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REVIEW AND SYNTHESIS

A dispersal-induced paradox: synchrony and stability in stochastic metapopulations

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Abstract

Understanding how dispersal influences the dynamics of spatially distributed populations is a major priority of both basic and applied ecologists. Two well-known effects of dispersal are spatial synchrony (positively correlated population dynamics at different points in space) and dispersal-induced stability (the phenomenon whereby populations have simpler or less extinction-prone dynamics when they are linked by dispersal than when they are isolated). Although both these effects of dispersal should occur simultaneously, they have primarily been studied separately. Herein, I summarise evidence from the literature that these effects are expected to interact, and I use a series of models to characterise that interaction. In particular, I explore the observation that although dispersal can promote both synchrony and stability singly, it is widely held that synchrony paradoxically prevents dispersal-induced stability. I show here that in many realistic scenarios, dispersal is expected to promote both synchrony and stability at once despite this apparent destabilising influence of synchrony. This work demonstrates that studying the spatial and temporal impacts of dispersal together will be vital for the conservation and management of the many communities for which human activities are altering natural dispersal rates.

Keywords

Autoregressive model, correlated environmental stochasticity, dispersal, dispersal-induced stability, metapopulation, negative binomial model, Ricker model, spatial heterogeneity, synchrony.

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INTRODUCTION

One of the most fundamental results in spatial ecology is that groups of subpopulations can have dramatically different dynamics when they are linked by dispersal than when they are isolated. In many instances, dispersal among spatially separated subpopulations can have a strong stabilising influence, and such dispersal-driven stability has been proposed as a key factor promoting coexistence in ecological communities.

There is an emergent consensus that, for dispersal to enhance stability, subpopulations must remain asynchronous. If dispersal is sufficient to cause complete intraspecific synchrony, the exchange of individuals among those identical subpopulations cannot stabilise otherwise unstable local dynamics (for a thorough review, see Briggs & Hoopes 2004). In discussing the stabilising effects of dispersal, synchrony has thus been treated largely as a caveat: dispersal can be stabilising *unless dispersal is strong enough to induce perfect synchrony*. Meanwhile, the synchronising effect of dispersal is itself an extremely well-studied topic (Bjørnstad *et al.* 1999; Koenig 1999; Liebhold *et al.* 2004), but it has been developed more or less separately from the work on dispersal-induced stability. Although it is clear that perfect synchrony (where fluctuations are identical and in phase) impedes dispersal-induced stability, the effects of imperfect levels of synchrony like those seen in nature (where fluctuations are positively correlated, but not identical) are far less obvious. Do processes that promote *any* degree of synchrony necessarily weaken dispersal-induced stability? Or can some processes simultaneously promote synchrony and stability, as long as synchrony is incomplete? These questions are complicated because the propensity for dispersal-induced stability is influenced directly by attributes such as dispersal rate, local density-dependent processes and environmental variability. These same attributes can also affect population synchrony and, due to synchrony's effect on dispersal-induced stability, they should have indirect effects on stability that are separate from their direct effects. A paradox can emerge when these direct and indirect effects work in opposite directions, and attempting to intuit the net result is a messy endeavor, as will become apparent below.

Fully understanding the effects of dispersal is of central importance to basic ecology because, to put it simply, we know that individuals move around and we know that such movement can have profound impacts at the population level (Levin 1974). Understanding the role of dispersal is fundamental to applied ecology as well. Human activities can interrupt (e.g. through fragmentation) or accelerate (e.g. by intentional and unintentional spreading of propagules) how species move through space, and to mitigate any of these actions, we must understand the function of dispersal. This is a difficult task, and ecologists have long recognised that dispersal has complex and sometimes contradictory effects on population dynamics. Dispersal has been described as a 'double-edged sword' (Hudson & Cattadori 1999): it may protect small subpopulations from local extinction by allowing an influx of immigrants, but dispersal may also elevate the risk of global extinction by synchronising subpopulations across a landscape, so that all are simultaneously small. A synthetic framework for the multiple simultaneous consequences of dispersal is ultimately needed for answering basic and applied questions about the role of dispersal in natural populations. In this article, I adopt the strategy of studying the theoretical relationship between dispersal's stabilising and its synchronising effects as a way to predict which direction the double-edged sword actually cuts.

Dispersal-induced stability (Taylor 1990; Briggs & Hoopes 2004) and synchrony (Bjørnstad *et al.* 1999; Koenig 1999; Liebhold *et al.* 2004) have each been reviewed separately elsewhere. Herein, I summarise key information from past studies that sheds light on the relationship between dispersal-induced stability and synchrony. I then characterise the synchrony–stability relationship for a series of models using both analytical and simulation methods. We see that although past studies would mostly lead us to expect synchrony-promoting factors to reduce stability, in many situations, synchrony and stability are strengthened simultaneously. These results constitute a step towards an integrated understanding of dispersal's effects on spatially distributed populations.

LESSONS FROM THE LITERATURE

Synchronising effects of dispersal

There is broad agreement that dispersal can synchronise spatially-distinct subpopulations (Bjørnstad *et al.* 1999; Koenig 1999; Liebhold *et al.* 2004; Goldwyn & Hastings 2008). Intuitively, we might expect that if some dispersal causes some degree of synchrony, more dispersal will cause more synchrony. This indeed appears to be the case for many models (e.g. Hanski & Woiwod 1993; Ranta *et al.* 1998; Lande *et al.* 1999), although others show a U-shaped (Jansen 2001), hump-shaped (Ripa 2000) or even decreasing (Koelle & Vandermeer 2005) relationship between dispersal and synchrony. Experimental studies have mostly shown higher synchrony with greater dispersal (Holyoak & Lawler 1996; Dey & Joshi 2006; Vogwill *et al.* 2009), and an observed correlation between dispersal ability and synchrony in natural bird populations supports this notion (Paradis *et al.* 1999).

For subpopulations with cyclic dynamics due at least partly to intrinsic nonlinearities, even small amounts of dispersal can result in very high synchrony by phase locking (Jansen 1999; Bjørnstad 2000). However, if local fluctuations in density are instead due entirely to stochastic variation, the synchronising effect of dispersal is often negligible (Hanski & Woiwod 1993; Haydon & Steen 1997; Vasseur & Fox 2009). Dispersal patterns that depend on local densities may result in lower synchrony than comparable amounts of density independent dispersal (Ims & Andreassen 2005; Li *et al.* 2005). In general, a given level of dispersal can result in very different levels of population synchrony depending on what other deterministic and stochastic factors affect local dynamics.

Stabilising effects of dispersal

Interpreted broadly, 'stability' can refer to the dynamical stability of an equilibrium solution, a small amplitude of fluctuations in oscillatory populations, a minimum population density that is not too low, a long persistence time or a low probability of extinction in a given time frame. All of these represent real phenomena relating to different aspects of stability and if any of these is strengthened when subpopulations are linked by dispersal relative to when they are isolated, then we have dispersal-induced stability ('DIS'; see Allen 1975; Reeve 1988; Martí & Masoller 2003; Hillary & Bees 2004; Ives *et al.* 2004, for examples with different types of stability). Random dispersal alone cannot increase population stability (Reeve 1988), but the combination of spatial heterogeneity and dispersal can generate DIS in several different ways (reviewed in Briggs & Hoopes 2004). First, stability can result when the net immigration rate into a particular subpopulation is essentially independent of the local population density (e.g. Crowley 1981; Hastings 1993; Amarasekare 2008). This can occur, for example, if individuals leave their current subpopulation at a constant rate and subpopulations are at least somewhat asynchronous. Transit time can likewise be stabilising by introducing a time delay that decouples immigration from current density (Neubert *et al.* 2002; Klepac *et al.* 2007).

DIS arises by a second mechanism in some multi-species models (e.g. Gurney *et al.* 1998; de Roos *et al.* 1998; Gurney & Veitch 2000), where self-organised spatial patterns result in spatially heterogenous population densities. As individuals move around this heterogenous landscape, the average demographic parameters they experience will differ from parameters in an analogous, but homogeneous landscape, due to the nonlinear nature of species interactions. In some cases, those nonlinear average parameters produce more stable dynamics than the parameters of the well-mixed landscape. Furthermore, this spatial heterogeneity in population densities can simultaneously promote the first type of dispersal-induced stability, by weakening the relationship between local density and net immigration (Hassell *et al.* 1991a; Comins *et al.* 1992).

Under conditions where populations can be deemed more stable with dispersal than without it, there is little consensus on whether adding more dispersal will be more or less stabilising (Gonzalez-Andujar & Perry 1993; Gyllenberg *et al.* 1993; Hastings 1993; Doebeli 1995). Indeed, DIS has been shown to either increase (e.g. Hassell *et al.* 1991b; Engen 2007), decrease (e.g. Taylor 1998; Hosseini 2003; Hirzel *et al.* 2007) or have a hump-shaped relationship (Allen 1975; Comins *et al.* 1992; Hastings 1993; Molofsky & Ferdy 2005; Abta *et al.* 2007) with increasing dispersal. Dispersal might even promote some types of stability while interfering with others (Reeve 1988, 1990; Legendre *et al.* 2008; Higgins 2009), and density dependent movement patterns may enhance (Hassell & May 1973; Chesson & Murdoch 1986; Doebeli 1995; Amarasekare 2004; Li *et al.* 2005) or inhibit (Reeve 1988; Murdoch *et al.* 1992; Rohani *et al.* 1994; Huang & Diekmann 2001; Sapoukhina *et al.* 2003; Higgins 2009) DIS. General arguments in favour of maximum DIS at intermediate dispersal rates recognise a balance between stabilising and destabilising effects of dispersal. When high dispersal rates maximise an individual's ability to migrate into high quality patches within a spatially variable landscape, and low dispersal rates minimise the risk of moving into a patch with declining quality within certain (autocorrelated) temporally variable landscapes, intermediate levels of dispersal allow populations to benefit most from both spatial and temporal sources of heterogeneity (Schreiber 2010). Intermediate dispersal may also balance a reduced local extinction risk from immigrants rescuing dwindling subpopulations, against an elevated global extinction risk from dispersal-driven synchrony allowing simultaneously low population densities (Reeve 1990; Adler 1993; Keeling 2000).

The interaction between synchrony and dispersal-induced stability

So far, we have seen two hints about the relationship between synchrony and DIS. First, dispersal among perfectly synchronous subpopulations has no stabilising effect (e.g. Abta *et al.* 2008; Higgins 2009; Hauzy *et al.* 2010). Second, dispersal among populations that are not perfectly synchronous can induce stability. If we think of these two statements as two points in the synchrony–DIS relationship, then we clearly have a negative relationship: greater synchrony is associated with less dispersal-induced stability. This is a bit unsatisfactory, as it

is obviously shaky to base a general relationship on only two points. Furthermore, one of these points has questionable relevance for real populations. Although population synchrony (that is, positively correlated population dynamics) is relatively common, perfect synchrony (identical population dynamics) is not expected for any real pairs of populations separated in space. If perfect synchrony is a special case and not indicative of the effects of strong, but incomplete synchrony on dispersal-induced stability, then we clearly lack the information needed to draw conclusions about the synchrony–DIS relationship (Hudgens 2007).

The synchrony–DIS relationship has rarely been studied explicitly. Araujo & de Aguiar (2008) found that both synchrony and DIS increased together as the number of subpopulations increased, suggesting a positive synchrony–DIS relationship. In contrast, Murdoch *et al.* (1992) varied the strength of density dependence in dispersal and found that DIS was nearly always higher when dispersal generated lower levels of synchrony. Together, these studies reveal that while some processes promote both synchrony and DIS, resulting in a positive relationship, others may promote one at the expense of the other, giving a negative one. The question at the heart of this review is whether changing the total amount of dispersal generates a positive or negative synchrony–DIS relationship. Increasing dispersal can have simultaneous synchronising and stabilising effects when there are fixed differences among patches (Gyllenberg *et al.* 1993). However, a dispersal rate with some given degree of DIS may produce very different levels of synchrony under different model conditions (Hastings 1993; Kendall & Fox 1998; Münkemüller & Johst 2007).

It is important to remember that 'stable' coupled subpopulations (those with equilibrial dynamics, low variance, high persistence, etc.) may or may not be stable due to DIS; DIS exists only if stability is greater with dispersal than without it. Linked subpopulations with complex population dynamics tend to have lower synchrony, lower global extinction risk and higher local extinction risk than those with simpler dynamics (Allen *et al.* 1993; Heino *et al.* 1997; Matter 2001). However, as local and global persistence are probably influenced simultaneously by dispersal and by the complexity of the local dynamics, the actual influence of synchrony on DIS is obscured. Similarly, other studies that report negative (Hanski & Woiwod 1993; Ripa 2000; Holland & Hastings 2008) or null (Griffen & Drake 2009) relationships between synchrony and various measures of stability in the presence of dispersal may not tell us much about how synchrony and DIS interact, as we need direct information on how much of the observed stability is due to dispersal to determine how synchrony modifies DIS. Nonetheless, an association between higher synchrony and increased variability or extinction emerges from many of these past studies, and this may lead us to also expect a negative relationship between synchrony and DIS.

Dispersal does not inevitably increase stability. Reaction-diffusion models, which lack the necessary combination of heterogeneity and subdivision, are either unaffected or destabilised by dispersal (Levin 1974), and even discrete and heterogeneous metapopulations are not invariably stabilised by dispersal (Kareiva 1987; Neubert *et al.* 1995; Rohani & Ruxton 1999; Vogwill *et al.* 2009). When dispersal is destabilising, it is easy to piece together a story that makes good intuitive sense: dispersal both decreases stability and increases synchrony, yielding a negative synchrony–DIS relationship that agrees with the prevailing idea that synchrony interferes with DIS. When dispersal *is* stabilising, the story becomes confusing. If dispersal promotes synchrony and stability simultaneously (Ruxton & Rohani 1999; Martí & Masoller 2003; Hillary & Bees 2004; Matthews & Gonzalez 2007), we expect an indirect positive synchrony–DIS relationship. However, if synchrony inhibits DIS, this suggests a direct negative relationship. One question with practical implications, then, is whether increasing linkages between habitat patches is likely to have an overall positive (due to DIS) or an overall negative (due to synchrony interfering with DIS) effect on population stability and persistence.

The role of environmental variability

Conditions affecting real populations are not uniform through time and space, and this can have important consequences for both synchrony and DIS (McMurtrie 1978; Heino 1998; Keeling *et al.* 2002; Bonsall & Hastings 2004). Variability due to fixed spatial heterogeneity (Ives 1992; Holt & Hassell 1993; Singh *et al.* 2004) or uncorrelated environmental stochasticity (Taylor 1998; Abta *et al.* 2007; Araujo & de Aguiar 2008) discourages synchrony and has thus been cited as a factor that can promote DIS (Abta *et al.* 2008). However, as countless synchrony researchers have pointed out, environmental variability is often spatially correlated (Koenig 1999) and may actually be a very common driver of population synchrony (e.g. Hanski & Woiwod 1993; Grenfell *et al.* 1998; Peltonen *et al.* 2002), challenging the notion that spatiotemporal variability necessarily promotes DIS by inhibiting synchrony. One goal of the present study is to consider spatially correlated environmental stochasticity while examining the relationship between synchrony and DIS.

QUANTIFYING THE SYNCHRONY–DIS RELATIONSHIP

For any metapopulation model, a given dispersal rate will lead to a particular expected level of synchrony and a particular expected strength of dispersal-induced stability. A different dispersal rate might give different levels of synchrony and DIS, and by looking across multiple dispersal rates, the synchrony–DIS relationship emerges. If dispersal rates that promote synchrony also inhibit stability, then we will see a negative synchrony–DIS relationship.

Following convention in the synchrony literature, I use the average pairwise correlation coefficient among subpopulation dynamics to measure population synchrony. Many different ways of measuring population stability have been employed, and I will consider several of them. For the analytical work presented below, I use the within-subpopulation variance in density as a metric of stability because the variance is relatively straightforward to calculate. Dispersal-induced stability, then, is the extent to which this variance is reduced in the presence of dispersal relative to an identical collection of subpopulations not linked by dispersal. I use the ratio of the variance without dispersal to the variance with dispersal as the main measure of DIS. When the variance with dispersal is small relative to the variance without dispersal, this ratio will be large and thus high values of the DIS metric imply greater dispersal-induced stability. Different measures of stability may behave differently, so for the simulations below, I consider not only the population variance but also the local and global extinction rates. For all metrics, I again use the ratio of the values without to with dispersal to quantify DIS.

ANALYTICAL SYNCHRONY–DIS RELATIONSHIP IN A SIMPLE MODEL

Ecological models become complicated quickly as spatial patches and species are added, so it is useful to begin with a single-species

model that is simple enough that quantities like synchrony and DIS can be solved for explicitly. Consider n first-order autoregressive (AR(1)) subpopulations linked by global, density independent dispersal. If $H_{i,t}$ is the density of our species of interest in subpopulation i at time t, then

$$H_{i,t} = a(1-d)H_{i,t-1} + \sum_{\substack{j=1 \\ j\neq i}}^{n} \frac{ad}{n-1} H_{j,t-1} + \varepsilon_{i,t}. \quad (1)$$

Herein, d is the fraction of each subpopulation to disperse each time step and a describes the effects of reproduction, death and density-independent species interactions. The term $\varepsilon_{i,t}$ is a random variable representing environmental stochasticity. I assume that this stochasticity has mean 0 and variance σ_ε^2, is temporally uncorrelated and has spatial correlation ρ_ε.

The within-patch temporal variance in density in the absence of dispersal, denoted σ_0^2, is $\sigma_\varepsilon^2/(1-a^2)$ and the variance in the presence of dispersal is $\sigma_H^2 = \sigma_\varepsilon^2 \frac{c_1 - c_2(1-(n-1)(1-\rho_\varepsilon))}{(1-a^2)(c_1-c_2)}$ (Ripa 2000), where the substitutions $c_1 = 1 - a^2\left((1-d)^2 + \frac{d^2}{n-1}\right)$ and $c_2 = -\frac{a^2}{n-1}\left(2d - \frac{nd^2}{n-1}\right)$ are used here for brevity. DIS, then, is the ratio

$$\frac{\sigma_0^2}{\sigma_H^2} = \frac{c_1 - c_2}{c_1 - c_2(1-(n-1)(1-\rho_\varepsilon))}. \quad (2)$$

Population synchrony is given by the spatial correlation in abundance,

$$\rho_H = \frac{c_1\rho_\varepsilon - c_2}{c_1 - c_2(1-(n-1)(1-\rho_\varepsilon))} \quad (3)$$

(Ripa 2000). These results assume $|a| < 1$, as subpopulations with $|a| > 1$ lack a stationary distribution of population densities for which statistical properties like the variance may be calculated.

Equations (2) and (3), respectively, give the levels of DIS and synchrony that are expected for specified values of a, d, n and ρ_ε. The synchrony–DIS relationship emerges from changes in these values that cause concurrent changes in both synchrony and DIS; herein we are interested in the concurrent effects of changing d. The partial derivatives of equations (2) and (3) with respect to d reveal that synchrony and DIS are always affected the same way by changes in d (Fig. 1a,b; Appendix S1). For most parameter values, this means that both quantities increase with d. In this simple model, then, the relationship between synchrony and DIS is positive for any values of a, n and ρ_ε (Fig. 1c).

The slope of the synchrony–DIS relationship is steepest for lower environmental correlations and flattens as ρ_ε increases (Fig. 1c). At $\rho_\varepsilon = 1$, the relationship condenses to a single point at which DIS and synchrony are both 1. This is the limiting case where dispersal cannot induce stability because the populations are perfectly synchronous. The other situation in which dispersal cannot induce stability is when $a = 0$, in which case the population dynamics are purely stochastic and dispersal therefore has no effect (the populations are not, however, perfectly synchronous at $a = 0$ unless $\rho_\varepsilon = 1$). Apart from these two special cases where d has no effect on either synchrony or DIS, levels of dispersal that enhance dispersal-induced stability always simultaneously promote synchrony.

SYNCHRONY–DIS RELATIONSHIP IN NONLINEAR METAPOPULATIONS

The AR(1) model has the advantage that it can be fully understood analytically, but it fails to capture the nonlinear intra- and interspecific interactions that characterise real communities. I now use classic nonlinear single-species and host–parasitoid models to study the synchrony–DIS relationship in more realistic ecological systems. The notion that dispersal can be stabilising has generated quite a lot of interest with respect to host-parasitoid communities, in particular, because, although such communities persist in nature, simple models

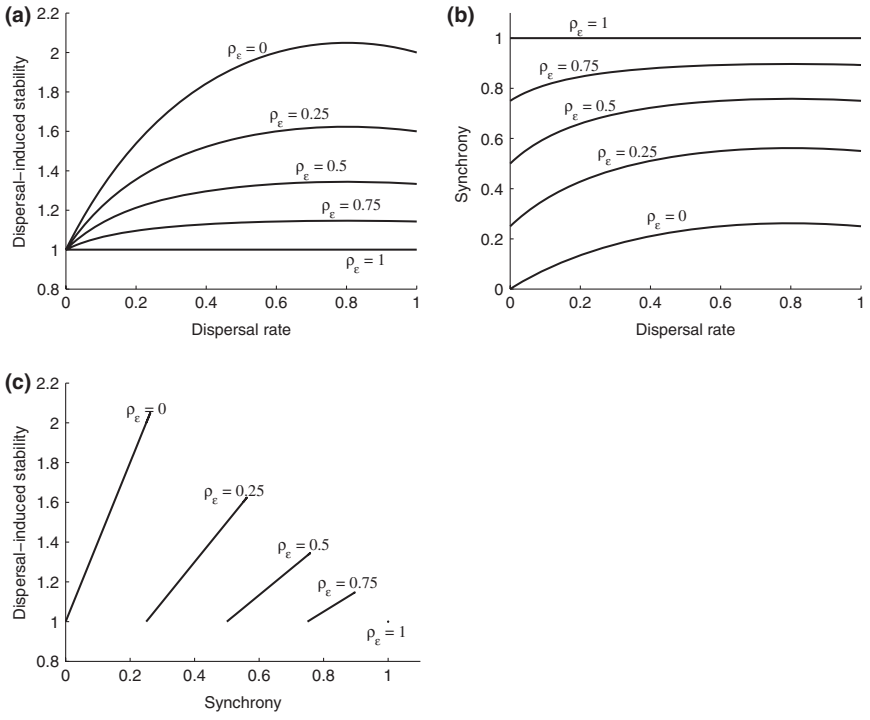

Figure 1 The effect of changing dispersal rate, d, on both dispersal-induced stability and synchrony in the AR(1) model. Synchrony was measured as the pairwise correlation in population densities. Stability was measured as the temporal variance in density and DIS is the ratio of this variance in the absence of dispersal to the variance with dispersal. The relationships shown here are given by equations (2) and (3). (a) Dispersal rate vs. DIS; (b) dispersal rate vs. synchrony; (c) synchrony vs. DIS. In all instances, $a = 0.8$ and $n = 5$; labels indicate the value of ρ_ε used for each line plotted.

of host–parasitoid interactions are particularly prone to instability. The work presented below shows that although the mathematical and biological assumptions of nonlinear models differ enormously from the very simple AR(1), the shape of the synchrony–DIS relationship is largely robust to these differences.

Models

A common model for nonlinear, single-species dynamics is the Ricker model,

$$H'_{i,t} = H_{i,t-1}\exp(\lambda(1 - H_{i,t-1})), \tag{4}$$

where $H'_{i,t}$ is the population density in subpopulation i at time t before accounting for any effects of dispersal, and $H_{i,t-1}$ is the density after accounting for dispersal that occurred during time step $t-1$. When subpopulations are linked by dispersal, a separate dispersal model, discussed below, describes the transition from $H'_{i,t}$ to $H_{i,t}$. The maximum local population growth rate is $\exp(\lambda)$, and the realised population growth rate decreases with local density. In the absence of dispersal and for $\lambda < 2$, the Ricker model has a stable equilibrium point at $H = 1$; for larger values of λ the model exhibits 2^n-point cycles and chaos.

Most host–parasitoid metapopulation models are based on the Nicholson–Bailey model or its variant, the negative binomial model:

$$H'_{i,t} = \lambda H_{i,t-1}\left(1 + \frac{bP_{i,t-1}}{k}\right)^{-k} \tag{5a}$$

$$P'_{i,t} = H_{i,t-1}\left(1 - \left(1 + \frac{bP_{i,t-1}}{k}\right)^{-k}\right). \tag{5b}$$

As before, $H'_{i,t}$ and $P'_{i,t}$ are, respectively, the densities of hosts and parasitoids in subpopulation i at time t before dispersal, and $H_{i,t-1}$ and $P_{i,t-1}$ are the densities after dispersal. In the absence of the parasitoid, the host population increases at rate λ. The mean per capita parasitism rate is b and parasitoid attacks are assumed to be distributed non-randomly among hosts according to a negative binomial distribution with shape parameter k. At $k = \infty$, attacks are randomly distributed among hosts and model (5) becomes equivalent to the standard Nicholson–Bailey model. With $\lambda > 1$ and $k < 1$, the negative binomial model predicts stable coexistence of the host and parasitoid. Otherwise, the populations undergo divergent oscillations and eventual extinction in the absence of dispersal.

It is interesting to notice that if we assume that the parasitoid population is constant in time, the host–parasitoid model is equivalent to the AR(1) with $a = \lambda\left(1 + \frac{bP}{k}\right)^{-k}$. This assumption changes the interpretation of the model in important ways: although models like equation (5) describe interactions between a tightly coupled pair of species, the simplified AR(1) model instead represents the case where hosts are attacked by a stable population of generalist parasitoids. Thus, although the algebra needed to get from the host-parasitoid model to the AR(1) model is quite simple, both the mathematical and biological characteristics of the models are dramatically different. Thus, the nonlinear host-parasitoid model provides a particularly useful means of testing the generality of the analytical results derived using the AR(1) model.

Simulations

I ran a series of simulations in which the local dynamics were governed by either the Ricker or the negative binomial model and examined the synchrony–DIS relationship for $H_{i,t}$. I again included an effect of environmental stochasticity, incorporated here by multiplying $H'_{i,t}$ in equation (4) or (5a) by $\exp(\varepsilon_{i,t})$. In time step t, a fraction $D_{H,i,t}$ of the host population (and, for host-parasitoid simulations, a fraction $D_{P,i,t}$ of the parasitoid population) in patch i emigrates after parasitism and reproduction occur. A fraction $I_{i,j}$ of these dispersers leaving i settle in patch j before the start of the next time step. With pre-dispersal densities given by equation (4) or (5), the complete model is then,

$$H_{i,t} = (1 - D_{H,i,t})\exp(\varepsilon_{i,t})H'_{i,t} + \sum_{\substack{j=1\\ j\neq i}}^{n} D_{H,j,t}I_{j,i}\exp(\varepsilon_{j,t})H'_{j,t} \tag{6a}$$

for all simulations, with

$$P_{i,t} = (1 - D_{P,i,t})P'_{i,t} + \sum_{\substack{j=1\\ j\neq i}}^{n} D_{P,j,t}I_{j,i}P'_{j,t} \tag{6b}$$

for host-parasitoid simulations.

As above, $\varepsilon_{i,t}$ has mean 0, variance σ^2_ε and spatial correlation ρ_ε. For the Ricker model, I also considered the possibility that dynamics were affected by both environmental and demographic stochasticity. For this,

$$H_{i,t} = (1 - D_{H,i,t})\exp(\varepsilon_{i,t} + \phi_{i,t})H'_{i,t} + \sum_{\substack{j=1\\ j\neq i}}^{n} D_{H,j,t}I_{j,i}\exp(\varepsilon_{j,t} + \phi_{j,t})H'_{j,t} \tag{7}$$

was used in place of equation (6a). The additional random variable $\phi_{i,t}$ represents demographic stochasticity and has mean 0, variance $\sigma^2_d/H_{i,t-1}$ and no spatial or temporal correlation. Demographic stochasticity could alternatively be incorporated by using equation (6a) to obtain an expected population density for each subpopulation, multiplying by a constant representing the carrying capacity to convert the local population density to an expected number of individuals, and then sampling about this expected number from a Poisson distribution to give the realised number of individuals. This is perhaps a more elegant method of representing demographic stochasticity, but it tends to make large metapopulations extremely resistant to global extinction in the presence of dispersal because the probability of sampling a realised population size of zero for each of many subpopulations simultaneously is quite small, even for subpopulations with rather low carrying capacities. To permit exploration of a model in which global extinction is a more meaningful threat, I show here only results using the formulation of demographic stochasticity shown in equation (7). With the exception of global extinction risk, equation (7) and the Poisson formulation of demographic stochasticity yield qualitatively identical results (K. Abbott, unpublished results).

The values of $D_{H,i,t}$ and $D_{P,i,t}$ were determined using a separate emigration model. I considered nine different emigration rules representing both density independent and -dependent dispersal of either or both species, and with or without dispersal mortality (Appendix S2). For all emigration rules, higher values of the parameter d always correspond to a greater tendency to disperse (Appendix S2). In combination with these emigration rules, I considered three different immigration models: global ($I_{i,j} = \frac{1}{n}$),

Table 1 Parameter combinations used in the simulations. For all parameter sets, d was varied between 0.05 and 0.5 at increments of 0.05 and $b = 1$, $\sigma_d^2 = 0$, 0.01 or 0.1, $v = 1.5$, $m = 2$, $\mu = 0.2$ and $h = 0.75$ times the equilibrium density of the dispersal-free model (the parameters f, v, m, μ and h are used in the models for dispersal; see Appendix S2). Abbreviations: BCs, boundary conditions; Refl, reflecting; Abs, absorbing; ||DE||, magnitude of the dominant eigenvalue for local dynamics in the absence of dispersal (values > 1 indicate that isolated subpopulations lack a stable equilibrium point)

Model	Ricker				Negative binomial			
Parameter set	(i)	(ii)	(iii)	(iv)	(v)	(vi)	(vii)	(viii)
BCs	Refl	Refl	Refl	Refl	Refl	Abs	Refl	Refl
λ	1.5	2.6	2.6	3	3	3	1.5	2
k	–	–	–	–	0.2	0.2	0.2	0.8
f	–	–	–	–	0.1	0.1	0.1	0.6
σ_ε^2	0.1	0.1	0.5	0.1	0.2	0.2	0.1	0.5
ρ_ε	0.1	0.3	0.1	0.1	0.3	0.3	0.05	0.1
||DE||	0.5	1.6	1.6	2	0.61	0.61	0.72	0.96

nearest-neighbor ($I_{i,j} = \frac{1}{4}$ for j adjacent to i; otherwise $I_{i,j} = 0$), or an intermediate rule whereby most individuals move short distances, but some may move farther (using a discretised exponential dispersal kernel; Appendix S2).

Spatial simulations were done on a 10 × 10 grid of subpopulations until global extinction or for 500 time steps, and I used log host densities from the final 50 time steps for analyses. The size of the grid was chosen based on Reeve's (1988) finding that, for a model similar to the host-parasitoid one used here, persistence initially increased with grid size, but reached an asymptote with fewer than 100 subpopulations. The simulations were done in pairs: one realisation with dispersal according to some combination of the above rules, and one realisation with no dispersal. Both members of the pair were subjected to the same sequence of random environmental variates, $\varepsilon_{i,t}$, and had the same initial conditions chosen randomly near the dispersal-free equilibrium of the model. The grid was assumed to have reflecting boundaries (with one exception; see Table 1), such that individuals dispersing off the grid were replaced by an equal number of immigrants from unmodeled populations assumed to exist around the grid.

To test the predictions of the AR(1) model, I varied d between 0.05 and 0.5, while holding the other parameters constant. The parameter b in the host-parasitoid model scales the equilibrium population densities, but has no effect on the model's dynamics, so for all simulations, $b = 1$. Other parameters were selected in combinations that covered a range of ecological situations; these combinations are summarised in Table 1 and were used in conjunction with the various dispersal rules. With each simulated scenario, 50 pairs of realisations (with and without dispersal) were generated for each value of d. The average level of synchrony and the average DIS were calculated for each d, and the correlation between these average values was used to describe the synchrony–DIS relationship across different dispersal rates.

For comparison with the AR(1) results, I measured stability in the simulations as the variance in log host density, with DIS equal to the ratio of the variance without dispersal to the variance with dispersal. Temporal variance is of course just one of many ways to characterise the stability of a population. Dispersal is known to affect local and global extinction risks (Roy *et al.* 2005), but it is unknown if the synchrony–DIS relationship will be the same when stability is measured in terms of extinction risk rather than by the variance in local density. To investigate this, I also recorded the frequency of local extinctions (the number of time steps with local density below an extinction threshold) as well as the global extinction rate (the inverse of the time until all local populations were simultaneously below the threshold). Subpopulations were considered extinct if the local density of either species dropped below 1/1000th of its dispersal-free equilibrium density. If a subpopulation persisted until the end of the simulation (500 time steps), its extinction time was assumed to be 501 time steps. This is obviously an underestimate, but these truncated cases showed the same general patterns as those where extinction occurred before 500 generations.

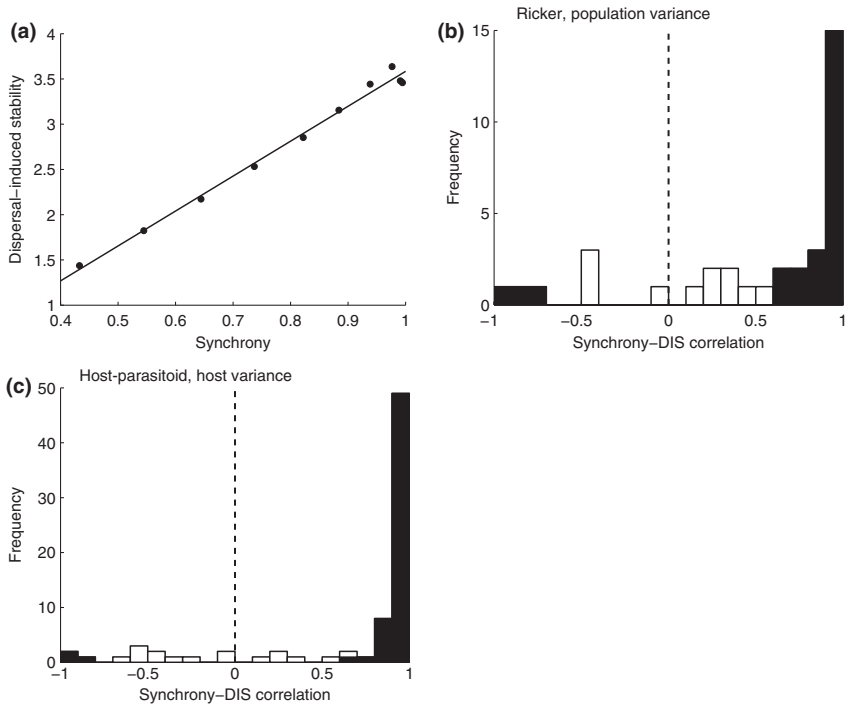

Figure 2 The relationship between synchrony and dispersal-induced stability that is generated by varying the dispersal rate, d. Synchrony was measured as the correlation in log host densities, and stability was measured as the variance in log host density. DIS is the ratio of this variance in the absence of dispersal to the variance with dispersal. (a) One representative example of a synchrony–DIS relationship. The dots show the means of 50 replicates for each value of d and the line is a linear regression fitted to these means. In this example, the negative binomial model is shown with parameter set (v), an immobile parasitoid and a host with global density-dependent dispersal ($m = 2$, $h = 1.65$ (= 0.75 times the dispersal-free host equilibrium)). (b, c) Summary of the synchrony–DIS relationships across a range of scenarios (Table 1 with $\sigma_d^2 = 0$ or 0.01 and additional assumptions as described in Appendix S2); (b) shows the Ricker model and (c) shows the negative binomial model. For each simulated scenario, a correlation coefficient between population synchrony and dispersal-induced stability was calculated based on log host densities. Significant ($P < 0.05$) synchrony–DIS correlations are shown in black, and non-significant correlations are shown in white.

Simulation results

For most simulations of the Ricker model and the negative binomial model, dispersal-induced stability (when measured as the ratio of host variance without and with dispersal) and host synchrony both typically increased with the dispersal parameter, *d*. The example shown in Fig. 2a is a good representative of the tight positive relationship between synchrony and this variance-based DIS seen in most of the simulations. Figure 2b,c shows histograms of correlations between DIS and synchrony from all the simulations, with significant ($P < 0.05$) correlations shown in black. Clearly, the majority of cases showed a very high positive correlation, in agreement with the positive relationship predicted by the AR(1) model. In a small minority of cases, dispersal was purely destabilising (DIS < 1) for every value of the dispersal parameter *d* and regardless of the level of synchrony. As explained in the literature review above, the main question motivating this study is the interplay between dispersal-induced stability and synchrony, and these examples of dispersal-induced instability do not speak to the issue at hand. Therefore, I show results only from scenarios in which dispersal was stabilising for at least one of the *d* values considered.

Although it was anticipated by the simple AR(1) model, it is nonetheless striking that so many of the nonlinear simulations reveal a positive synchrony–DIS relationship. This positive relationship was predicted based on a very simple model with density independent, global dispersal of one species. The simulation results show that the positive relationship is robust to many possible dispersal behaviours and to much more complex local dynamics. Close examinations of individual simulation results revealed that boundary conditions (i.e. whether dispersing individuals 'bounced' back or disappeared if they attempted to disperse off the edge of the grid), specific immigration rules, dispersal mortality and demographic stochasticity had no qualitative impact on these conclusions.

Interestingly, the synchrony–DIS relationship becomes strongly negative when DIS is measured in terms of either local or global extinction rather than by host variance (Fig. 3). That is, dispersal rates that result in the highest levels of synchrony also give the weakest protection against both local and global extinction. High synchrony has long been thought to increase at least global extinction risk (Allen *et al.* 1993; Heino *et al.* 1997; Matter 2001). Dispersal is also known to affect some measures of stability differently (Reeve 1988, 1990), and more variable subpopulations may, counter-intuitively, actually be less extinction prone when embedded in a metapopulation (Legendre *et al.* 2008). Therefore, the contrast between Figs 2 and 3 is in many ways expected. Results in the next section, however, reveal that the relationship between synchrony and extinction-based measures of DIS are not invariably negative, nor are they always at odds with the variance-based DIS measure.

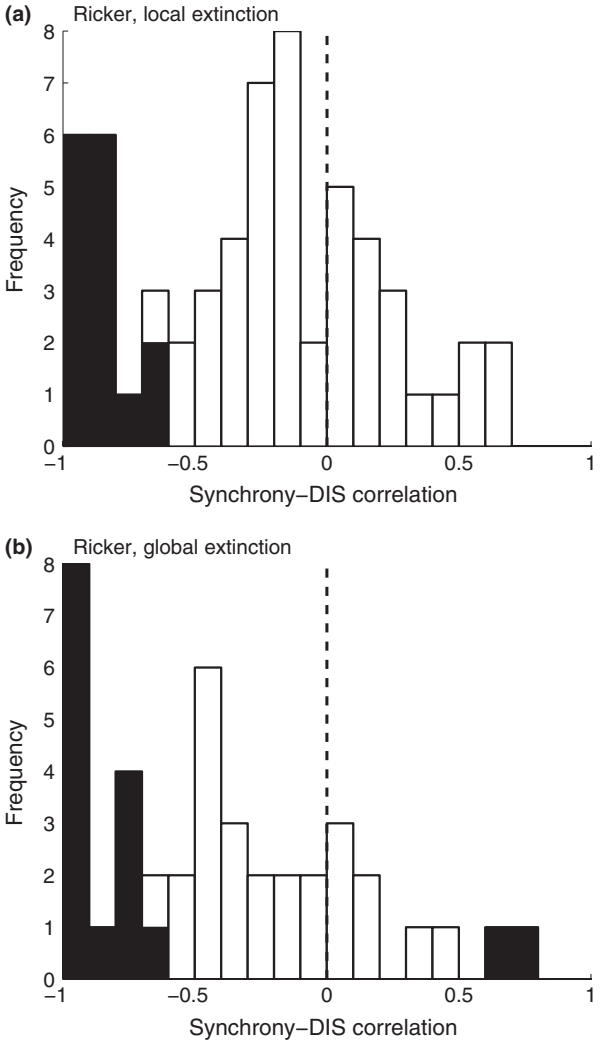

Figure 3 The relationship between synchrony and dispersal-induced stability that is generated by varying the dispersal rate, *d*. Synchrony was measured as the correlation in log host densities, and stability was measured by (a) the frequency of local extinctions or (b) the global extinction rate. In both cases, DIS is the ratio of stability in the absence of dispersal to stability with dispersal. Histograms show the synchrony–DIS relationships for the Ricker model across a range of scenarios (Table 1 with $\sigma_d^2 = 0.01$ or 0.1 and additional assumptions as described in Appendix S2). Significant ($P < 0.05$) synchrony–DIS correlations are shown in black and non-significant correlations are shown in white.

OTHER FACTORS THAT AFFECT BOTH SYNCHRONY AND DISPERSAL-INDUCED STABILITY

The positive synchrony–DIS relationships shown in Figs 1 and 2 appear to contradict past results suggesting that synchrony counteracts dispersal-induced stability. This is actually not as surprising as it may at first seem, because this study has considered only models in which stochasticity prevents perfect synchrony as in natural populations. In contrast, the perception that the synchrony–DIS relationship should be negative has been heavily influenced by the lack of DIS in perfectly synchronous populations. One study that did consider ecologically relevant levels of synchrony also reported a negative synchrony–DIS relationship (Murdoch *et al.* 1992, who measured DIS as the change caused by dispersal in the dominant eigenvalue of the within-subpopulation model), in contrast to the positive relationship shown here for some forms of DIS. Recall, though, that the synchrony–DIS relationship is generated when some aspect of the model is changed. Earlier, I changed the dispersal rate parameter, *d*, whereas Murdoch *et al.* (1992) varied either the magnitude of fixed spatial differences in the local model's parameters or the strength of density dependence in dispersal. This comparison suggests that although some factors can simultaneously promote both synchrony and DIS (Figs 1 and 2), others that promote synchrony inhibit DIS (Murdoch *et al.* 1992). In other words, the synchrony–DIS relationship might change not just

for different definitions of stability (as for Fig. 2 vs. Fig. 3) but also for different underlying causes of synchrony and dispersal-induced stability.

Like dispersal, correlated environmental perturbations can bring distinct subpopulations into synchrony (Moran 1953; Hanski & Woiwod 1993; Grenfell *et al.* 1998; Lande *et al.* 1999). Although uncorrelated environmental variability can increases metapopulation persistence by inhibiting synchrony (Crowley 1981; Comins *et al.* 1992), real subpopulations that are close enough to be linked by dispersal may also experience environmental perturbations that are similar. A metapopulation's ability to increase when rare can be reduced by spatial correlations in environmental fluctuations (Schreiber 2010), and global extinction risk can increase with environmental correlation due to increased synchrony (Harrison & Quinn 1989; Heino *et al.* 1997; Palmqvist & Lundberg 1998; Engen *et al.* 2002). If environmental correlations persist over large enough spatial scales, DIS can be lost (Crowley 1981; Hassell *et al.* 1993). These results suggest, and the AR(1) confirms (Appendix S1) that the synchrony–DIS relationship that emerges from changes in environmental correlation, ρ_ε, should be negative. To test this relationship using simulations, I held d constant at 0.1 or 0.3 and varied ρ_ε between 0.1 and 0.9. Figure 4 shows a strong negative synchrony–DIS relationship in the majority of simulations and for all measures of stability considered. This means that environmental correlations that promote synchrony weaken dispersal-induced stability.

Another well-studied contributor to population synchrony is the dynamical behaviour of subpopulations in the absence of dispersal. Weakly regulated subpopulations are sometimes more readily synchronised by dispersal than ones in which strong regulation quickly returns densities to equilibrium following perturbations (Reeve 1990; Lande *et al.* 1999; Ripa 2000; Engen *et al.* 2002). Taylor (1998) indeed found that populations with more dynamically-unstable local dynamics (meaning those that lack the tendency to return to an equilibrium following disturbance) were at higher risk of global extinction, due apparently to the combination of higher synchrony and the inherently higher risk of local extinction that is associated with dynamical instability. In contrast to these examples, however, there is a body of theory showing that reduced dynamical stability can actually result in lower synchrony. Specifically, cyclic subpopulations are particularly prone to dispersal-driven synchronisation, whereas some chaotic subpopulations strongly resist synchrony (Allen *et al.* 1993; Heino *et al.* 1997; Jansen 1999; Bjørnstad 2000; Earn *et al.* 2000; but see Blasius *et al.* 1999). Furthermore, dispersal can actually change the dynamical stability of subpopulations in some models (Ives 1992; Murdoch *et al.* 1992), perhaps then changing their propensity to become synchronous. The actual relationship between DIS and synchrony that results from changes in local dynamics therefore cannot be readily resolved from existing literature. The AR(1) model predicts that changing a, the absolute value of which determines local dynamical stability, causes synchrony and dispersal-induced stability to increase together (Appendix S1). To examine the role of dynamical stability in the simulations, I varied k between 0.1 and 0.9 (negative binomial simulations) or λ between 1.5 and 3 (Ricker simulations), with d and ρ_ε held constant ($d = 0.1$ or 0.3 and ρ_ε as in Table 1). Figure 5 shows strong support for the predicted positive relationship, regardless of the model or measure of stability employed.

DISCUSSION

Perhaps the most basic question in spatial ecology is, what happens when populations are linked by dispersal? That spatial ecology is such a rich and exciting field is due at least in part to the fact that this question has so many answers. Two such answers have received a great deal of attention: First, dispersal can bring populations into synchrony. Second, populations may become more stable when they are linked by dispersal. Herein, I have highlighted some of the ways

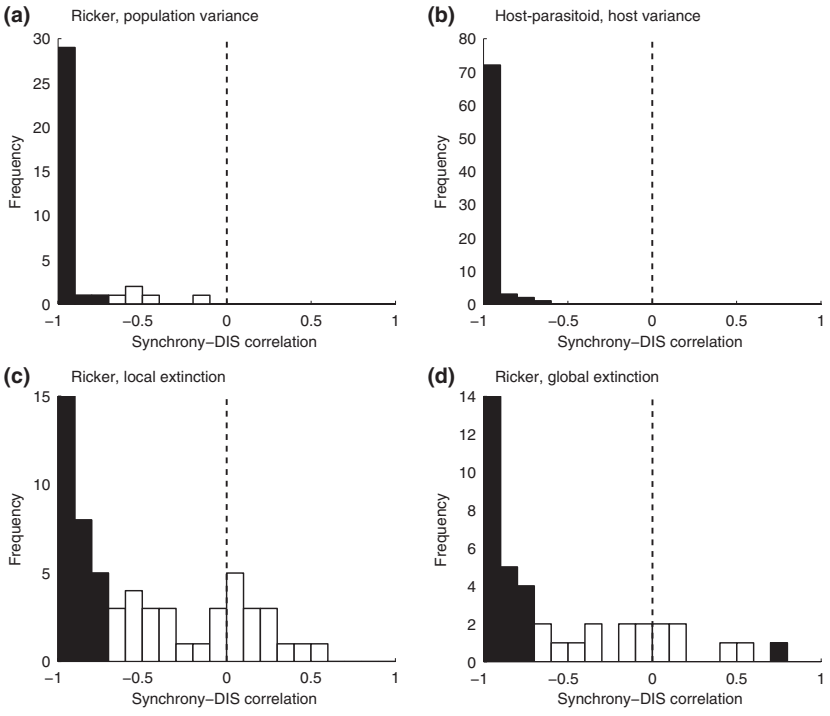

Figure 4 The relationship between synchrony and dispersal-induced stability that is generated by varying the strength of environmental correlations, ρ_ε. Synchrony was measured as the correlation in log host densities, and stability was measured by (a, b) variance in host density, (c) the frequency of local extinctions or (d) the global extinction rate. In all cases, DIS is the ratio of stability in the absence of dispersal to stability with dispersal. Histograms show the synchrony–DIS relationships for (a,c–d) the Ricker model and (b) the negative binomial model across a range of scenarios (see Table 1 and Appendix S2). Significant ($P < 0.05$) synchrony–DIS correlations are shown in black and non-significant correlations are shown in white. The relationship is usually negative, as predicted by the AR(1) model.

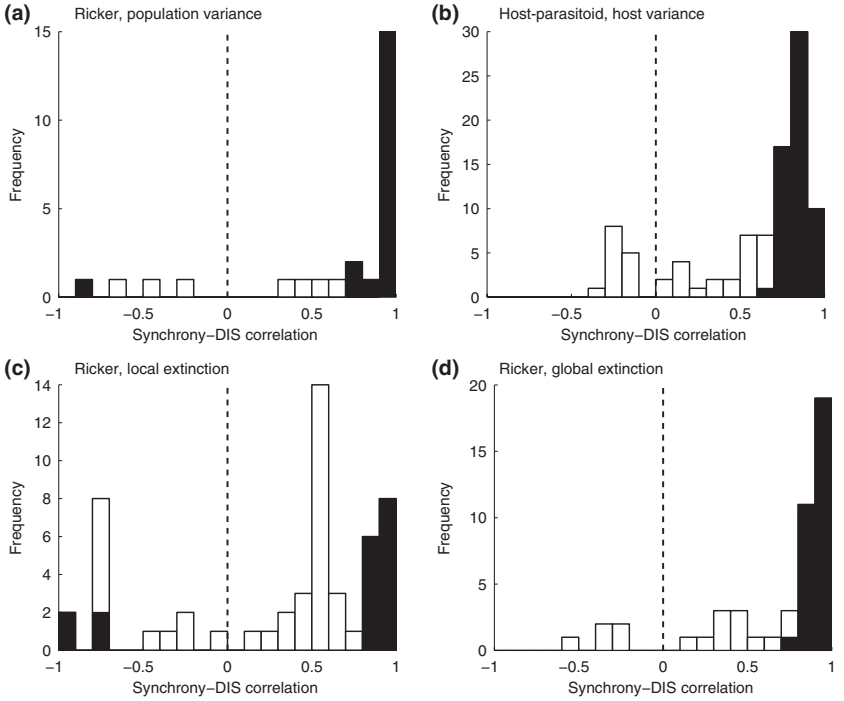

Figure 5 The relationship between synchrony and dispersal-induced stability that is generated by varying the local dynamics, determined by k and λ. Synchrony was measured as the correlation in log host densities, and stability was measured by (a, b) variance in host density, (c) the frequency of local extinctions or (d) the global extinction rate. In all cases, DIS is the ratio of stability in the absence of dispersal to stability with dispersal. Histograms show the synchrony–DIS relationships for (a,c-d) the Ricker model and (b) the negative binomial model across a range of scenarios (see Table 1 and Appendix S2). Significant ($P < 0.05$) synchrony–DIS correlations are shown in black and non-significant correlations are shown in white. The relationship is usually positive, as predicted by the AR(1) model.

in which these two effects interact with one another. In particular, I have shown that dispersal rates that promote synchrony can simultaneously promote some forms of DIS, that synchrony can have the same or opposite relationships with different forms of DIS, and that different mechanisms can produce different synchrony–DIS relationships.

So what, really, is the effect of synchrony on dispersal-induced stability? The work presented here shows that synchrony and DIS can either be positively or negatively correlated. Positive correlations like those shown in Fig. 2 could have two different explanations. One possibility is that factors that promote synchrony simultaneously promote DIS. We see evidence for this in the AR(1) and nonlinear models: amounts of dispersal (and strengths of local regulation) that promote dispersal-induced stability can also promote synchrony, leading to a positive indirect synchrony–DIS relationship (Figs 1, 2 and 5). A second possible explanation for positive synchrony–DIS correlations is that synchrony per se promotes dispersal-induced stability. From previous DIS studies, we would actually expect the opposite. Interestingly, it appears as though this expected negative relationship also exists in the simulations presented here. Figure 6 shows the results from a single set of model assumptions, simulated 50 times to give the 50 points that appear in the figure. Any variation among the 50 points is due entirely to randomness rather than to differences in the deterministic drivers of synchrony and DIS. The clear negative relationship shown in Fig. 6 example is representative of the other simulated scenarios as well, and it suggests that synchrony per se *is* indeed destabilising. However, the results in Figs 1, 2 and 5 make it clear that the indirect positive synchrony–DIS relationship, due to common factors promoting both processes, can swamp the direct negative synchrony–DIS relationship, resulting in a strong positive correlation.

The positive synchrony–DIS relationship is of course not universal. When we look across a range of different environmental correlations, for instance, no such positive relationship emerges (Fig. 4). This is

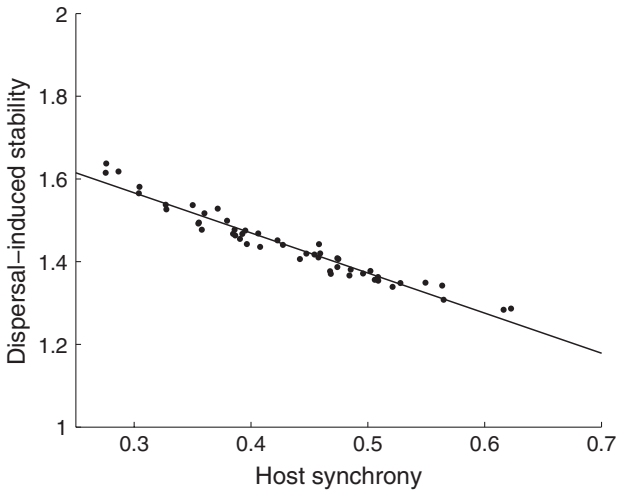

Figure 6 An example showing variation among replicate simulations. The dots, shown with a regression line, are from 50 realisations of the negative binomial model under parameter set (v) and global density independent prey dispersal ($d = 0.1$). Stability is measured as host variance, as in Fig. 2. (Note, each dot in Fig. 2a represents the average of 50 such realisations for a given value of d.)

because, although the strength of the environmental spatial correlation (ρ_ε) is important for determining population synchrony, it is instead the temporal variance in the environment (σ^2_ε) that directly affects stability. As a result, environmental conditions that promote synchrony need not also promote DIS and the indirect positive synchrony–DIS relationship is not present. In this case, therefore, the net relationship between synchrony and dispersal-induced stability is strongly negative. A negative net synchrony–DIS relationship also emerges when we look across dispersal rates, but define stability in terms of either local or global extinction (Fig. 3), suggesting that

dispersal rates giving high synchrony decrease population variance the most, but decrease extinction risk the least (see also Reeve 1988, 1990; Legendre *et al.* 2008).

The results presented here have focused on models with bounded dynamics. In the absence of dispersal, the dynamics of the negative binomial model with $k > 1$ are dramatically different than when $k < 1$: when isolated, these subpopulations exhibit diverging oscillations and eventual deterministic extinction. When linked by dispersal, some such populations will be saved from extinction via dispersal-induced stability, but others will not. It would be interesting to study the synchrony–DIS relationship in these highly unstable populations as well, but methods different from those used here would be needed. Populations that are on their way to deterministic extinction are non-stationary and for these, statistics like the variance and correlation of population densities are time-dependent and thus not useful descriptors of stability and synchrony, respectively. Stability could of course be measured by extinction rates, but an entirely different measure of synchrony would be necessary. For subpopulations experiencing frequent local extinctions and recolonisations, Hudgens (2007) suggested measuring synchrony as the correlation in the timing of local extinctions. Preliminary analyses of the Ricker model and the unstable ($k > 1$) host-parasitoid model suggest that although these two measures of synchrony – correlation in population densities and correlation in local extinction times – tend to be positively correlated, they can behave very differently and result in quite different synchrony–DIS relationships. The same simulations, for instance, can show that global extinction-based DIS is negatively related to correlation in population density and positively related to correlation in local extinction times. Additional research is needed to identify the best ways to compare synchrony–DIS relationships across some very different types of models.

We all know that ecological processes occur in both time and space, but the complexity of ecological systems often forces us to study just the spatial dimension or the just temporal dimension of the patterns we see. This approach is indispensable, as it is often impossible to gain meaningful insight into any ecological problem without first breaking it down into smaller, focused questions. However, once the separate pieces are sufficiently well-studied such that we have a good understanding of how they work, it serves us well to return to the bigger picture. Herein, I have provided a start at synthesising spatial and temporal effects of dispersal (synchrony and stability, respectively) that are reasonably well-understood in isolation. Such efforts to bridge separate, but related lines of inquiry should ultimately help us arrive at new theoretical insights. For multi-species models, there is significant recent interest in understanding the consequences of interspecific synchrony; that is, correlations in the dynamics of competitors or of a consumer and its resource (e.g. Ripa & Ives 2003; Vasseur & Fox 2007; Gouhier *et al.* 2010). It would likely be informative to build on the ideas presented here to address community-scale questions.

This synthesis brings to light some specific ideas that warrant attention. The emphasis in the DIS literature on the important destabilising influence of perfect synchrony has overshadowed a consideration of any positive indirect synchrony–DIS relationships. The results in this study demonstrate that under a broad range of circumstances, the indirect relationship is actually the main determinant of how synchrony and stability coincide. Understanding the multiple effects of altering dispersal rates is much more than an abstract theoretical question; it is indeed an immensely important endeavor at a time when human activities can dramatically alter natural dispersal rates. Predicting and ameliorating the effects of human activities that interfere with natural dispersal requires a sophisticated understanding of the total impact of dispersal on populations. Some have argued that fragmentation might reduce regional extinction risk by inhibiting synchrony and thus reducing the potential for all local subpopulations to become extinct at once (e.g. Heino *et al.* 1997; Earn *et al.* 2000; Johst & Schöps 2003). On the other hand, one could argue that fragmentation should elevate extinction risk by interfering with dispersal-driven stabilising mechanisms (e.g. Reeve 1988; Hassell *et al.* 1991b; Adler 1993; Engen *et al.* 2002). Clearly, we need to examine both the stabilising and synchronising effects of dispersal, and the way those effects interact to identify which human activities pose the greatest threat to species persistence.

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SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article:

Appendix S1 Analysis of the AR(1) model.
Appendix S2 Methods for modelling dispersal in simulations.

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