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Population synchrony in small-world networks

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Network topography ranges from regular graphs (linkage between nearest neighbours only) via small-world graphs (some random connections between nodes) to completely random graphs. Small-world linkage is seen as a revolutionary architecture for a wide range of social, physical and biological networks, and has been shown to increase synchrony between oscillating subunits. We study small-world topographies in a novel context: dispersal linkage between spatially structured populations across a range of population models. Regular dispersal between population patches interacting with density-dependent renewal provides one ecological explanation for the large-scale synchrony seen in the temporal fluctuations of many species, for example, lynx populations in North America, voles in Fennoscandia and grouse in the UK. Introducing a small-world dispersal kernel leads to a clear reduction in synchrony with both increasing dispersal rate and small-world dispersal probability across a variety of biological scenarios. Synchrony is also reduced when populations are affected by globally correlated noise. We discuss ecological implications of small-world dispersal in the frame of spatial synchrony in population fluctuations.

Keywords: small-world; dispersal; synchrony; Moran; network

1. INTRODUCTION

Research on the long-term dynamics of spatially structured populations has revealed that population densities of a given species tend to fluctuate in synchrony over vast geographical areas (Ranta *et al.* 1995, 1997, 2006; Leibold *et al.* 2004). The flagship example comes from the continental scale patterns of synchrony in population fluctuations of the Canadian lynx (Elton 1924; Lindström *et al.* 2001). Presently, the number of animal taxa displaying large-scale synchronous fluctuations is several hundreds, including insects, crustaceans, fishes, mammals and birds (Ranta *et al.* 1999), and the number is increasing (Paradis *et al.* 2000; Leibold *et al.* 2004). Much of these data share a common feature: the degree of synchrony goes down with increasing distance between populations (Ranta *et al.* 1995, 1997, 2006).

The redistribution of individuals among local populations is one of the explanations provided for the emergence of synchronicity (Ranta *et al.* 1995, 2006; Kendall *et al.* 2000). It has repeatedly been shown that only a small fraction of individuals redistributing between breeding seasons among local population sites is capable of synchronizing population fluctuations that follow a variety of renewal processes (Ranta *et al.* 2006). Other explanations for synchrony include interactions with natural enemies (Ydenberg 1987) and external perturbations (Ranta *et al.* 1995; Greenman & Benton 2001), that is, climatic forcing, also known as the Moran effect (Moran 1953). All these mechanisms work by disturbing the local density-dependent feedback systems in the population renewal process. The major explanations of population synchronicity merge in one respect: the synchronizing agent works at the scale of the spatial dimension. Dispersal is distance dependent (it is less risky to travel short than long distances); if the external forcing is climate, nearby localities are more likely to share a common climate than more distant ones; and natural enemies also tend to be distance limited in their dispersal.

Against this background, it is natural to suggest that spatially structured biological populations can be viewed as coupled network systems. This analogy becomes even more apparent when the within-patch density-dependent population renewal process is equated with an oscillator. As with physicists and communication researchers, ecologists are placing more emphasis on the study of networks of coupled oscillators (Ranta *et al.* 2006). A revolutionary breakthrough in the research of network architecture was the innovation and rediscovery of 'small-world' networks (Karinthy 1929; Milgram 1967; Watts & Strogatz 1998; Buchanan 2002; Strogatz 2003; Watts 2003).

Regular networks consist of interactions between neighbouring oscillators, which are then 'rewired' with some probability p between randomly selected nodes in the network. When $p=0$ the network is regular, when $p=1$ the network is completely random, while the small-world network is somewhere $0<p<1$. The merits of small-world network architecture in various contexts are discussed in detail elsewhere (Buchanan 2002; Strogatz 2003; Watts 2003), however, there are dissenting voices as to the ubiquity of such networks (Kleinfeld 2002). There are examples of how such architecture can impact on biological systems, for example, epidemiological networks (Boots & Sasaki 1999; Pastor-Satorras & Vespignani 2001) or food web persistence (Sinha 2005; Sinha & Sinha 2005).

There are important ecological and evolutionary implications of small-world networks. As a regular network (dispersal) pattern is changed to incorporate increasingly irregular links between nodes (population patches), the patterns in important biological processes may be expected to change. For example, empirical studies on plant and animal dispersal repeatedly observe

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that the majority of the dispersing individuals move only a short distance, while a few manage to move over longer distances (Dingle 1996; Turchin 1998). The dynamics of infectious diseases is another field where the small-world network framework is likely to have important implications (Boots & Sasaki 1999; Pastor-Satorras & Vespignani 2001; Saramaki & Kaski 2005; Verdasca *et al.* 2005), as infectious diseases will eventually die out in small populations unless the pool of infected individuals is refreshed now and then. The majority of research into small-world networks has so far been in the evolution of social groups (Palla *et al.* 2007). Furthermore, population genetics has stressed that it takes only one immigrating individual per generation to dilute the effects of random genetic drift and adaptation to local conditions (e.g. Templeton 2006).

Here, we demonstrate—focusing on the temporal match (synchrony) in fluctuations of populations—for the first time that an increase in the probability of small-world network links can lead directly to a reduction in the synchrony of between-patch population fluctuations. In small-world networks, increasing dispersal rate also leads to a reduction in synchrony, in contrast to regular dispersal kernels. It is important to note that the small-world dispersal networks we address are not distant dependent. The probability that an emigrant undertaking a small-world dispersal event will arrive at any given patch in the system is drawn at random from a uniform distribution. Our results hold under a range of different commonly used population renewal kernels and are in direct contrast to those from other studies of linked oscillators which do not show density-dependent renewal (e.g. Hong *et al.* 2002; Guan *et al.* 2006).

2. MATERIAL AND METHODS

We develop the small-world concept to model the interaction of local populations—semi-independent nodes obeying the same density-dependent structure in their renewal—coupled to each other by dispersing individuals. Renewal occurs at the local node level, with a given fraction of individuals in each local subpopulation redistributing around the network. The majority of dispersing individuals in our model move to neighbouring nodes in a regular fashion, but there is a probability p that some will arrive at any node within the network selected at random. Thus, we assess the impact of small-world architecture on a network of populations with fluctuating density, linked through dispersal of individuals between nodes, the Moran effect, or both.

All the results reported here were simulated in a network of $N=100$ nodes arranged in a loop (i.e. a linear array of nodes with periodic boundaries). The renewal process in each patch was initiated with random numbers (independent and identically distributed) approximately $\pm 50\%$ of the renewal kernel-specific long-term average. In each run, the process was iterated over 1000 steps. The final 100 time steps were used to calculate the synchrony in the fluctuations of population density between all pairs of patches (using cross correlation with lag zero; Chatfield 1996). Normalizing time series to have mean 0 and s.d. 1 did not qualitatively change our results. We experimented with varying number of nodes N and iteration numbers but have observed our results to be qualitatively robust. For each value of p (the probability of non-regular or the small-world dispersal), the simulations were replicated 100 times, averages of which are reported here. Population renewal followed a discrete time function that updated population density X over consecutive time steps t in each node of the network i, as

$$X_{i,t+1} = (1-m)f(X_{i,t},\varepsilon_t) + \sum_{j=i-n,j\neq i}^{i+n} \frac{mf(X_{j,t},\varepsilon_t)}{2n}, \tag{2.1}$$

where m (if not otherwise stated $m=0.1$) is the fraction of individuals dispersing; n is the neighbourhood distance (number of nodes) that dispersers move in either direction from each focal node i when obeying regular dispersal (we used $n=1$ for results presented here unless stated otherwise); and ε_t is the globally correlated environmental noise, with limits $[1\pm w]$. Here the noise time series (ε_t) are generated as described by Ripa & Lundberg (1996).

We present results from the following three specific forms of the renewal function. First, we updated populations with a first-order linear model

$$f(X_{i,t}) = \varepsilon_t(d + aX_{i,t-1}), \tag{2.2}$$

where d (here 100) and a (0.7) are constants, where a determines the form of the density-dependent feedback. The second renewal kernel incorporates delayed density dependence using a nonlinear second-order function. This allowed us to generate populations that fluctuated with a characteristic period over time as

$$f(X_{i,t}) = \varepsilon_t X_{i,t-1}\exp(r + b_1X_{i,t-1} + b_2X_{i,t-2}). \tag{2.3}$$

Here r is intrinsic growth rate; and b_1 and b_2 are parameters relating to the strength of direct and delayed density dependence, respectively. These parameters were varied to produce fluctuations with periods of four ($r=2.01$, $b_1=-0.005$, $b_2=-0.005$), six ($r=1.01$, $b_1=-0.0001$, $b_2=-0.006$) or nine ($r=0.47$, $b_1=0.0027$, $b_2=-0.005$) time steps (Kaitala *et al.* 1996). Equation (2.2) is a first-order linear autoregressive model, while equation (2.3) is better known as the delayed Ricker function (Ranta *et al.* 2006). We also examined a spatially structured host–parasitoid model (Beddington *et al.* 1975), where X is the host density and P is the parasitoid density in a given patch, which are updated as follows:

$$\begin{aligned} f(X_{i,t}) &= X_{i,t-1}\exp\left[r\left(1-\frac{X_{i,t-1}}{K}\right) - c_1P_{i,t-1}\right], \\ f(P_{i,t}) &= c_2P_{i,t-1}[1-\exp(-c_1X_{i,t-1})]. \end{aligned} \tag{2.4}$$

In this case, parameter r (here, 1) is the host's intrinsic growth rate; K (100) is the host equilibrium density in the absence of parasitoids; and c_1 (0.035) and c_2 (1) are the parasitoid searching efficiency and conversion rates, respectively. Both hosts and parasitoids followed the same dispersal kernel between population patches. We also analysed the case where hosts dispersed between patches, but parasitoids did not. This did not lead to any qualitative change in the results.

To produce simulation conditions comparable with other research fields (e.g. from physics), in one set of simulations of equation (2.3), we allowed slight variation in the cycle period length in each of the oscillators (patches) in the network. To achieve this, we selected r to yield 9-year periodicity, and used this value as the mean of a Gaussian distribution with a standard deviation of 0.005. The realized r-value for each node in the network was then drawn randomly and independently from this distribution. In ecological terms, this can be considered as habitat heterogeneity, with conditions in each patch differing slightly from all others in

the system, feeding back through variation in the local intrinsic growth rate.

The novel aspect of the work presented here arises in the dispersal kernel employed to redistribute migrating individuals. Dispersers leave their natal patch (node), and are redistributed in a given manner among the N patches in the network. The important aspect is that when $p=0$, dispersing individuals from a focal patch always arrived in the nearest neighbouring patches to the left and right. With small-world dispersal, $0<p<1$, at each time step we drew a uniformly distributed random number θ between 0 and 1. If $\theta<p$, dispersing individuals moved to any patch in the network, selected at random from a uniform discrete distribution with limits [1,N]. This process occurred independently for those dispersers moving to patches on the left and right of each focal node. It is possible that a network created from such a dispersal rule will be either fixed (network wiring was done before population renewal began) or flexible (the target patches for each focal patch were updated each generation when $\theta<p$) over time. These scenarios represent three different but biologically plausible network wiring methods.

Therefore, we examined how variation in the probability of small-world dispersal (p) and/or dispersal rate (m) affected population synchrony in three different, commonly used population models, showing increases in either dispersal parameter led to a decrease in between-patch population synchrony under a range of important biological scenarios.

3. RESULTS

The major findings from our analyses are framed in terms of the degree of synchrony in population fluctuations averaged across the N nodes of the network. Initially, our results echo numerous previous findings (Ranta *et al.* 1995, 1999, 2006; Kendall *et al.* 2000) that with regular dispersal ($p=0$) synchrony in population fluctuations is easily achievable. More interestingly, however, we show that introducing small-world dispersal ($0<p<1$) can lead to a considerable loss in the degree of synchrony, even with relatively small values of p (figure 1).

In order to assess the degree of synchrony between different network nodes, it is necessary to study populations whose dynamics are not in a stable equilibrium state. Following Moran's (1953) example, we first used a simple, discrete-time, first-order linear function to represent population growth over time, which was forced by globally correlated noise. Increasing the probability of small-world dispersal events leads directly to a reduction in the synchrony of fluctuations in population density (figure 1*a*), with synchrony being completely lost under sufficiently high levels of p. The generality of this result is further emphasized through the analyses of different functions adopted for the population renewal process. By allowing small-world dispersal in populations that cycle with different characteristic periods, we can easily demonstrate that increasing p leads to a reduction in synchrony (figure 1*b*,*c*). Increasing p can desynchronize populations that otherwise show a high degree of synchrony in the absence of global forcing (figure 1*b*). Furthermore, gradual loss of synchrony with increasing p remains even with the influence of the globally correlated noise (figure 1*c*). By varying both the migration rate (m) and p, we show that with a small-world dispersal kernel, increasing either parameter leads to a reduction in synchronicity (figure 1*d*). We find that the loss of synchrony against increasing p is robust to a wide variety of ecological conditions and network configurations, including small-world networks that are either fixed over time, or vary in configuration between generations and host–parasitoid interactions (figures 1*f* and 2).

With a low probability of small-world dispersal, populations maintain a relatively high degree of synchrony, with a non-zero dispersal rate. With increasing dispersal rates, increasing p leads again to a loss of synchrony, with the degree of synchrony decreasing faster as dispersal rate increases (figures 1*d* and 3). This occurs as the synchronizing effects that arise from regular dispersal between adjacent patches are disrupted. Even with very low rates of dispersal between populations, a group of populations with cyclic dynamics can easily become synchronized (Rosenblum 1996). By introducing a probability that some dispersers will not arrive in an adjacent patch, but some other patch in the network, we have effectively disrupted the synchronizing process. It was previously suggested that increasing the potential for connection between different patches in the environment would lead to an increase in global synchrony (Watts & Strogatz 1998). However, due to the random and discrete nature of migration events under our ecological small-world dispersal rule, this is not the case.

Differing population renewal functions are likely to influence the degree of population synchrony differently. Hence, we studied three commonly used functions to model population renewal (as well as the different cycle period lengths produced by one of these functions) and find our results (figures 1 and 2) to be robust to the presence and absence of noise and to the kind of different renewal functions tested. It is interesting to observe that the Moran effect does not prevent small-world dispersal reducing the amount of synchrony between populations, although the colour (autocorrelation structure) of noise does affect the magnitude of the change in synchrony. Furthermore, comparison of results with small-world networks that are either fixed over time, or those that include heterogeneity in the population growth rate among patches or trophic interactions produce the same qualitative results (figures 1*e*,*f* and 2). Thus, dispersal across small-world networks leads to a loss of synchrony across a wide range of important ecological conditions. The loss and gain of synchrony corresponding to switching a small-world dispersal kernel on and off is illustrated clearly in figure 3.

4. DISCUSSION

The relative importance of dispersal and correlated environmental forcing on synchrony in the dynamics of spatially structured populations has received a considerable amount of research effort (Kendall *et al.* 2000; Paradis *et al.* 2000; Lindström *et al.* 2001; Leibold *et al.* 2004; Ranta *et al.* 2006). As yet, no clear consensus has been reached on the precise mechanism leading to synchrony (Blasius *et al.* 1999; Lundberg *et al.* 2000). Different authors have promoted (Schwartz *et al.* 2002) and dismissed (Peltonen *et al.* 2002; Post & Forchhammer 2002; Rueness *et al.* 2003) the potential for dispersal to act as a synchronizing agent. Different species or assemblages are likely to be influenced by various synchronizing agents

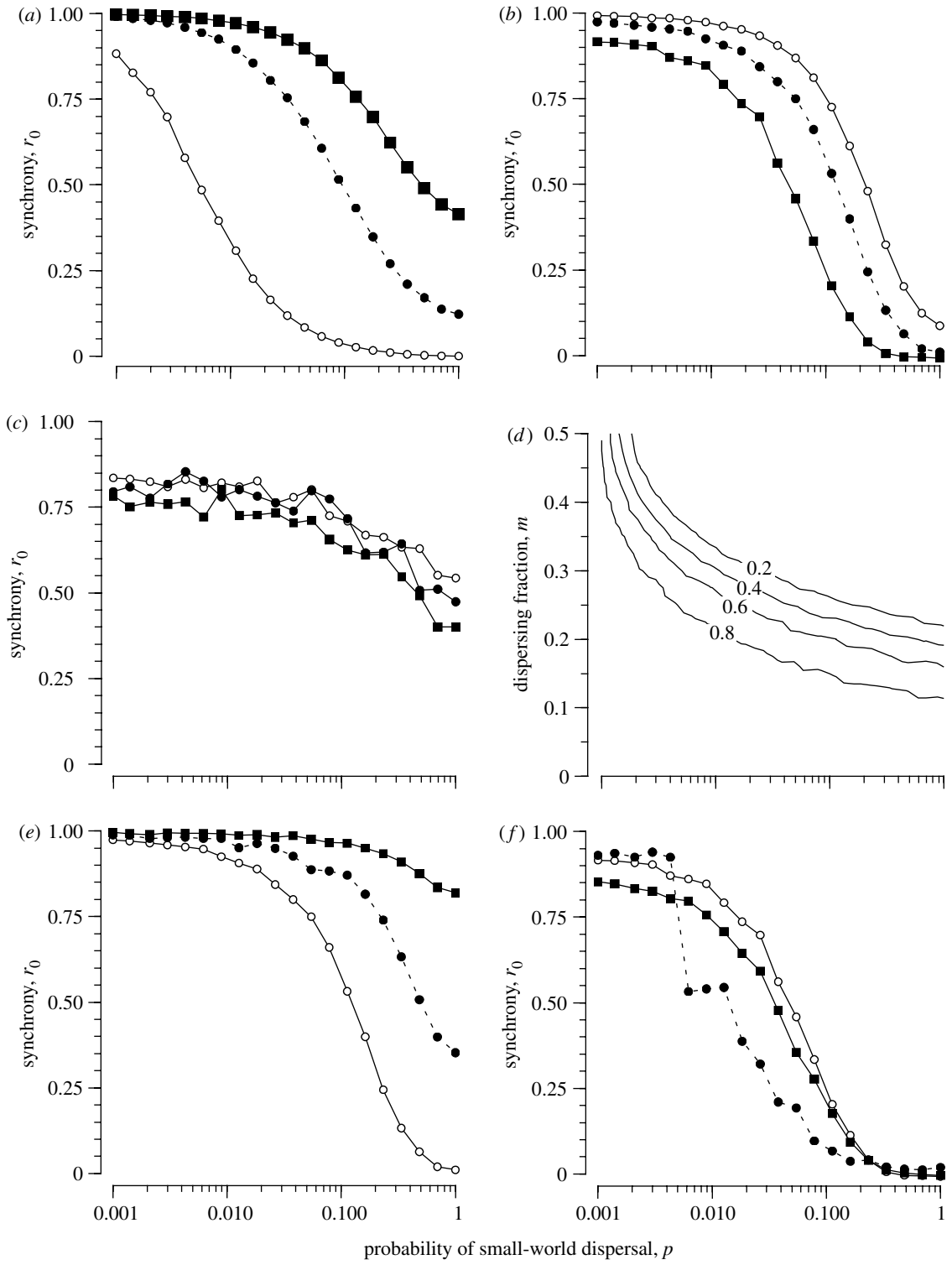

Figure 1. Small-world dispersal leads to a reduction in synchrony in a number of biologically plausible scenarios. (*a*) First order linear with noise: following the classic work by Moran (1953), we studied a population governed by a linear autoregressive equation, with environmental forcing of different strengths ($1 \pm w$; black squares, $w = 0.1$; black circles, $w = 0.05$; open circles, $w = 0.01$) and $m = 0.15$. In all cases (despite the environmental forcing), increasing the probability of small-world dispersal p leads to a reduction in synchrony in population fluctuations. This is true in cyclical populations of different period lengths governed by the delayed Ricker function (equation (2.3)) with environmental noise either (*b*) absent (delayed Ricker) or (*c*) present (delayed Ricker with noise). Cycle period length: open circles, four; filled circles, six; filled squares, nine. (*d*) Delayed Ricker, period six, synchrony contours: interaction between the dispersing fraction m and small-world dispersal. Increasing either m or p (when both parameters are non-zero) can lead to a considerable reduction in the synchrony of between population fluctuations. (*e*) Delayed Ricker, period six: in a network with $N = 100$ nodes, increasing the number of neighbours linked by dispersal increases the level of synchrony but the effect of reduced synchrony with increasing p is still clearly visible. Neighbourhood size: open circles, one; filled circles, two; filled squares, five. (*f*) Delayed Ricker, period nine: finally, we explored the significance of flexible network wiring, as well as that of spatial heterogeneity in population growth rate on synchrony. Configuration: open circles, random; filled circles, fixed; filled squares, stochastic. The conclusion is that the loss of synchrony against increasing small-world dispersal is robust to a wide variety of ecological conditions and network configurations.

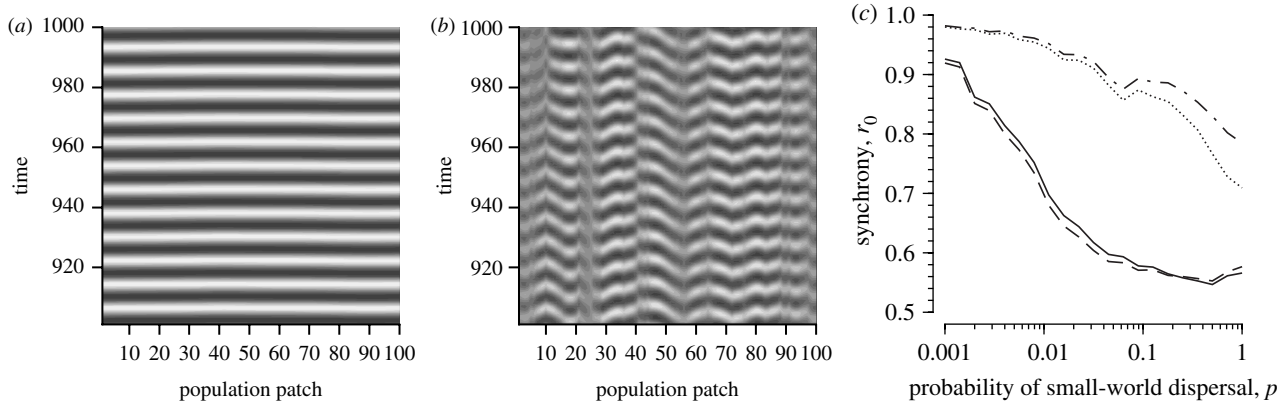

Figure 2. Small-world dispersal in a cyclical host–parasitoid system. (*a*) In a spatially structured population where each patch is linked to its two nearest neighbours through dispersal ($m=0.15$), the host (and parasitoid) populations synchronize in their fluctuations over time (dark shading, low population density and light shading, high population density). (*b*) Introducing a small-world dispersal pattern with $p=0.1$ leads to the breakdown of synchrony. (*c*) Increasing the probability of small-world dispersal leads to a reduction in synchrony for both the hosts (solid line) and parasitoids (dashed line) with flexible network wiring or with a temporally fixed small-world network (hosts, dotted line; parasitoids, dash-dot line). Other parameter values used: $r=c_2=1$, $K=100$, $c_1=0.035$.

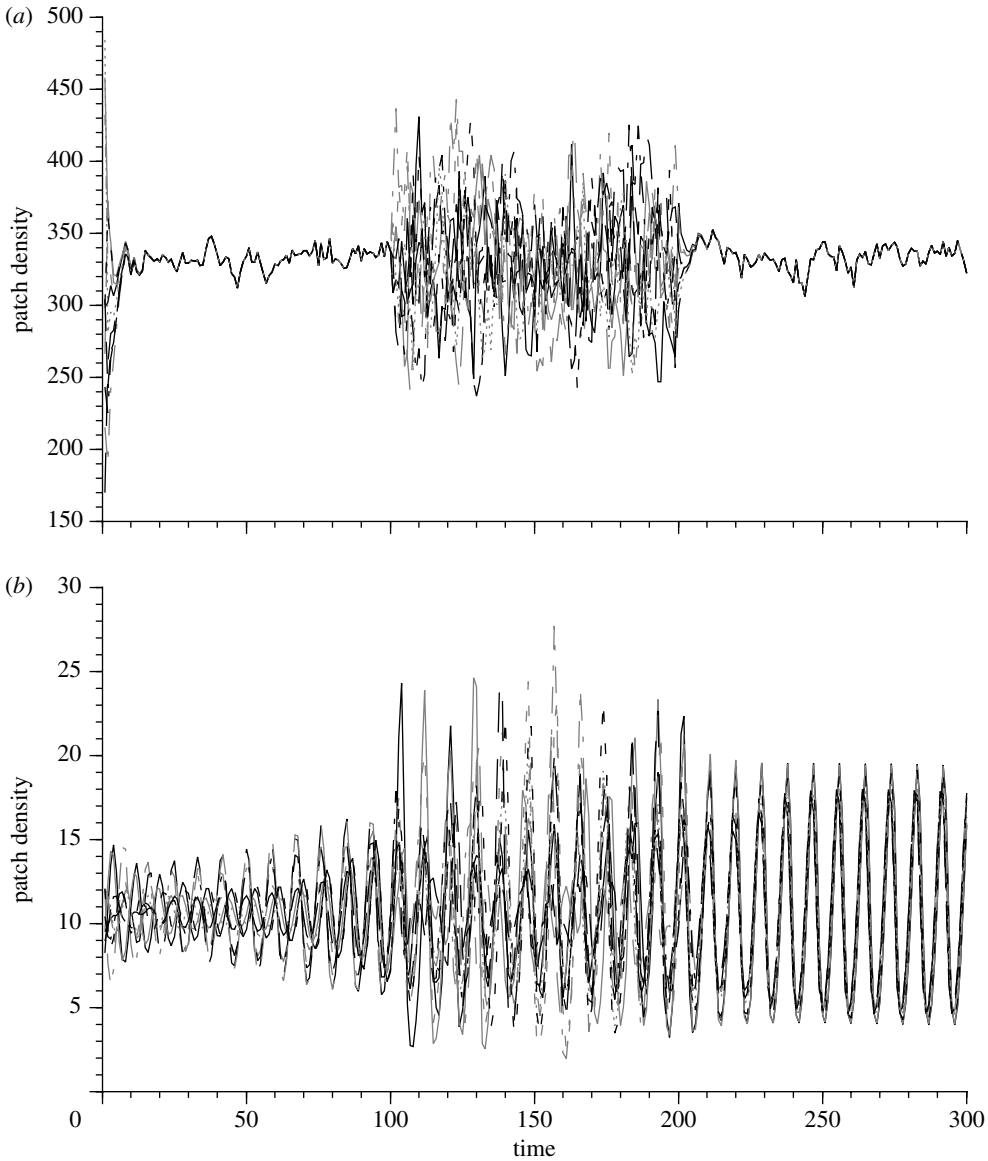

Figure 3. Gain and loss of synchrony in fluctuating population dynamics. Ten population patches with either (*a*) linear population regulation affected by stochastic environmental variation (equation (2.2)) or (*b*) deterministic cyclic dynamics (equation (2.3)) were initiated out of phase, and each patch was linked to its two nearest neighbours for the first 100 time steps ($m=0.15$), leading to a synchronized state of population fluctuations. At $t=100$, the regular dispersal kernel was replaced with a small-world dispersal network ($p=0.5$). The synchronized state is quickly lost under these conditions, and returns again when $p=0$ after $t=200$. Parameter values used: (*a*) $a=0.7$, $d=100$, $w=0.05$ and (*b*) $r=0.47$, $b_1=0.05$, $b_2=-0.1$.

according to differences in their inherent dispersal abilities, spatial separation and their specific ability to filter environmental noise (Cattadori *et al.* 2000; Botsford & Lawrence 2002; Laakso *et al.* 2003).

We tested whether characteristic aspects of synchrony remained robust to small-world dispersal patterns ($0<p<1$), taking our lead from Watts & Strogatz (1998, p. 440): 'Models of dynamical systems with small-world coupling display enhanced signal-propagation speed, computational power, and sycnhronizability.' It has been reported that a small-world network could produce high levels of phase synchronization (Buchanan 2002; Hong *et al.* 2002; Strogatz 2003; Guan *et al.* 2006) in a system of coupled oscillators with '*even a tiny fraction* p *of shortcuts*' (Hong *et al.* 2002, p. 65). With our focus on synchronous fluctuation of populations we explored the effects of small-world dispersal under a wide range of biologically important conditions, including environments where local populations were influenced by globally correlated noise (the Moran effect, figure 1*a*,*c*), or where they experienced spatial heterogeneity in model parameters (figure 1*f*). We experimented with a wide range of dispersal rates and rules, values of p, and varied the precise form of the underlying dynamics by using a variety of commonly used first- and second-order linear and nonlinear models and a trophically structured model as kernels of the density-dependent population renewal process.

The main feature leading to a difference between our results and those that find an increase in synchrony with increasing small-world probability can be found in the underlying formulation of the fluctuating population patches (oscillating nodes). We have used density-dependent regulation in all of our renewal functions, while this is not the case in other formulations of mechanism describing the oscillating units. Figures 2 and 3 demonstrate the effect of switching from a regular to a small-world network structure. Population patches that were initiated at random quickly move into synchrony with each other, as expected from previous research (summarized by Ranta *et al.* 2006). As the linkage between nearest neighbours is broken down and some nodes are connected at random (e.g. at $t=100$ in figure 3), the oscillation behaviour of each patch changes due to small differences among the population densities in each patch being propagated around the network in a different way. The whole system therefore shifts away from the highly synchronous fluctuations. As each node in our models is always connected to two other nodes in the system, we do not explore the effect of scale-free network structure in our networks, which has also been addressed elsewhere (e.g. Pastor-Satorras & Vespignani 2001).

To our knowledge, this is the first time that the small-world network concept has been introduced to spatial population biology. Our results are in direct contrast to results from small-world networks in other fields. Further interesting extensions to this modelling framework include examining the potential for travelling waves to arise under small-world networks, introducing the small-world concept to other (e.g. distant or density dependent) dispersal kernels or implementation under scenarios that include trade-offs between fitness and dispersal ability. Introducing a cost to dispersal, in the form of mortality of some proportion of the dispersers, reduces population growth rate. This has previously been shown to stabilize and synchronize population fluctuations in the absence of any small-world effects (Ruxton & Rohani 1999). In some of the models presented here, reducing population growth rate through dispersal mortality leads to the loss of cyclic dynamics independently of small-world effects. Analysis of the first-order growth function (equation (2.2)) revealed that increasing dispersal mortality (with $p=0$) corresponds to an increase in synchrony. However, for any given value of dispersal mortality (less than 100%), increasing the probability of small-world dispersal ($p>0$, $m>0$) still reduces synchrony across populations. Therefore, introducing dispersal mortality does not qualitatively change the results shown here.

Our results suggest that it is unlikely that there is one overriding or general mechanism behind synchronous fluctuations in population size. Dispersal can synchronize fluctuations, if it is regular, or desynchronize if it is irregular. Deriving the mechanism behind the underlying degree of synchrony between populations will be problematic to resolve until the questions being asked have been tailored to suit the particular system under consideration. Regular dispersal patterns (spatially implicit or explicit) are known to be highly synchronizing from results in theoretical work (e.g. Ranta *et al.* 1995, 2006; Ruxton & Rohani 1999; Kendall *et al.* 2000; Lindström *et al.* 2001), but we have shown here that disrupting this regular form of dispersal can lead to a significant reduction or even to a total loss of synchrony between populations, even those under the influence of the Moran effect. The specific form of movement between populations will undoubtedly be crucial in determining the relative importance of dispersal and its interaction with the Moran effect in promoting synchrony between populations.

Small-world networks lend themselves readily to a conceptual frame for many pertinent ecological and evolutionary questions. One of the basic questions is the significance of arrival of stray individuals into a local population. Often the immigrants bring in characteristics originating from their natal patch. Here we have elaborated one example, the significance of small-world dispersal on the synchrony of fluctuations in density over several local patches with independent population renewal processes, that is, ensemble (global) synchrony. The results suggest that global synchrony can easily be broken down with just a few stray dispersers deviating from the regular dispersal pattern that the majority of individuals follow. This can clearly be an important factor in explaining the differences in the synchrony patterns between species or localities of species occupancies. Changing environmental conditions (e.g. climate) may also promote (or prevent) such stray dispersal events.

While some empirical and theoretical research has been carried out estimating species' dispersal kernels and the importance of these different kernels (Dingle 1996; Kot & Lewis 1996; Turchin 1998; Clobert *et al.* 2001), this issue clearly calls for further investigation. A common finding in empirical data on dispersal in animals and plants is that the majority of dispersers move a short distance only, while less make it further. The kernel for this kind of dispersal is negatively exponential against distance with a long tail to right (Turchin 1998), and many are estimated to be leptokurtic. The small-world dispersal concept differs from such kernels. Here, the probability of immigrating into any patch in the arena following a small-world dispersal event is independent of distance from the

original patch emigrated from. In fact, the small-world concept could also be applied to distant-dependent kernels, for example, the exponential dispersal kernel [$\exp(-\gamma D_{ij})$]. This represents another interesting avenue for further exploration. Figure 1*e* demonstrates the effect of increasing the neighbourhood size, equivalent to increasing the dispersal distance of some dispersers, but this extension does not qualitatively change the results.

Experimental systems already exist that can be tailored to adopt the small-world dispersal kernel. Dey & Joshi (2006) studied the impact of regular dispersal (of various intensity) on emerging synchrony in laboratory populations of *Drosophila*. A minor modification of their redistribution protocol would allow such a system to include small-world dispersal. Our theoretical explorations have provided a strong, testable prediction for such an experiment. Many microcosm systems would be amenable to a small-world dispersal approach. The impacts of small-world systems are unlikely to be limited to aspects of synchronicity in population fluctuations. Patterns of synchrony in infectious disease dynamics are strongly affected by the dispersal patterns among populations. Case studies exist using data of measles outbreaks from the UK (Grenfell *et al.* 2001) and influenza in the USA (Viboud *et al.* 2006) that highlight synchronous dynamics across towns and cities in these countries. When coupled with historical data about the changes in the importance and movement patterns around different population centres, small-world patterns of dispersal may arise. Thus, small-world dispersal is a framework that will have important implications for the most pertinent questions in ecology and evolutionary biology.

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