no doubt be more widely substituted for the disappearing commercial caviar species. At present, these commercial species are not included in the appendices of the Convention on International Trade in Endangered Species of Wild Fauna and Flora (CITES), the only international agreement on endangered species trade. CITES will consider placing all sturgeon species in the appendices at a meeting (23–27 September 1996) in Pruhonice, Czech Republic. The enforcement of such a listing depends largely on the ability to diagnose these species accurately. Our method will give wholesalers an alternative to the crude identification methods they use now and give consumer groups a weapon to ensure accurate labelling.

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# Red/blue chaotic power spectra

SIR — Cohen[1] has reported that the time series of chaotic, single-species ecological models have blue power spectra rather than the reddened ones associated with natural populations[2]. In other words, the dynamic behaviour of real populations is apparently dominated by longer-term trends, but population models, of one common type at least, fail to capture this crucial characteristic, being dominated instead by shorter-term responses. This calls into question both the usefulness of the models and the applicability of chaotic dynamics to natural systems. However, equivalent analyses (see Fig. 1) of chaotic metapopulation time series from explicitly spatial models (comprising coupled-map-lattices of $n \times n$ identical patches, describing host–parasitoid[3] or host–pathogen[4] dynamics, with the eight nearest-neighbouring patches linked by dispersal) give rise to distinctly reddened spectra — like the natural population data, but quite unlike the simpler models.

Note that the two-dimensional models within patches are dominated by short-term responses to population density (governed by 'biotic' mechanisms). This tends to undermine any suggestion[1,2] that the important contrast is necessarily between systems dominated by biotic factors (blue) and external, 'climatic' factors (red). Moreover, similar spectral analyses of chaotic but non-spatial two-dimensional models[5,6] still give rise to blue spectra. Thus, we judge that including the spatial dimension is crucial in reddening our power spectra, and that those of natural populations may still be explained (at least partly) by chaotic dynamics.

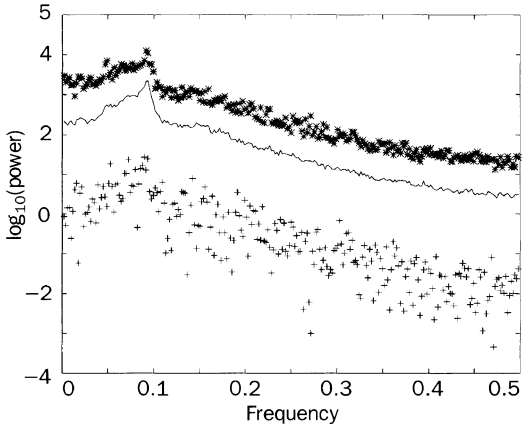


FIG. 1 Power spectral densities of the metapopulation of hosts in a spatially explicit host–parasitoid model[3] (similar portraits are observed for the host–pathogen model[4]). Parameter values, taken from ref. 4, are $r = 2$, $\sigma_1 = 0$, $\sigma_2 = 1$, $\Lambda = 1$, $\nu = 1$, $\mu_X = 0.2$, $\mu_W = 0.89$ and $n = 30$; similar results are obtained for larger values of $n$. In common with natural populations, the spectra are reddened, with greater power at low frequencies. (Scale on axes was chosen to allow comparison with ref. 1.)

We can also deduce why, biologically, space can produce reddened spectra. In the spatially explicit lattice, subpopulations respond quickly only to the density in their immediate neighbourhood. Hence, the concerted response of the whole metapopulation to its own total density can occur only on much longer timescales that allow initially local effects to spread and to influence the whole. This echoes, and puts flesh on, the complaints of empiricists that population density may be uninformative because individuals experience only local crowding.

Chaotic population dynamics, when generated by spatially explicit systems, may be important in explaining the reddened spectra seen in natural populations.

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SIR — We wish to add to the debate about colour in the power spectra of the complex dynamics of natural poplulations[1,2,7,8]. Power spectra of population trajectories are observed to be white (no dominating frequency) or red (dominance of low-frequency fluctuations[2,9]). Thus, low frequencies might be expected to dominate in population models producing chaos-like oscillations. But Cohen showed in various nonlinear models that high frequencies dominate in their chaotic fluctuations[1], making the power spectra blue. Here we report that adding delayed density dependence[10–12] to these population models reduces the dominance of high-frequency oscillations and either whitens or reddens their power spectra.

In a non-delayed density-dependent system, the population size $P_t$ at time $t$ defines the population size at time $t+1$ with a given growth rate $r$. We analysed the power spectra of chaotic trajectories of six of the eight models discussed by Cohen[1] by incorporating delayed density dependence into their dynamics. As an example, the Moran–Ricker discrete-time nonlinear dynamics[13,14], $P_{t+1} = P_t \exp[r(1 - P_t)]$, modifies into $P_{t+1} = P_t \exp\{r[1 - (P_t + cP_{t-1})]\}$. Of the models analysed by Cohen, we omitted the Verhulst model, known to produce white noise[1], and the Malthus– Condorcet–Mill model, in which delayed density dependence creates typically isolated spikes in spectra.

It is generally expected that the addition of time delays in population models will lower the stability of the dynamics. However, the consequences of delayed density dependence for the colour of the power spectra of chaotic population dynamics are not known. Adding delayed density dependence in the models can remove the dominance of high-frequency oscillations and can whiten or redden their power spectra (see Fig. 2), which brings them better into line with data from natural populations[9].

Simple population models have drawn attention to the possibility of complex dynamics in natural populations[7,8], but they may be of limited use in explaining the rich phenomena observed in dynamics of natural populations. Chaos in models of natural populations can be obtained in several ways, including periodic doubling (used in simple models[1]), quasiperiodicity and intermittency[15,16]. Depending on the way in which chaos is obtained, there may be fundamental differences in the power spectra of the population trajectories. For example, the quasiperiodic route, which strongly affects the power spectra of the population trajectory, occurs often in the context of species or population interactions, or in age–class interactions in a single population. Delayed density dependence may also produce the quasiperiodic route to chaos. Thus, when adding delayed density dependence to population models, we not only altered the assumptions on the density dependence, but also increased the dimension of the dynamics and moved to

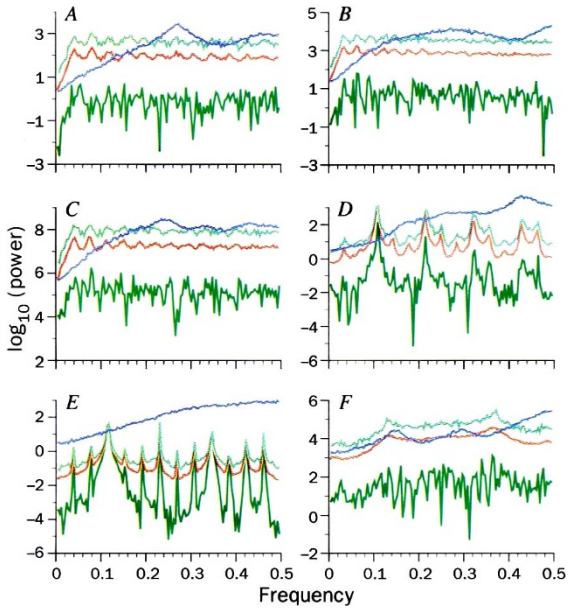


FIG. 2 Power spectral densities (red line, average of 100 simulations; light and dark green lines, maximum and minimum values observed) of six models with delayed density dependence. Panel *A*, Moran–Ricker: $P_{t+1} = P_t \exp\{r[1 - (P_t + cP_{t-1})]\}$; $r = 3.7$, $c = 6$. Panel *B*, Pennycuick: $P_{t+1} = rP_t/\{1 + \exp[-a(1 - (P_t + cP_{t-1})/b)]\}$; $r = 50$, $a = b = 0.1$, $c = 8$. Panel *C*, Hassell: $P_{t+1} = rP_t/[1 + a(P_t + cP_{t-1})]^b$; $r = 55$, $a = 0.0001$, $b = 100$, $c = 8$. Panel *D*, Maynard Smith–Slatkin: $P_{t+1} = rP_t/\{1 + [a(P_t + cP_{t-1})]^b\}$; $r = 5$, $a = 0.5$, $b = 4$, $c = 18$. Panel *E*, Varley: $P_{t+1} = rP_t$ if $(P_t + cP_{t-1}) \le C$, $P_{t+1} = rP_t(P_t + cP_{t-1})^{-b}$ if $(P_t + cP_{t-1}) > C$; $r = 4$, $b = 3$, $C = 1$, $c = 43$. Panel *F*, Austin–Brewer: $P_{t+1} = P_t\{1 + r[1 - \exp(-s(P_t + cP_{t-1}))][K - (P_t + cP_{t-1})]\}$; $r = 0.06$, $s = 0.13$, $K = 45$, $c = 0.011$. By contrast with the earlier results[1] (blue line), low-frequency oscillations dominate in the models. In each run, the model was simulated for 512 generations to remove the initial transient from the time series. Then the next 512 values were subjected to fast Fourier transform and power-spectrum computations. For ease of comparison, we restricted the parameter values to those used by Cohen[1], adding only the delayed density dependence (the patterns presented in panels *D* and *E* can be modified to match those in the other panels more closely by changing parameter values).

a qualitatively different route to chaos. At the same time, we inherited chaotic solutions with different properties and, in particular, different, reddened, power spectra.

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SIR — Both the above suggestions are interesting and plausible extensions of classical models of population change. Both can, in principle, redden the power spectra in a way that matches observations from nature, without invoking external forcing by climate or other factors. Both are important theoretical results. But, in the case of the Chinese locusts[2,17], it seems equally likely that Ma's hypothesis involving climate effects is the main cause of the higher variability seen at the low frequencies there.

All this discussion exemplifies the difficulty of the debate over whether natural population fluctuations represent regulation by internal biological mechanisms or whether they are mostly the result of external environmental forcing[1,2]. Furthermore, it points to the unlikelihood that this classical dilemma between the climate and biotic schools of population regulation will ever be resolved by any simple (for example, linear) study of the phenomenology of population change. Rather, I believe that this debate points to the need to look deeper: either to more fundamental studies of the basic biology involved, or to sharper quantitative methods that can expose the specific biological mechanisms involved in generating a complex population time series.

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# Radiation doses from Ural region

SIR — Electron paramagnetic resonance (EPR) using tooth enamel is a well-known method for measuring external exposure to γ-rays retrospectively. This method is based on determining the concentration of radiation-induced radicals in hydroxyapatite, a mineral component of teeth and bones. It has already been used to deduce the exposure doses of survivors of the atomic bombs of Hiroshima and Nagasaki[1], and of the Chernobyl accident[1], as well as for monitoring doses to nuclear workers in the south Urals[2]. EPR dosimetry has also been proposed for measuring doses of bone-seeking radiopharmaceuticals[3]. Nevertheless, EPR tooth-enamel dosimetry has not previously been applied to the reconstruction of doses from a combination of internal and external exposure.

Since 1948, the population of the Techa river valley in the Urals has been exposed to a massive amount of waste from the Mayak nuclear facility near the source of this river. During 1949–51, about $76 \times 10^6$ $m^3$ of radioactive waste water containing a total activity of $10^{17}$ Bq was discharged into the Techa river. Strontium-90 ($^{90}Sr$) and caesium-137 ($^{137}Cs$) respectively contributed 11.6 and 12.2% to the radioactivity. Consumption of the river water caused considerable internal overexposure of the population of the Techa river valley and an excessive incidence of leukaemia[4].

Dose reconstruction for the Ural region is very important for epidemiological studies. $^{90}Sr$ substitutes for calcium in bone tissues and can accumulate in the body over a long period; its biological half-life is about 20 years. Around 28,000 people lived on the Techa river banks during the period of this massive release. About 4,500 of them were living in the upper Techa riverside, where the level of internal and external exposure was very high. Individual doses due to $^{90}Sr$ can be estimated from measurements of body burdens using a whole-body counter[5]. During the 1970s, individual measurements of $^{90}Sr$ in the skeleton were carried out on 12,248 people (about half the population living in this region during 1949–52). Many people have been examined up to 35 times. The maximum value of the $^{90}Sr$ body burden found for one individual is 0.23 MBq (6,206 nCi). However, there are no reliable data about individual doses resulting from external exposure.

We report here two new approaches to dose reconstruction, which we have applied to survivors of this radioactive exposure in a pilot study.

The first approach is based on the fact that the tooth enamel of an adult human is an unchanging, highly mineralized tissue (97% hydroxyapatite), which does not metabolize $^{90}Sr$, whereas dentine, which