Using Mechanistic Models to Understand Synchrony in Forest Insect Populations: The North American Gypsy Moth as a Case Study

Karen C. Abbott* **and Greg Dwyer†**

Department of Ecology and Evolution, University of Chicago, Chicago, Illinois 60637

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abstract: In many forest insects, subpopulations fluctuate concurrently across large geographical areas, a phenomenon known as population synchrony. Because of the large spatial scales involved, empirical tests to identify the causes of synchrony are often impractical. Simple models are, therefore, a useful aid to understanding, but data often seem to contradict model predictions. For instance, chaotic population dynamics and limited dispersal are not uncommon among synchronous forest defoliators, yet both make it difficult to achieve synchrony in simple models. To test whether this discrepancy can be explained by more realistic models, we introduced dispersal and spatially correlated stochasticity into a mechanistic population model for the North American gypsy moth *Lymantria dispar*. The resulting model shows both chaotic dynamics and spatial synchrony, suggesting that chaos and synchrony can be reconciled by the incorporation of realistic dynamics and spatial structure. By relating alterations in model structure to changes in synchrony levels, we show that the synchrony is due to a combination of spatial covariance in environmental stochasticity and the origins of chaos in our multispecies model.

Keywords: spatial synchrony, *Lymantria dispar*, difference equation model, dispersal kernel, correlated environmental stochasticity, route to chaos.

Many outbreaking forest insects show high levels of spatial synchrony, in which booms and busts occur at roughly the same time in different locations (Hanski and Woiwood 1993; Liebhold et al. 2000; Liebhold and Kamata 2000; Peltonen et al. 2002; Raimondo et al. 2004; Johnson et al. 2005). Because of the vast geographical areas involved, identifying the cause of synchrony is difficult without mathematical models. For example, models have shown that population synchrony can be caused by a combination of spatially correlated weather and dispersal among populations (reviewed in Bjørnstad et al. 1999; Hudson and Cattadori 1999; Koenig 1999; Liebhold et al. 2004). The extent of synchrony, however, can be strongly affected by temporal fluctuations in population density. Specifically, models that show chaotic temporal fluctuations tend to display low synchrony unless dispersal or spatial correlations in weather are very high (Grenfell et al. 1998; Lande et al. 1999; Bjørnstad 2000; Earn et al. 2000; Kendall et al. 2000; Engen and Sæther 2005). This result is surprising, since previous work by G. D. and colleagues has suggested that forest insect populations, which do exhibit synchrony, often undergo chaotic or chaos-like fluctuations (Dwyer et al. 2004).

One explanation for this contradiction is that most models of spatial synchrony make very simple assumptions about local dynamics, spatial structure, and dispersal and are thus far simpler than nature. The conclusion that chaotic dynamics require high dispersal or strong environmental correlations to produce synchrony may therefore not hold for more realistic models. To explore this possibility quantitatively, in this article we use the gypsy moth *Lymantria dispar* (Lepidoptera: Lymantriidae) as a case study of whether a more detailed model can explain synchrony over large spatial scales. Our approach is to employ a well-studied temporal model for forest insect dynamics and to combine it with realistic dispersal rules and realistic levels of spatial correlation in environmental stochasticity. Although our model uses parameter values estimated for the gypsy moth, we emphasize that it is in fact quite general and could be applied to a wide variety of forest insects.

The gypsy moth is a forest defoliator that reaches outbreak density about every 10 years in its invasive range in

* Present address: Department of Zoology, University of Wisconsin, 430 Lincoln Drive, Madison, Wisconsin 53706; e-mail: kcabbott@wisc.edu.

† E-mail: gdwyer@uchicago.edu.

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the northeastern United States and southeastern Canada (Liebhold et al. 2000). Population fluctuations are roughly synchronous in the sense that correlation coefficients are positive among populations that are hundreds of kilometers apart (Peltonen et al. 2002; Johnson et al. 2005). Gypsy moth demography is density dependent and is influenced by weather, disease, and predation (Waggoner 1985; Williams and Liebhold 1995*a*, 1995*b*; Elkinton et al. 1996; Dwyer et al. 1997; Nealis et al. 1999; Andresen et al. 2001). We seek to understand how these factors interact with dispersal and regional correlations in weather patterns to produce regionwide correlations in gypsy moth densities. Previous approaches to studying synchrony have generally relied on statistical summaries of observational data or have used models that are so simple that they are unlikely to describe any real systems. Our approach instead is to use a temporal model that has survived extensive empirical testing and for which parameter estimates for the gypsy moth are available (Dwyer et al. 1997, 2000, 2004). To this model we add dispersal and environmental stochasticity and we ask whether the processes included in our model can explain observed correlations in gypsy moth populations. We find that our model, despite displaying chaotic dynamics, is readily synchronized by correlated stochasticity. Our results suggest that gypsy moth synchrony is due to a combination of multispecies interactions at the local scale and regional correlations in the weather. More broadly, our study reconciles the observation that the population dynamics of many forest insects seem to be both chaotic and synchronous, a combination that could not be readily explained by previous studies.

Methods

Gypsy Moth Population Dynamics

The two most important groups of natural enemies affecting gypsy moth population dynamics are specialist pathogens and generalist predators and parasitoids (Elkinton and Liebhold 1990; Elkinton et al. 1996). Therefore, we began with the model of Dwyer et al. (2004) that describes gypsy moth population dynamics as a function of a host-pathogen interaction and generalist predation. In this model, gypsy moth outbreaks occur when the insect moves between a low-density equilibrium maintained by predation and a large-amplitude cycle driven by pathogen infection. This model reproduces many temporal aspects of gypsy moth dynamics, such as large-amplitude outbreaks with highly irregular timing (Dwyer et al. 2004). Extending the model to allow for spatial structure gives the following model:

$$N_{i,t} = \varepsilon_{i,t}\lambda N'_{i,t-1}(1 - I_{i,t-1})\left(1 - \frac{2abN'_{i,t-1}}{b^2 + N'^2_{i,t-1}}\right), \quad (1a)$$

$$Z_{i,t} = fN'_{i,t-1}I_{i,t-1}, \quad (1b)$$

with the fraction of individuals infected by the specialist viral pathogen, $I_{i,t}$, given by

$$1 - I_{i,t} = \left[1 + \frac{\bar{\nu}}{\mu k}(N'_{i,t}I_{i,t} + \eta Z'_{i,t})\right]^{-k}. \quad (1c)$$

Here, $N_{i,t}$ and $Z_{i,t}$ are the predispersal densities of hosts and specialist viral pathogens in patch i at the end of generation t, and $N'_{i,t}$ and $Z'_{i,t}$ are postdispersal densities, with dispersal occurring immediately prior to the start of each generation.

In equation (1a), λ represents gypsy moth population growth at low density and takes into account both fecundity and density-independent mortality. The random variable $\varepsilon_{i,t}$ represents environmental stochasticity and is discussed in greater detail below. The parameter a is the maximum fraction of the gypsy moth population killed by predators, whereas b is the insect density at which the predation rate is maximized. We assume that predation follows a Type III functional response, as represented by the term $1 - (2abN'_{i,t-1})/(b^2 + N'^2_{i,t-1})$, the fraction of hosts to escape consumption by generalist predators. For gypsy moths, efforts to document a Type III functional response using experiments have been unsuccessful for at least some generalist predators (Elkinton et al. 2004). However, for many forest insects, including gypsy moths, observational data have shown that low-density populations tend to fluctuate around a stable equilibrium (Elkinton et al. 1996; Dwyer et al. 2004), which is consistent with a Type III but not a Type II response. Moreover, there is good evidence of Allee effects ahead of the advancing front of the gypsy moth invasion (Johnson et al. 2006; Whitmire and Tobin 2006), similarly consistent with a Type III but not a Type II response. In short, because we are attempting to explain population dynamic phenomena and because data collected at the population level support a Type III but not a Type II functional response, we use a Type III functional response in our model.

A fraction f of the virus population overwinters through the environmental contamination of egg masses (Murray and Elkinton 1989, 1990). As larvae hatch in the spring from virus-contaminated eggs, some become infected, reintroducing the virus into the host population (only larvae can become infected). Given this initial input of virus, the fraction of hosts that become infected in the epidemic is then described by the implicit solution of equation (1c). This equation, in turn, is derived from a differential equa-

tion model that describes the dynamics of the epidemic within the larval population and that has survived repeated confrontations with both experimental and observational data (Dwyer et al. 1997, 2000, 2005). A key feature of the epidemic model is that it includes heterogeneity in susceptibility in the form of a distribution of transmission rates with mean $\bar{\nu}$ and inverse-squared coefficient of variation k. The model assumes that the effects of small population sizes on virus epidemics are negligible, which is a reasonable assumption in the outbreaking gypsy moth populations in which epidemics occur. The effects of heterogeneity in susceptibility are, therefore, deterministic in our model. The model also allows for the breakdown of the pathogen on foliage at rate μ, and the higher susceptibility of hatching larvae relative to later-stage larvae is accounted for by the parameter η in equation (1c) (Watanabe 1987). Nondimensionalization shows that f and η affect the dynamics only as the product $f\eta$. Likewise, the composite parameter $b\bar{\nu}/\mu$ fully accounts for the dynamical effects of b, $\bar{\nu}$, and μ. In what follows, we therefore refer to f and η only as $f\eta$, which, roughly speaking, is the overwinter impact of the pathogen, and we refer to b, $\bar{\nu}$, and μ only as $b\bar{\nu}/\mu$, which gives the ratio of the insect density at maximum predation (b) to the minimum insect density required to start a virus epidemic ($\mu/\bar{\nu}$).

Using empirically derived estimates for the parameter values ($\lambda = 74.6$, $f\eta = 60$, $a = 0.967$, $b\bar{\nu}/\mu = 0.14$, and $k = 1.06$ [Elkinton et al. 1996; Dwyer et al. 1997, 2004]) and in the absence of dispersal and environmental stochasticity ($\varepsilon_{i,t} = 1$ for all values of i and t), equations (1a)–(1c) show chaotic population fluctuations (Dwyer et al. 2004). When stochasticity is included, the fluctuations show chaos-like behavior for a broader range of parameter values (Dwyer et al. 2004), a phenomenon that some have termed "noise-induced chaos" (Ellner and Turchin 2005). The temporal model thus provides a useful example of an empirically tested, mechanistic model with which to test the effects of chaos on synchrony.

To allow for spatial structure and, thus, synchrony, we assumed that populations resided in a 50 × 50 grid of discrete habitat patches connected by dispersal. The patches were scaled such that they covered an area of 1,000 km × 1,000 km. We assumed that the boundaries of the grid were reflecting, so that all individuals who dispersed outside the grid were replaced by an equal number of immigrants from unmodeled populations assumed to exist around the grid. Given the large scale of our simulations relative to the rate of dispersal, it is unlikely that this assumption had much effect on our results.

Larval gypsy moths disperse locally by ballooning (Collins 1917), whereas individuals in other life stages, especially egg masses, can be transported over longer distances by humans (Liebhold et al. 1992). Irrespective of the dispersal mechanism, it is highly likely that the fraction of individuals that move a given distance declines roughly continuously with distance (Kot et al. 1996). Because our main interest is in large-scale synchrony, we included only long-distance dispersal. Indeed, the typical larval ballooning distance is much less than 50 m (Hunter and Elkinton 2000), whereas populations in our simulations were separated by 20 km. Ballooning, therefore, has little effect on synchrony at the scale of our simulations (Abbott 2006). Note that our model does not assume that there is no local dispersal, but instead that local dispersal is high enough within a grid cell that hosts and pathogens are well mixed. To mimic the long-distance movement of egg masses on humans' vehicles, we assumed that a small fraction q of each population was transported to other patches between the time the egg masses were laid in the autumn of year $t - 1$ and when they hatched in the spring of year t. In virus epidemics, contamination of egg masses is widespread, so it is likely that the virus disperses in this way as well.

We used an exponential function to describe the distance traveled by dispersing individuals. Because our model describes discrete patches, the probability of dispersing between grid cells i and j was determined by the discretized exponential kernel

$$k(d_{i,j}) = \kappa \exp(-D_k d_{i,j}). \tag{2}$$

In equation (2), $d_{i,j}$ is the distance in kilometers between populations i and j and D_k determines how steeply the fraction of individuals moving from i to j declines with increasing distance between the populations. The variable κ is a scaling constant that forces the fractions dispersing all possible distances to sum to 1. We calculated κ by first defining dispersal to be possible for any value $d_{i,j}$ for which $\exp(-D_k d_{i,j}) \geq 10^{-10}$. We then arranged habitat patches on a square grid large enough that the distance from the center cell c to the edge of the grid exceeds the farthest possible dispersal distance. We computed $\exp(-D_k d_{c,j})$ for all j on this grid to which dispersal from c was possible, and we summed these values to find $1/\kappa$. In equation (2), we set $k(d_{i,j})$ equal to 0 for values of $d_{i,j}$ such that $\exp(-D_k d_{i,j}) < 10^{-10}$. In our simulations, we used the values $q = 10^{-5}$ and $D_k = 0.175$. These are maximum likelihood values estimated by Fujita (2007) to simultaneously explain spatial structure in the allele frequencies of the gypsy moth's virus and observed rates of gypsy moth range expansion. Fujita (2007) fitted a stratified dispersal kernel that accounted for both short- and long-distance movement. As explained above, short-distance movement (by larvae) occurs at a small enough spatial scale that movement between populations in our simulation is extremely unlikely. In our dispersal kernel, equation (2), we therefore

use only the long-distance piece of Fujita's best-fit stratified kernel.

At the start of each simulation, all habitat patches were empty except for three cells in the center of one edge of the grid. These initial conditions mimic the gypsy moth invasion in North America, which spread north, south, and west from its starting point in Massachusetts (Liebhold et al. 1989). We then ran simulations for 100 generations, which is on the order of how long the gypsy moth has been invading North America. Analyses were performed on the last 20 years from each of 25 replicate simulations, and we present the results averaged across realizations.

Overwhelming empirical evidence has shown that gypsy moth populations are influenced by weather (Waggoner 1985; Williams and Liebhold 1995*a*, 1995*b*; Nealis et al. 1999; Hunter and Elkinton 2000; Andresen et al. 2001). Because many weather variables are spatially correlated and because the correlation is greater at shorter distances (Koenig 2002; Peltonen et al. 2002), we assumed that the covariance of the environmental stochasticity experienced by two populations in a given year declined with the distance between those populations. To allow for this correlation, we write $\varepsilon_{i,t}$ in equation (1a) as a lognormally distributed random variable:

$$\varepsilon_{i,t} = \exp(\mathbf{M}_t(i)), \tag{3a}$$

$$C_{i,j} = c^2 \exp(-D_\varepsilon d_{i,j}), \tag{3b}$$

where $\mathbf{M}_t(i)$ is the ith element of a multivariate normal random variable drawn in year t with a mean value of 0 and variance-covariance matrix $\mathbf{C}$, the elements of which are given in equation (3b). As above, $d_{i,j}$ is the distance in kilometers between population i and population j. It is very difficult, if not impossible, to determine values for c and D_ε empirically, so we ran simulations under many different combinations of values. For the simulations we present here, we used $c = 1.5$ and $D_\varepsilon = 3.3 \times 10^{-3}$, under which the model produces levels of gypsy moth synchrony that agree well with data on the insect. With these parameter values, correlations among $\varepsilon_{i,t}$ for patches separated by different distances are within the range of correlations observed in real weather data (table 1). Our simulated environmental correlations are, in fact, on the low end of the range observed in the data. Given that we argue that synchrony is largely driven by weather, this means that our assumptions about c and D_ε are conservative.

Table 1: Correlation coefficients for the values of $\varepsilon_{i,t}$ drawn in our simulations and for some real weather variables at different distances

Distance (km)	$\varepsilon_{i,t}$	Monthly precipitation	Date of last spring frost	No. days <-26°C
100	.46	.48	.32	.67
300	.22	.40	.19	.55
500	.10	.21	.08	.53

Note: Data were obtained from the National Oceanic and Atmospheric Administration from weather stations covering part of New England (i.e., Maine, New Hampshire, Vermont, and Massachusetts) over which gypsy moth populations outbreak in synchrony (Liebhold et al. 2000). We fitted nonparametric correlation functions (Bjørnstad and Falck 2001; R Development Core Team 2005) and report here the mean correlations at several distances for some of the weather variables that are believed to influence gypsy moth populations: precipitation (Williams and Liebhold 1995*a*, 1995*b*); spring frosts, which influence the timing of early spring events such as tree budburst and gypsy moth egg hatch (Hunter and Elkinton 2000); and temperatures that drop below what appears to be a physiological threshold at -26°C (Waggoner 1985).

Statistical Methods

To quantify regional synchrony, we used a modified correlogram (after Koenig 1999) in which the distances between pairs of populations were divided into discrete distance classes and the average pairwise correlation coefficient between population trajectories was plotted for each distance class. We created these distance classes such that each class contained at least 2,500 correlation coefficients (i.e., 2,500 pairs of populations). The correlation coefficients we present were calculated on the basis of annual differences in log abundances in the simulated time series. In other words, for each simulated population we constructed a time series of transitions, $(\log_{10} N_{i,t+1} - \log_{10} N_{i,t})$, $(\log_{10} N_{i,t+2} - \log_{10} N_{i,t+1})$, …. We then calculated a Pearson's correlation coefficient for this series of transitions among each pair of populations. This approach allows us to directly compare our results to those of previous analyses of gypsy moth data that used the same method (Peltonen et al. 2002; Johnson et al. 2005).

Most standard statistical tests cannot be applied to measures of synchrony among population trajectories because time series data are temporally autocorrelated (Buonaccorsi et al. 2001) and because the set of all pairwise population comparisons is not a set of independent data points (Koenig 1999). Bootstrapping algorithms that resample the sources of randomness in the population trajectories have been proposed to compensate for these dependencies (Bjørnstad and Falck 2001; Buonaccorsi et al. 2001; Lillegård et al. 2005). We used this type of approach by simulating our model 25 times and using different draws from the distribution of $\varepsilon_{i,t}$ to generate environmental stochasticity in each realization, as that is the source of randomness in our model. We then present the mean correlation coefficient for each distance class on the basis of the 25 iterations and the 95% confidence interval about this mean correlogram.

Interpreting the Simulation Results

The goal of our study was to test whether a biologically motivated model can reproduce observed levels of insect synchrony under realistic assumptions about dispersal and environmental correlations. As explained in the introduction to this article, such synchrony is not expected under classical population models, so it is important to explore why our model behaves differently. We therefore carried out additional simulations of our model with some of the assumptions altered to identify the components of the model that are responsible for the degree of synchrony that the model displays.

Dispersal and correlated environmental stochasticity each potentially synchronize the subpopulations in our model. To understand the contributions of these two synchronizing agents, we ran additional simulations with either dispersal or correlated stochasticity removed. Because gypsy moths are an invasive species in North America, in our original simulations we began with gypsy moth populations resident in only three grid cells so that the remaining cells were subsequently colonized by dispersing individuals. If we simply set the fraction of the population that disperses q to 0, the grid would remain empty aside from the three initially occupied cells. We therefore eliminated dispersal in two different ways. First, we allowed individuals to disperse until the entire grid was colonized, then we set $q = 0$ for the remainder of the time steps. Second, we used nonzero initial conditions in all the grid cells and set $q = 0$ for the entire simulation. In this latter case, we used randomly chosen initial densities that were near the attractor of the nonspatial version of equation (1). To eliminate the effects of correlated stochasticity, we simply set all of the off-diagonal elements of the variance-covariance matrix **C** (eq. [3b]) to 0.

Next, we tested how the maximum covariance in the environment (c^2) and the rate at which the covariance declines with distance (D_ε) affect the resultant population synchrony. We ran simulations in which the maximum covariance was lower ($c = 0.5$) or the covariance declined 10 times as fast with distance ($D_\varepsilon = 3.3 \times 10^{-2}$). It is generally very difficult to directly measure the environment's effect on a population, so our additional simulations provide meaningful information about how the properties of $\varepsilon_{i,t}$ influence population synchrony.

Preliminary simulations suggested that synchrony among invaded patches is higher at the early stages of the gypsy moth invasion, so we ran additional simulations for 50, rather than 100, years. We also examined simulations that ran for 200 and 500 years. Finally, we tested the sensitivity of our results to the coarseness of our population grid by dividing the 1,000 km × 1,000 km habitat into either a 20 × 20 patch or a 100 × 100 patch grid of populations, rather than a 50 × 50 patch grid.

Most nonlinear discrete-generation models in the literature follow the period-doubling or the quasi-periodic route to chaos (Rasband 1990; Kuznetsov 1995). In contrast, the chaos in our temporal model appears to be influenced by the occurrence of a chaotic repeller for nearby parameter values. Chaotic repellers occur as a result of interactions among multiple attractors (Tél 1990). In our spatial simulations, we set the pathogen overwintering impact $f\eta = 60$, which results in a global chaotic attractor. To see the influence of the chaotic repeller, we can instead set $f\eta = 20$, for which the model has multiple attractors, including a stable point equilibrium, a phase-locked limit cycle, a quasi-periodic attractor, and a saddle point (Dwyer et al. 2004). The stable point equilibrium is maintained at low density by predation, whereas the limit cycle and the quasi-periodic attractors are associated with a high-density equilibrium maintained by the pathogen. As $f\eta$ is increased, however, the low-density equilibrium undergoes a series of period doublings that produce a small-amplitude global attractor, but this global attractor coexists with a large-amplitude chaotic repeller. As is characteristic of chaotic repellers, most population trajectories take many generations to approach the global attractor, and during this transient period, they are chaotic even though the attractor itself is not chaotic (Tél 1990). This transient chaos can be sustained by the addition of a small amount of stochasticity (Ellner and Turchin 2005), which leads to large-amplitude, chaos-like fluctuations about the global attractor. As $f\eta$ is increased still further, the large-amplitude chaotic repeller becomes a global chaotic attractor, and this is apparently the attractor produced by the parameter values that we used in our spatial simulations. Numerical evidence thus suggests that the chaos in our model is approached by way of a chaotic repeller, in contrast to the more commonly studied period-doubling or quasi-periodic routes.

In the absence of predation, other parameter values produce different routes to chaos, such as period doubling and quasi periodicity, as well as chaos that is associated with nonstandard or degenerate quasi-periodic cycles due to strong resonances or homoclinic orbits (Kuznetsov 1995; G. Dwyer, unpublished results). Period doubling is the route to chaos characteristic of the single-species models that have been the focus of most previous work on synchrony and chaos (e.g., Allen et al. 1993; Ranta et al. 1998, 1999; Bjørnstad 2000; Greenman and Benton 2001). In multispecies models, chaos is also often reached through the quasi-periodic route (Rohani et al. 1994; Kot 2001). In order to test the importance of the route to chaos in allowing synchrony, we ran additional simulations of the

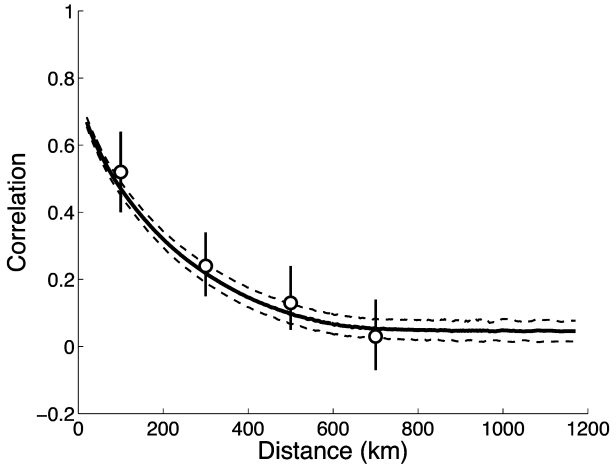

Figure 1: Average population synchrony at different distances. The solid line shows our simulation result and dashed lines represent the 95% confidence intervals (CIs) calculated from our 25 replicate simulations. The circles (with error bars showing 95% CIs) are estimates obtained from 20 years of gypsy moth data by Peltonen et al. (2002).

host-pathogen-only model with parameter values that give chaos that is associated with these alternative routes.

The full host-pathogen-predator model exhibits long-period fluctuations in density, matching the long-period fluctuations of gypsy moth populations in nature. In contrast, chaotic fluctuations in the host-pathogen-only model have shorter periods except when chaos is due to a homoclinic loop (G. Dwyer, unpublished results). In a homoclinic loop, trajectories following the unstable manifold of a saddle point, which is near the origin in our model, reconnect with the stable manifold of the saddle point, leading to cycles that can have a very long period (Kuznetsov 1995). Unfortunately, long-period chaotic fluctuations in the host-pathogen-only model are only weakly chaotic (G. Dwyer, unpublished results), so we were unable to simultaneously match both the long period and strong chaos of the full model using an alternative route to chaos. Nonetheless, simulating the host-pathogen-only model for parameter values giving a long-period homoclinic loop allowed us to see whether it is the long period of our full model's chaotic fluctuations, rather than the route to chaos per se, that permits synchrony.

We quantified the strength of chaos using the Lyapunov exponent of the within-population dynamical model, which describes the tendency of two model trajectories to grow apart, given that they start close together. If the Lyapunov exponent is positive, the model is chaotic, and larger exponents indicate stronger chaos.

Results

The correlogram produced by our stochastic spatial model closely matches published data on gypsy moth synchrony (fig. 1; data from Peltonen et al. 2002). This synchrony in the model appears to be driven by correlations in the environment, because simulations run with an uncorrelated environment were asynchronous (fig. 2, *dark gray line*). Simulations with no dispersal were as synchronous as the full simulation, except perhaps at very long distances, where the correlations for the models without dispersal began to drop below the 95% confidence interval for the full model (fig. 2, *medium* and *light gray lines*).

Changing the values of c and D_ε in equation (3), the only parameters for which we had no empirical estimates, had some effect on the correlogram. As one would expect, reducing the maximum covariance c or increasing the rate of decline of the covariance with distance by reducing D_ε causes the population synchrony to be weaker than the synchrony in the data (fig. 3, *gray lines*). Because it is not possible to independently estimate c and D_ε (see "Discussion"), we have approached the problem of unknown environmental correlations by determining how correlated the environment would need to be to explain population synchrony and then asking whether that level of correlation is realistic (following Grenfell et al. 1998). We therefore adjusted c and D_ε until there was agreement between our simulated populations and the gypsy moth data. To independently assess whether the resulting values of c and

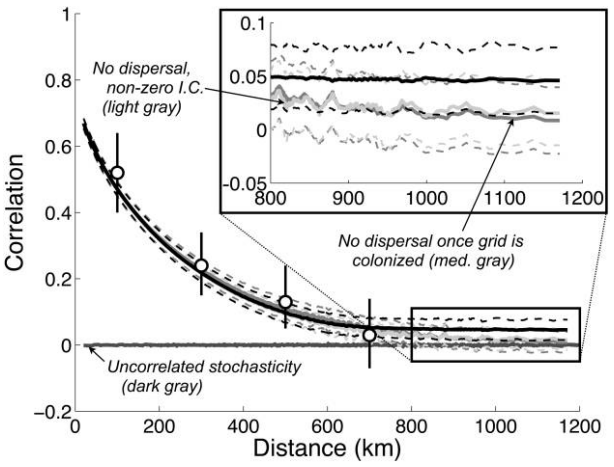

Figure 2: Average population synchrony without dispersal (*light gray*, *medium gray*) or without correlated stochasticity (*dark gray*). The model and data from figure 1 are replotted in black for comparison. Two models with no dispersal were considered; see "Interpreting the Simulation Results" for details. The inset gives a closer view of long-distance synchrony, with the model lacking correlated stochasticity removed to emphasize the relationships among the other three models. *I.C.* = initial conditions.

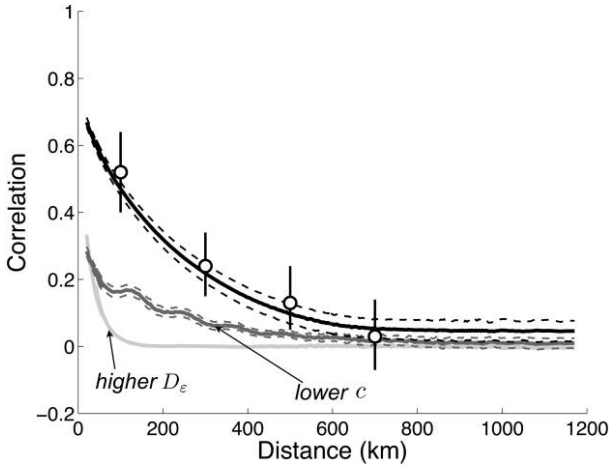

Figure 3: Average population synchrony under different environmental conditions. The black line shows the simulation from figure 1: $c = 1.5$, $D_\varepsilon = 3.3 \times 10^{-3}$; the darker gray line shows a simulation with lower maximum covariance in $\varepsilon_{i,t}$: $c = 0.5$, $D_\varepsilon = 3.3 \times 10^{-3}$; and the lighter gray line shows a simulation with a sharper decrease in the covariance in $\varepsilon_{i,t}$ with distance: $c = 1.5$, $D_\varepsilon = 3.3 \times 10^{-2}$.

D_ε were realistic, we compared the spatial correlations in the random variates produced by the model to spatial correlations in weather data (table 1), on the assumption that weather fluctuations are the most likely driver of stochasticity in gypsy moth populations. Reassuringly, there is a good correspondence between the two.

Our conclusions appear to be robust to changes in most of our other assumptions (fig. 4). Simulations run for 200 or 500 years showed the same levels of synchrony as the simulation run for 100 years, although population synchrony was greater at all distances when simulations were run for only 50 years. When we divided the habitat into a 20 × 20 or 100 × 100 grid of populations, rather than a 50 × 50 grid, the correlogram was again essentially unchanged.

We also simulated the host-pathogen-only model using unrealistic parameter values that produced different routes to chaos. As in previous studies, when chaos was reached through period doubling, synchrony was quite low relative to the data (fig. 5, cases 1 and 3). For chaos reached through quasi periodicity, the model again showed much lower synchrony than in the data (fig. 5, case 4). The same was true for degenerate quasi periodicity caused by strong resonance (fig. 5, case 2) or a homoclinic loop (fig. 5, case 5). Therefore, it appears that the chaotic repeller of the full host-pathogen-predator model is unique in permitting both chaos and realistic synchrony. This is particularly striking because the chaos in our full model was substantially stronger than the chaos reached by some of our alternative routes (fig. 5, cases 3–5). We cannot say with certainty that the route to chaos per se, rather than the combination of strong chaos and long period, permitted synchrony in our full model, since we were unable to mimic this combination using an alternative route to chaos. If the long period does contribute to synchrony, however, we are unable to suggest an explanation that is independent of route to chaos for why strongly chaotic long-period fluctuations (fig. 1, full model) should be more synchronous than very weakly chaotic long-period fluctuations (fig. 5, case 5). Clearly, the question of how routes to chaos affect synchrony is poorly understood, but it appears to be the case that route to chaos plays an important role in allowing synchrony when the temporal dynamics are chaotic.

In spatial models, it is possible for dispersal to cause qualitative changes to within-patch population dynamics that often result in simpler or more stable dynamics (e.g., Gurney and Nisbet 1976; Reeve 1988; Hastings 1993; Amarasekare 1998; Ruxton and Rohani 1999). If populations in our spatial model are exhibiting different dynamics, then the spatial model might no longer give an accurate representation of local gypsy moth populations. To check whether this was the case, we compared a phase plot for the nonspatial model to a phase plot for one population within the spatial model. The plots were vir-

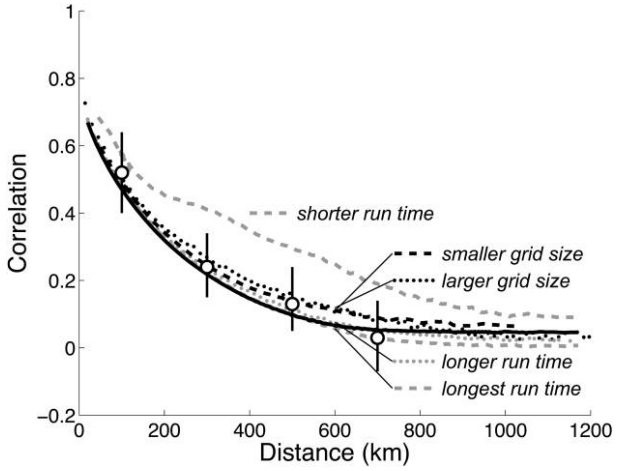

Figure 4: Average population synchrony predicted by versions of our model that have different assumptions about time and space. The model and data from figure 1 are shown again (*circles* and *black solid line*), and variants of that model are shown with dashed and dotted lines. The original model was run on a 50 × 50 grid of populations for 100 generations. The following variants were considered: smaller (20 × 20) or larger (100 × 100) population grids; shorter (50 generations) or longer (200 and 500 generations) simulations. Confidence intervals for these simulations are not shown because of the large number of lines plotted in this figure.

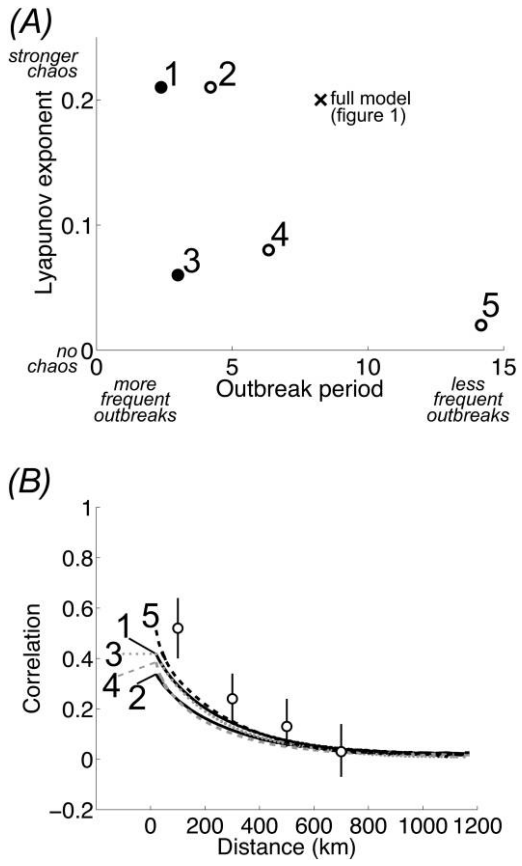

Figure 5: Comparison of levels of synchrony exhibited by simulations with different mean outbreak periods, strengths of chaos, and routes to chaos. *A*, Filled circles represent cases in which chaos is reached through period doubling and open circles represent cases in which the route to chaos is quasi periodicity (cases 2 and 5 are degenerate). Each circle corresponds to the line in *B* marked with the same number. In all cases, $a = 0$; other parameters are, for case 1: $\lambda = 74.6$, $f\eta = 0.01$, $k = 3.85$; for case 2: $\lambda = 21$, $f\eta = 1$, $k = 5$; for case 3: $\lambda = 74.3$, $f\eta = 0.01$, $k = 3.81$; for case 4: $\lambda = 15$, $f\eta = 5$, $k = 3$; and for case 5: $\lambda = 3$, $f\eta = 160$, $k = 1.25$. For reference, the outbreak period and Lyapunov exponent are marked with a cross for the full host-pathogen-predator model with empirical parameter estimates ($\lambda = 74.6$, $f\eta = 60$, $a = 0.967$, $b\bar{v}/\mu = 0.14$, $k = 1.06$). The correlogram for this reference case is shown in figure 1.

tually identical (not shown), suggesting that dispersal in the spatial model did not cause a qualitative change to within-patch dynamics.

Our simulated gypsy moth invasion spread at an average rate of 15.1 km year^{-1}, which agrees with an analysis by Liebhold et al. (1992) showing that the rate of spread of the gypsy moth invasion in North America has historically ranged from 2.8 to 20.8 km year^{-1}. This result is not surprising, because the parameter estimates for our long-distance dispersal kernel came from an analysis that was, in part, aimed at fitting observed rates of spread (Fujita 2007). More interesting is the result from our simulations that the invasion advanced in pulses. We saw intermittent peaks in the number of new grid cells colonized per generation, with an average dominant period of 3.7 years. Johnson et al. (2006) showed that gypsy moths have significant invasion pulses with a 4-year period and suggested that this is due to a combination of an Allee effect and long-distance movement by a small portion of the population. Both of these mechanisms are present in our model, since the density-dependent predation term in equations (1a)–(1c) causes small gypsy moth populations to decline, producing an Allee effect. Thus, our simulations agree with empirical estimates not only of synchrony but also of the average rate of spread and of the temporal variability in the spread rate of the gypsy moth.

Discussion

It is difficult, using existing ecological theory, to reconcile strong synchrony with the apparent chaos of forest insect populations. Previous theoretical investigations have found that populations governed by chaotic or strongly nonlinear dynamics cannot be synchronized by moderate levels of correlated stochasticity (Allen et al. 1993; Grenfell et al. 1998; Greenman and Benton 2001), unless dispersal is extremely high (Ranta et al. 1998, 1999). A key feature of our study is that we used a multispecies population model, whereas most previous studies have instead used simple single-species models (Allen et al. 1993; Ranta et al. 1998, 1999; Bjørnstad 2000; Greenman and Benton 2001). This is important because single-species models can reach chaos by the period-doubling route, but they cannot have quasi-periodic attractors and therefore cannot reach chaos by the quasi-periodic route. Rohani and Miramontes (1995) demonstrated that chaotic dynamics that are reached by these two different routes respond very differently to the addition of an immigration term. It seems likely that the route to chaos could also have an effect on other spatial phenomena, such as synchrony. Chaotic single-species models might therefore have requirements for synchrony that are different from those of chaotic multispecies models. More directly, the few studies that look specifically at synchrony in multispecies models have not reached a consensus on whether chaotic multispecies systems resist synchrony as strongly as do chaotic single-species systems. Blasius et al. (1999) found a high incidence of phase synchrony (which is distinct from, but related to, the concept of synchrony that we use here) in a chaotic tritrophic model. In contrast, Gao et al. (2007) reported low synchrony in chaotic host-parasitoid dynamics. Most studies of synchrony in multispecies models, meanwhile, have focused on fundamentally different questions, such as how the movement of one species can induce synchrony

in a species with which it interacts (Ims and Steen 1990) or how multispecies interactions change the overall shape of the synchrony-distance relationship (Ranta et al. 1997; Bjørnstad and Bascompte 2001).

Our results suggest that the route to chaos affects synchrony in that only the host-pathogen-predator model, with its chaotic repeller route, showed realistic levels of synchrony. All other routes to chaos, whether by period doubling or by quasi periodicity, showed lower synchrony (fig. 5), in keeping with previous results from single-species models. Although we suspect that route to chaos is important, it is worth considering whether removing generalist predation necessarily reduces synchrony, since we used the host-pathogen-only model to explore the alternative routes to chaos. For many parameter values, the host-pathogen-only model exhibits limit cycles. Interestingly, these limit cycles may produce synchrony that is either higher or lower than what is observed, depending on the parameter values. Because some host-pathogen-only limit cycles do show high synchrony, we conclude that removing generalist predation does not necessarily reduce synchrony and that route to chaos indeed appears to be the key feature underlying the lower synchrony of chaotic host-pathogen-only simulations.

In the host-pathogen-predator model, attractors associated with multiple equilibria merge to form the chaotic attractor. The component attractors include a predator-driven, small-amplitude, short-period cycle produced by period doubling and two pathogen-driven, large-amplitude, long-period cycles produced by a Hopf-type bifurcation. As the population trajectory moves around the global chaotic attractor, it appears to visit the regions of the global attractor that are influenced by each of these component equilibria. The insect and pathogen populations apparently switch erratically between the component equilibria, and environmental stochasticity enhances this behavior by promoting random switching (Dwyer et al. 2004). When the switching is partly or entirely due to environmental stochasticity, we speculate that correlations in the environment may be especially effective at inducing synchrony. The role of multiple equilibria in producing chaos in our model may therefore make it particularly prone to synchronization by correlated stochasticity. Moreover, complex dynamics that are driven by multiple coexisting equilibria and environmental stochasticity may be common in multispecies interactions (e.g., Hall et al. 2005; Abbott and Dwyer 2007; Ives et al. 2008). Whether other such models also show high synchrony is therefore an exciting avenue of future research.

Relevant to the discussion of when models can and cannot show synchrony is the work of Earn et al. (2000), who studied analytical conditions for coherence caused by dispersal (see also Earn and Levin 2006). When densities in different subpopulations are equal or within a small percentage of each other, they are said to be coherent. Earn et al. (2000) showed that the strength of chaos, but not the particular route to chaos, affected the possibility of coherence in deterministic models, and their results were found to be quite robust to the addition of uncorrelated stochasticity. How the conditions for coherence change with the introduction of correlated stochasticity, however, remains an open question. Most importantly, with regard to our results, it is unknown whether the route to chaos should remain unimportant when coherence is caused by correlated stochasticity rather than dispersal.

A basic assumption of our model is that all populations are governed by the same underlying deterministic model with the same demographic parameter values. In contrast, Liebhold et al. (2006) found geographical variation in gypsy moth demographic rates and used a linear model to show that such variability can sometimes reduce the strength of population synchrony due to environmental correlation. In preliminary simulations of our nonlinear demographic model, however, we found no such reduction in synchrony when we allowed the demographic parameters to vary among patches (Abbott 2006). Indeed, Liebhold et al. (2006) also noted that nonlinear models did not always behave analogously to the linear model that was the focus of their study. We therefore believe that our simulation results will hold under more complicated assumptions about how demographic processes vary among populations.

Different types of environmental stochasticity can have different effects on the spatial pattern of synchrony among populations (Abbott 2007). In our simulations, we used a multivariate lognormal model for environmental stochasticity in which covariance declined exponentially with distance. This is reasonable for at least some weather variables (Koenig 2002), but it is certainly not the only possible model. There is evidence that gypsy moth dynamics are affected by temperature and precipitation (Williams and Liebhold 1995*a*, 1995*b*), by the timing of egg hatching relative to tree budburst (Hunter and Elkinton 2000), and by the number of extremely cold winter days (Waggoner 1985), so we do have some knowledge of how gypsy moth populations are affected by weather. Determining the precise contribution of weather to gypsy moth synchrony, however, would require detailed information on how these and probably other factors interact, how they are correlated through space, and precisely how they alter population dynamics. In the absence of such information, we have used a simple and commonly employed model of correlated weather in our simulations. Because we were forced to estimate the parameters of this weather model by fitting our demographic model to the synchrony data, we cannot use our simulation results to definitively infer

that correlated weather is largely responsible for synchrony in nature. The simplest alternative explanation is that gypsy moth synchrony is caused by high rates of human-induced egg dispersal, but this scenario appears to be unrealistic (Fujita 2007). Meanwhile, the level of correlation that we used in our model falls within the range of values seen in various types of weather data (table 1), suggesting that our results are robust. Moreover, in a comparative analysis of data on six forest insects (including the gypsy moth), Peltonen et al. (2002) also concluded that correlated weather and not dispersal was likely the proximate cause of synchrony. For these reasons, we suspect that correlated environmental stochasticity is indeed an important contributor to gypsy moth synchrony in nature as it is in our model, but we cannot state this with certainty. Our main contribution, therefore, is not to elucidate how the details of correlated weather may contribute to gypsy moth synchrony but to demonstrate that, to achieve synchrony among chaotic populations with low levels of dispersal, we need not impose extraordinarily high degrees of correlation in the stochastic environment.

Although human-induced dispersal is likely an important factor in the rapid spread of the gypsy moth invasion (Liebhold et al. 1992), our study suggests that long-distance egg dispersal caused by humans is not driving gypsy moth synchrony. Instead, synchrony in our model appears to be due to correlations in the environment acting on complex multispecies population dynamics. Although we have used the gypsy moth as an example, the assumptions of our model are appropriate for many outbreaking forest insects (Dwyer et al. 2004). Consequently, our results more generally suggest that synchrony among other outbreaking forest insect populations might likewise arise because of correlated stochasticity and the route to chaos. More broadly, the contrast between our results and those on synchrony in single-species models emphasizes the important role of mechanistic models in examining the causes underlying complex population dynamics.

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