Journal of Animal Ecology 2000, **69**, 869–873

Cycles and synchrony: two historical 'experiments' and one experience

OTTAR N. BJØRNSTAD

National Center for Ecological Analysis and Synthesis, 735 State St., Suite 300, Santa Barbara, California 93101–3351 USA

Summary

1. Theoretical models predict that spatial synchrony should be enhanced in cyclic populations due to nonlinear phase-locking.
2. This is supported by Rohani *et al.*'s (1999) comparison of spatial synchrony of epidemics in two childhood diseases prior to and during the vaccination era. Measles is both more synchronous and more cyclic before vaccination. Whooping cough, in contrast, is more synchronous during the vaccination era, during which multiannual fluctuations are also more conspicuous.
3. Steen *et al.* (1990) analysed historic records of cyclic rodents, to show that cyclicity was lost during the early part of the 20th century. I reanalyse the data, and find that the loss of cyclicity is associated with loss of regional synchrony.
4. I use a coupled map lattice model to show that imperfect phase-locking provides an alternative explanation for regionwide synchrony of cyclic populations.

Key-words: coupled map lattice, measles, metapopulation correlation, microtine rodents, population dynamics, spatial covariance, vaccination.

Journal of Animal Ecology (2000) **69,** 869–873

Introduction

The 'spatial frontier' has been pushed with vehemence throughout the 1990s (Tilman & Kareiva 1997). A common goal has been to understand the effect of space on interactions and dynamics of ecological systems. In population ecology, empiricists and theoreticians have converged in a quest to quantify spatial synchrony (Bjørnstad, Ims & Lambin 1999a). Field studies have uncovered widespread – sometimes continentwide – synchrony of fluctuations (Ranta *et al.* 1997; Koenig 1999). Initially, theoretical ecology offered wide-scaled correlation in weather as the explanation (Ranta *et al.* 1995). This solution is based on the pioneering work of Moran (1953) who noted that populations governed by linear dynamics would share exactly the correlation of the environment. Theoretical modelling have since argued that the inherent nonlinearities of population growth sabotage this simple equivalence, synchrony may be much greater or much lower than that of the environment (Grenfell *et al.* 1998; Ranta, Kaitala & Lundberg 1998; Blasius, Huppert & Stone 1999; Jansen 1999; Ranta, Kaitala & Lindström 1999). An important special case is that of spatially extended cyclic systems. By *cyclic systems*, in this context, I mean metapopulations where the local populations exhibit limit cycles in abundance. [It is crucial, here, to stress that the discussion presented in this note applies to cycles arising from nonlinear population regulation. It does not apply to the periodic fluctuations that arise from certain stochastic linear maps; Nisbet & Gurney (1982); Kaitala, Ranta & Lindström (1996); Stenseth, Bjørnstad & Saitoh 1996]. With cyclic dynamics, theory suggests that even local dispersal or local climatic correlation can induce regionwide synchrony (Bjørnstad *et al.* 1999a; Jansen 1999). Figure 1 predicts the effect of nonlinear regulation on metapopulation synchrony. I use a coupled map lattice where the predispersal abundance, $N'_{i,t}$, of population i at time t governed by a stochastic Ricker equation:

$$N'_{i,t} = N_{i,t-1} \exp[r(1 - N_{i,t-1}) + u_t], \qquad \text{eqn 1}$$

where $N_{i,t}$ represents postdispersal abundance, r is the scaled growth rate, and u is a sequence of identically distributed zero-mean Gaussian random variables (with variance σ^2) that represent environmental stochasticity. In this way, the realized

Correspondence: Dr Ottar N. Bjørnstad, National Center for Ecological Analysis and Synthesis, 735 State St., Suite 300, Santa Barbara, California 93101–3351 USA. E-mail: bjornsta@nceas.ucsb.edu. Tel.: (805) 892–2520. Fax: (805) 892–2510.

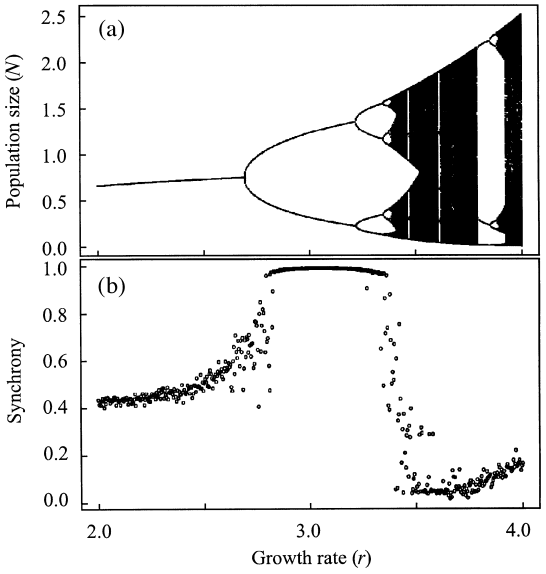

Fig. 1. Simulation of 10 populations on a one-dimensional coupled map lattice. The local populations are coupled by nearest-neighbour dispersal (50% of the population are allowed to disperse evenly to the two neighbouring populations; the lattice is assumed circular to avoid boundary effects). Local dynamics are assumed to follow stochastic Ricker dynamics (eqn 1); σ is the standard deviation of the environmental stochasticity affecting the population growth. (a) Bifurcation diagram for the deterministic system ($\sigma = 0$) as the growth-rate, r, is changed. The population size represents the average population size across the 10 local populations (after allowing for a 500-generation transient period). (b) Average level of synchrony among the 10 local populations (after allowing for a transient period) when the populations are affected by correlated environmental stochasticity ($\sigma = 0{\cdot}1$, correlation = 0·3) as the growth rate, r, is tuned to change the dynamics. The near-perfect correlation for the cyclic dynamics is due to non-linear phase-locking. Locally stable populations are slightly more correlated than the environment (due to the added effect of dispersal), as predicted by linear theory. Chaotic populations exhibit very little synchrony; Neither dispersal nor regional correlation synchronize the dynamics.

population growth is log-normal and density-dependent (e.g. Engen & Lande 1996). The stochasticity is assumed to be uncorrelated through time. However, it is assumed to be spatially correlated in the simulations (Fig. 1). If we denote the full vector of local populations by bold face, the equations for the post-dispersal abundance, $\mathbf{N_t}$, at time t can be represented as:

$$\mathbf{N_t} = \kappa \times \mathbf{N}\prime_\mathbf{t}, \qquad \text{eqn 2}$$

where κ is the redistribution matrix and $\times$ denotes matrix multiplication. By increasing the growth rate, model 1 changes from stable through cyclic to chaotic dynamics (Fig. 1a). Parallel to the transition in dynamics, there is a marked change in metapopulation synchrony; locally stable populations reflect the correlation in the environment, as predicted by linear theory (Moran 1953; Lande, Engen & Sæther 1999; Ripa 2000). Cyclic populations, in contrast, are near perfectly synchronized reflecting the non-linear phase-locking. Chaotic populations exhibit very little synchrony; neither dispersal nor regional correlation synchronize the dynamics. In this way, the theory predicts a clean-cut relationship between patterns of dynamics and the propensity for synchronization (Bjørnstad *et al.* 1999a; Blasius *et al.* 1999; Ranta *et al.* 1999). Unfortunately for theoretical ecologists, theoretical ecology can proclaim clean-cut relationships, yet never be more than *possible*, *plausible* or *consistent*. For complex problems, such as the present, empirical corroboration is paramount.

Historical 'experiment' 1: vaccination and disease

Rohani *et al.* (1999) report a detailed comparative analysis of two childhood diseases – whooping cough and measles – and their pattern of synchrony before and after the development (and continual application) of successful vaccines. This study describes a grand-scale historical experiment with relevance to the theoretical debate. Together, these diseases represent an intriguing ecological contrast, since they are sympatric diseases transmitted with similar reproductive potential and pathology (Rohani *et al.* 1999). The details of transmission, however, are such that measles naturally undergo multi-annual cycles (with a period of 2 years) in large unvaccinated populations, while the incidence of whooping cough is more erratic (albeit with an important multi-annual component). Medical records testify that upon vaccination, the measles' cycle collapses onto seasonal and erratic dynamics. Whooping cough dynamics, in contrast, bifurcates into a 3·5-year oscillation (see Fig. 1 in Rohani *et al.* 1999). I have tentatively calculated the proportion of the variability in log(case-count) that can be 'explained' – in the loose sense of statistically captured – by multi-annual cyclicity (2–5-year cycles in the periodogram) for the two childhood diseases in the 10 biggest cities in Britain. The results are broken down by the prevaccination and the vaccination era (Table 1). The analyses mirror the divergent effect of vaccination; cyclic variability increases in whooping cough and decreases in measles during the vaccination era. Rohani *et al.* (1999) document that the transition in dynamics is associated with a marked shift in synchrony – a shift that is favourable to theoretical ecology: as measles collapse from cyclic dynamics, the synchrony in incidence across England and Wales declines significantly from 0·29 (CI = 0·24, 0·34) to 0·17 (CI = 0·14, 0·21). As the dynamics of whooping cough undergo the contrasting enhancement of cyclicity, the synchrony increase

Table 1. The proportion of the variability in weekly case-counts of two childhood diseases (Rohani *et al.* 1999) accounted for by multi-annual cycles (2–5 years), before (*Pre-Vacc*) and during vaccination era (*Vacc*) in UK. The proportions are calculated on the basis of the periodograms of log-transformed case counts. I only use data up to 1988 because incidences (particularly of measles) are very low after that. The numbers represent the average (standard error) across the 10 largest cities in England. Mass vaccination started in 1957 for whooping cough and in 1968 for measles. The whooping cough data span 13 years (1944–56) pre-vaccination and 32 years during the vaccination era (1957–88). The measles data span 25 years (1944–67) and 21 years (1968–88), respectively

	Proportion cyclic (2–5 years) variability	
	Pre-Vacc	Vacc
Whooping cough	29·0% (SE = 3·7%)	35·8% (SE = 2·0%)
Measles	48·2% (SE = 4·6%)	35·6 (SE = 2·6%)

from 0·14 (CI = 0·11, 0·18) to 0·25 (CI = 0·20, 0·29). Thus, the patterns of synchrony are near perfectly reversed through the historical vaccination treatment. Vaccination thus appears to provide an orthogonal treatment to investigate 'the effect of cyclicity' on population synchrony.

The transition in local dynamics are intriguing in their own right. Earn *et al.* (2000) use a seasonally forced SEIR model to show how vaccination can shift the measles dynamics from a region in parameter space where a biannual attractor is prevalent to an area with multiple and intertwined attractors. Stochasticity can thus shift different local populations onto different attractors so as to provide a quick route to asynchronization. In contrast, Rohani *et al.* (1999) show that a theoretically plausible transition in whooping cough dynamics is *not* one where a multi-annual attractor gains dominance. Rather the cycles are seen to arise through the interaction between stochasticity and an unstable attractor (see also Rand & Wilson 1991; Cushing *et al.* 1998).

Historical 'experiment' 2: rodent cycles

As was pointed out by one of the referees, I have used the term 'historical experiment' frivolously above: the data did not actually arise from a study with replication, randomization and control. The conclusions are therefore less robust than from an ideal experiment. In this spirit, it is natural to ask why nobody has carried out a proper experimental test of the predictions of the relationship between local dynamics and synchrony. A mundane answer – over and above the fact that spatial ecology has only recently regained fashion – is that few ecological funding-bodies can sustain the economic burden of the necessary spatiotemporal treatment. Furthermore, it may not even be possibly to conduct a randomised manipulation of cyclicity, since the appropriate scale may be *biogeographic* in extent (e.g. Bjørnstad, Stenseth & Saitoh 1999b). Such worry aside, the patterns observed in childhood diseases may carry generality. Steen *et al.* (1990) reported a study of the historical data of Norwegian rodent abundance as compiled by Wildhagen (1952). The data consist of binary observations – 1 for high abundance, 0 for low abundance – in three regions in Norway. Two of the regions are in Southern Norway. The last region, in Northern Norway, is geographically separate and shows somewhat different patterns (N.G. Yoccoz, personal communication). They investigated the effect of a countrywide predator extermination programme initiated around the turn of the century on rodent dynamics. Detailed statistical analysis of time-series testified that the result of this effort was a (temporary) loss of cyclicity in the rodents' dynamics (Steen *et al.* 1990). I have reanalysed the data reported in this study with respect to synchrony. I report the mean synchrony in the time series in the cyclic and the non-cyclic period in Table 2. The loss of cyclicity in the early part of the century is associated with a noticeable drop in regional synchrony. In fact, the synchrony in the two Southern regions drops significantly from nearly 0·9 in the cyclic periods to under 0·4 during the non-cyclic period.

An historical 'experience': rodent synchrony

The 'first generation' studies of synchrony used linear theory to predict that synchrony should mirror the correlation in the environment (Moran 1953; Royama 1992; Ranta *et al.* 1995). This theoretical benchmark suggested local and regional synchrony

Table 2. The Pearson correlation between the binary time series of rodent peaks in three regions of Norway (from Steen *et al.* 1990) broken down into the cyclic period and the intermittent non-cyclic period. The first row represents the mean correlation between all three regions (with 95% bootstrap confidence intervals; calculated by resampling with replacement among years). The second row represents the correlation between the two regions in Southern Norway

	Cyclic (1871–99, 1921–50)	Non-cyclic (1900–20)
Synchrony	0·72 {0·57, 0·84}	0·45 {0·18, 0·70}
Synchrony (Just Southern)	0·88 {0·76, 0·97}	0·37 {-0·05, 0·72}

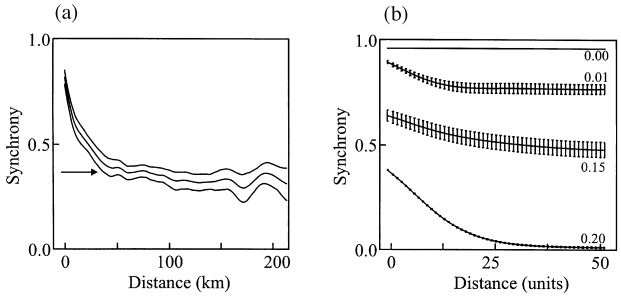

Fig. 2. (a) Population synchrony in the cyclic grey-sided vole of Hokkaido is initially high, and then levels off to regionwide synchrony of around 0·36 (arrow) (Bjørnstad *et al.* 1999b). The lines represent the non-paramatric covariance function with 95% confidence interval, and quantify how synchrony drops with distance (Bjørnstad *et al.* 1999a). (b) Correlation as a function of distance in a coupled map lattice with 100 populations coupled by nearest-neighbour dispersal (see Fig. 1). Local dynamics is assumed to follow stochastic Ricker dynamics (eqn 1) with $r = 3$, giving rise to local limit cycles. The lines represents mean correlation functions (with SE) across 20 replicate models (using 10 000 time steps; after a 10 000-step transient period), as estimated using the non-parametric covariance function (Bjørnstad *et al.* 1999a); The standard deviation of the environmental stochasticity is assumed to be uncorrelated between populations (i.e. no Moran effect). The top line represents deterministic runs, giving rise to perfect phase-locking where synchrony does not drop with distance. The three lower functions reflect successively higher levels of environmental variability ($\sigma = 0{\cdot}01$, 0·15 and 0·20). High levels of stochasticity erode the phase-locking altogether. However, intermediate levels of stochasticity trap the metapopulation at intermediate levels of synchrony – resembling the pattern produced by the Moran effect.

to arise from different processes; local synchrony due to dispersal or other local effects, and regional synchrony due to climatic forcing (e.g. Sutcliffe, Thomas & Moss 1996; Bjørnstad *et al.* 1999b). Bjørnstad *et al.* (1999b) studied the patterns of synchrony of the grey-sided vole (*Clethrionomys rufocanus* [Sundevall]) in Hokkaido, Japan (Fig. 2a). In light of the 'linear' theory, the regionwide synchrony (at around 0·36) was interpreted as due to climatic forcing. The local synchrony was interpreted as due to dispersal. However, since the rodents' own dispersal capabilities were found insufficient to account for the 50 km extent of the local synchrony, mobile predators were invoked as a tentative additional ingredient to the puzzle (Bjørnstad *et al.* 1999b; see also Ims & Steen 1990). Because the grey-sided vole undergoes multi-annual population cycles in parts of Hokkaido (Stenseth *et al.* 1996; Bjørnstad *et al.* 1998), I feel encouraged to revisit this speculation. I investigate whether the recent theoretical qualifications – showing how population cycles can enhance synchrony (Fig. 1) – can provide an alternative interpretation.

In Fig. 2b, I simulate an extended coupled map lattice that consists of 100 local populations that are connected by local dispersal (eqns 1–2) to see how synchrony is a function of distance in cyclic metapopulations. In this simulation there is no spatial correlation in stochasticity, so the Moran effect (*sensu* Ranta *et al.* 1995) does not operate. The local dynamics are assumed to be cyclic ($r = 3$; see Fig. 1a), but affected by environmental stochasticity. For low levels of stochasticity, nonlinear phase-locking induces regionwide synchrony (Fig. 2b). High levels of stochasticity, in contrast, erode the regional synchrony completely so that only local (dispersal-induced) synchrony remains. However, intermediate levels of environmental stochasticity traps the cyclic metapopulation at intermediate levels of regionwide synchrony. The only way to distinguish this cause of regional synchrony from the Moran effect will be to study patterns of local dynamics. Imperfect phase-locking may, for this reason, provide a viable hypothesis for regional synchrony in cyclic rodents.

Concluding speculations

A critical – but until recently unsupported – theoretical prediction from the 'second generation' studies of synchrony is that cyclicity promotes synchrony. The comparative study of the effect of vaccination on childhood diseases (Rohani *et al.* 1999) help bridge the gap between theoretical speculations and the world of data. A similar historical enquiry into rodent dynamics and synchrony (Steen *et al.* 1990) reaffirm the broad implications of the epidemiological study.

So, as an answer to the much-belaboured question of whether it is dispersal or correlated climate that induces regionwide synchrony in **cyclic** populations, theory now suggests that it is possibly either, but possibly neither – cyclic populations yearn to align themselves.

Acknowledgements

Pej Rohani gracefully allowed me access to the historical data on whooping cough and measles epidemics. Jordi Bascompte, Bryan T. Grenfell, Pej Rohani and Nigel G. Yoccoz provided valuable

comments on the manuscript. This work is supported by the National Center for Ecological Analysis and Synthesis (a Centre funded by NSF Grant #DEB-94–21535, the University of California Santa Barbara, and the State of California) and the Norwegian Science Foundation.

References

Bjørnstad, O.N., Ims, R.A. & Lambin, X. (1999a) Spatial population dynamics: analysing patterns and processes of population synchrony. *Trends in Ecology and Evolution*, **11**, 427–431.

Bjørnstad, O.N., Stenseth, N.C. & Saitoh, T. (1999b) Synchrony and scaling in dynamics of voles and mice in northern Japan. *Ecology*, **80**, 622–637.

Bjørnstad, O.N., Stenseth, N.C., Saitoh, T. & Lingjære, O.C. (1998) Mapping the regional transitions to cyclicity in *Clethrionomys rufocanus*: spectral densities and functional data analysis. *Researches on Population Ecology*, **40**, 77–84.

Blasius, B., Huppert, A. & Stone, L. (1999) Complex dynamics and phase synchronization in spatially extended ecological systems. *Nature*, **399**, 354–359.

Cushing, J.M., Dennis, B., Desharnais, R.A. & Costantino, R.F. (1998) Moving towards an unstable equilibrium: saddle nodes in population systems. *Journal of Animal Ecology*, **67**, 298–306.

Earn, D.J.D., Rohani, P., Bolker, B.M. & Grenfell, B.T. (2000) A simple model for complex dynamical transitions in epidemics. *Science*, **287**, 667–670.

Engen, S. & Lande, R. (1996) Population dynamic models generating the lognormal species abundance distribution. *Mathematical Biosciences*, **132**, 169–183.

Grenfell, B.T., Wilson, K., Finkenstädt, B.F., Coulson, T.N., Murray, S., Albon, S.D., Pemberton, J.M., Clutton-Brock, T.H. & Crawley, M.J. (1998) Noise and determinism in synchronised sheep dynamics. *Nature*, **394**, 674–677.

Ims, R.A. & Steen, H. (1990) Geographical synchrony in microtine population cycles: a theoretical evaluation of the role of nomadic avian predators. *Oikos*, **57**, 381–387.

Jansen, V.A.A. (1999) Phase locking: another cause of synchronicity in predator–prey systems. *Trends in Ecology & Evolution*, **14**, 278–279.

Kaitala, V., Ranta, E. & Lindström, J. (1996) External perturbations and cyclic dynamics in stable populations. *Annales Zoologica Fennici*, **33**, 275–282.

Koenig, W.D. (1999) Spatial autocorrelation of ecological phenomena. *Trends in Ecology and Evolution*, **14**, 22–26.

Lande, R., Engen, S. & Sæther, B.E. (1999) Spatial scale of population synchrony: Environmental correlation versus dispersal and density regulation. *American Naturalist*, **154**, 271–281.

Moran, P.A.P. (1953) The statistical analysis of the Canadian lynx cycle. II. Synchronization and meteorology. *Australian Journal of Zoology*, **1**, 291–298.

Nisbet, R.M. & Gurney, W.S.C. (1982). *Modelling Fluctuating Populations*. Wiley, Chichester.

Rand, D.A. & Wilson, H.B. (1991) Chaotic stochasticity: a ubiquitous source of unpredictability in epidemics. *Proceedings of Royal Society London B*, **246**, 179–184.

Ranta, E., Kaitala, V. & Lindström, J. (1999) Spatially autocorrelated disturbances and patterns in population synchrony. *Proceedings of the Royal Society Biological Sciences Series B*, **266**, 1851–1856.

Ranta, E., Kaitala, V., Lindström, J. & Lindén, H. (1995) Synchrony in population dynamics. *Proceedings of Royal Society London B*, **262**, 113–118.

Ranta, E., Kaitala, V. & Lundberg, P. (1998) Population variability in space and time: the dynamics of synchronous populations. *Oikos*, **83**, 376–382.

Ranta, E., Lindström, J., Kaitala, V., Kokko, H., Lindén, H. & Helle, E. (1997) Solar activity and hare dynamics: a cross-continental comparison. *American Naturalist*, **149**, 765–775.

Ripa, J. (2000) Analysing the Moran effect and dispersal: their significance and interaction in synchronous population dynamics. *Oikos*, **89**, 175–187.

Rohani, P., Earn, D.J. & Grenfell, B.T. (1999) Opposite patterns of synchrony in sympatric disease metapopulations. *Science*, **286**, 968–971.

Royama, T. (1992). *Analytical Population Dynamics*. Chapman & Hall, London.

Steen, H., Yoccoz, N.G. & Ims, R.A. (1990) Predators and small rodent cycles: an analysis of a 79-year time series of small rodent population fluctuations. *Oikos*, **59**, 115–120.

Stenseth, N.C., Bjørnstad, O.N. & Saitoh, T. (1996) A gradient from stable to cyclic populations of *Clethrionomys rufocanus* in Hokkaido, Japan. *Proceedings of Royal Society London B*, **263**, 1117–1126.

Sutcliffe, O.L., Thomas, C.D. & Moss, D. (1996) Spatial synchrony and asynchrony in butterfly population dynamics. *Journal of Animal Ecology*, **65**, 85–95.

Tilman, D. & Kareiva, P., eds. (1997). *Spatial Ecology: the Role of Space in Population Dynamics and Interspecific Interactions*. Princeton University Press, Princeton.

Wildhagen, A. (1952). *Om Vekslingene I Bestanden Av Smågnagere I Norge 1871–1949*. Statens viltundersøkelser, Drammen. (in Norwegian).

Received 23 December 1999; revision received 27 March 2000

Journal of Animal Ecology 2001
70, 353

Erratum

Bjørnstad, O.N. (2000) Cycles and synchrony: two historical 'experiments' and one experience. *Journal of Animal Ecology*, **69**, 869–873.

The following figure rectifies an error in Fig. 1 of Bjørnstad (2000). While all the qualitative results remain, errors in the *x*- and *y*-axes scale are corrected.

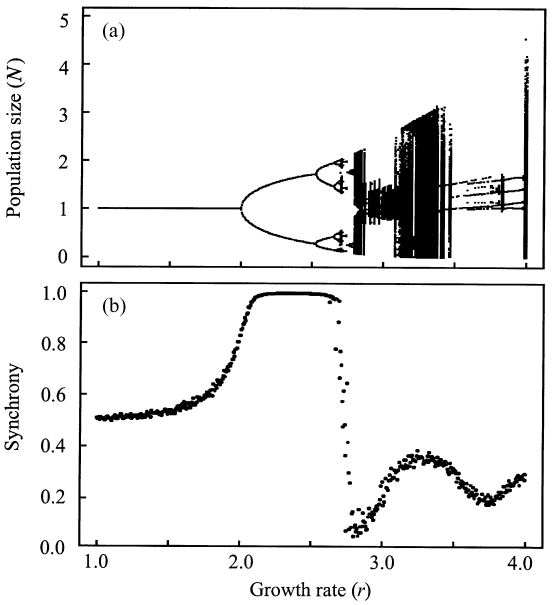

Fig. 1. Simulation of 10 populations on a linear coupled lattice map. The local populations are coupled by nearest-neighbour dispersal (50% of the population are allowed to disperse evenly to the two neighbouring populations; the map is assumed circular to avoid boundary effects). Local dynamics are assumed to follow Ricker dynamics: $N_{t+1} = N_t \exp[r(1 - N_t) + u]$, where N is population size, r is growth rate, and u is normal random variate with zero mean and standard deviation, σ, representing environmental stochasticity. (a) Bifurcation diagram for the deterministic system ($\sigma = 0$) as the growth rate is changed. The population size represents the average population size across the 10 local populations (after allowing for a 500-generation transient period). (b) Average level of synchrony among the 10 local populations (after allowing for a transient period) when the populations are further affected by correlated environmental stochasticity ($\sigma = 0{\cdot}1$, spatial correlation = 0·3) as the growth rate is tuned to change the dynamics. The near perfect correlation for the cyclic dynamics is due to non-linear phase locking. Locally stable populations are slightly higher than the regional synchrony (due to the added effect of dispersal), as predicted by linear theory. Chaotic populations exhibit very little synchrony; neither dispersal nor regional correlation synchronize the dynamics.