local behavioral drivers of movement such as predator avoidance can affect the consequences of travel time across the landscape on the maintenance of regional variability (out-of-phase synchrony). More specifically, by aggregating movement during phases of high predator densities, prey can reinforce the destabilizing effect their travel time has on population in-phase synchrony. This is an important connection between local behaviour and regional community dynamics, which could improve the prediction of whole community consequences of habitat fragmentation.

B. In-phase synchrony and ecological persistence

Regional persistence of communities is key to predicting their structure and evolution, and it relates to management goals and conservation priorities. Strong fluctuations of abundance are commonly observed[21] and can drive local populations to extinction. Spatial structure and movement of organisms between habitats can stabilize such fluctuations.[8] Given the lack of in-phase synchrony among local communities, the resulting maintenance of spatiotemporal heterogeneity has been studied as a mechanism of regional persistence in the face of habitat fragmentation.[28] However, studies have shown how even weak dispersal can lead to in-phase synchrony[13] and have thus challenged the maintenance of regional variability as a mechanism of persistence of metapopulations and metacommunities. We have shown that very common characteristics of weak dispersal, its local density-dependence, and travel time across landscapes, can additively prevent the stability or slow down the convergence to in-phase synchrony. Our results thus provide novel ground to motivate the investigation of dispersal as a cause of regional heterogeneity and persistence. Because we highlight the role of non-consumptive effects of predation on synchrony through predator avoidance, our results also contribute to the integration between theories of metacommunities and of trait-mediated indirect interactions.[3,33] Finally, the importance of landscape feature for movement of organisms has been investigated through habitat fragmentation and corridors, which are often assumed to affect the rate of movement between habitats in theoretical studies. Our analysis shows that the temporal scales of movements driven by both landscape properties and behavior are key for explaining the maintenance of heterogeneity across weakly connected communities.

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