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# Chaos and the dynamics of biological populations

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As first emphasized in the early 1970s, the nonlinearities that are inherent in simple models for the regulation of plant and animal populations can lead to chaotic dynamics. This review deals with a variety of instances where chaotic phenomena can arise, particularly in interactions between prey and predators (including hosts and pathogens, hosts and parasitic insects, and harvested populations). Some of the complications in disentangling deterministic chaos from environmental noise will be discussed. The combination of population biology with population genetics leads to an even richer assortment of nonlinear phenomena and to the suggestion that many genetic polymorphisms may vary cyclically or chaotically (rather than being steady, as usually is assumed implicitly).

I argue that complex dynamics – including chaos – is likely to be pervasive in population biology and population genetics, even in seemingly simple situations. But superimposed environmental noise, in heterogeneous natural settings, will usually complicate the dynamics, making it unlikely that population data will exhibit elegant properties (such as universalities in period doubling) associated with the underlying maps. The existence of chaotic régimes of dynamical behaviour can, however, invalidate standard techniques for analysing population data to reveal density-dependent mechanisms; this, I believe, may currently be the most significant implication of dynamical chaos for population biology.

## 1. INTRODUCTION

A central task for population biologists is to disentangle, from the superimposed fluctuations caused by environmental noise and other chance events, the underlying mechanisms that regulate natural populations so that no one species of plant or animal increases without bound. Such studies lead us to consider simple equations that might describe the dynamics of natural populations if environmental noise and heterogeneity could be stripped away. A clear understanding of the dynamics of these simple and deterministic, but nonlinear, models then serves as a point of departure for evaluating the effects of various kinds of complications associated with environmental unpredictability and heterogeneity.

As is by now well known, such investigations in the early 1970s led to the realization that the simplest nonlinear models for populations with discrete, non-overlapping generations (first-order difference equations with one critical point) could exhibit a surprising array of dynamical behaviour (May 1974, 1976; Li & Yorke 1975; May & Oster 1976). Subsequent work showed that even richer dynamical behaviour could be generated by simple, deterministic equations for single populations with discrete but overlapping generations (higher-order

difference equations), for single populations with continuous growth where regulatory effects contain time lags (time-delayed differential equations), and for two or more interacting populations. The dynamical properties of these models have been the subject of several recent reviews (Rogers 1981; Olsen & Degn 1985; Kloeden & Mees 1985; Lauwerier 1986*a*, *b*; May 1983, 1986). Section 2 therefore does not attempt a comprehensive review, but rather is a guide to the existing literature with selective emphasis on a few points that are new or are not widely appreciated.

Given that chaos arises in the simplest equations propounded by 'muddy-boots' ecologists as natural descriptions of the underlying dynamics of their insect, fish, pathogen or other populations, the question of to what extent are chaotic dynamics actually observed arises. Section 3 summarizes recent studies of this question. My conclusion is that, in controlled laboratory settings, the array of dynamics from stable points, to stable cycles, to chaos can be seen, but that even in these artificial situations one cannot hope to see fine details of period doubling and the like (as one arguably can in some physical contexts, such as the onset of turbulence). In the natural world, the role of nonlinear phenomena (including possibly chaos) in the dynamics of many infectious diseases of humans and other animals is being understood in an increasingly explicit way. But for most natural populations, I believe environmental noise and other complications make it difficult to find examples of time series that show period doubling, intermittency, transitions to chaos, and other dynamical features that are clearly exhibited in some physiological and biochemical systems.

The fact that chaotic dynamics can arise from simple, density-dependent mechanisms does, however, have profoundly important implications for the way population biologists analyse data. Most existing work is based, usually implicitly, on the assumption that if density-dependent 'signals' could be dissociated from the confounding environmental noise, the population would be regulated to a steady, constant value. But if deterministic nonlinearities actually give chaotic time series which are effectively indistinguishable from stochastic fluctuations, the task of uncovering the regulatory signal can be much more complex. Some current work on this subject is reviewed in §4.

Ultimately, environmental noise does not act on populations as such, but on their constituent individuals. Thus we really need to derive deterministic models for the dynamics of populations from assumptions about the behaviour of individuals, so that the parameters in the population model derive from the biology of individuals. The effects of environmental noise can then be introduced in the proper way, through their effects on individuals. When this is done for insect populations in patchy environments, preliminary studies show that the interplay among nonlinear dynamics (giving rise possibly to cycles and chaos), spatial heterogeneity and environmental noise can invalidate standard techniques for detecting density-dependent mechanisms in natural populations. This work is also reviewed in §4, and it may represent the most significant implication that nonlinear dynamics holds for population biologists.

Section 5 extends the discussion to the dynamics of gene frequencies in populations where fitness functions are derived from ecological considerations (and

thus can be frequency- or density-dependent). Again §5 is a brief outline of existing work. The conclusion is that the combination of population biology with population genetics can lead to a very rich assortment of nonlinear phenomena, with the implication that many genetic polymorphisms may vary cyclically or chaotically. Section 6 re-emphasizes the main messages in this review.

## 2. Chaotic dynamics in simple ecological models

### 2.1. *One-dimensional maps (single populations)*

Most readers will by now be familiar with the dynamical behaviour exhibited by the quadratic map,

$$x_{t+1} = ax_t(1-x_t). \tag{2.1}$$

If $3 > a > 1$, the fixed point at $x^* = 1-1/a$ is an attractor, and the system settles to the stable point made familiar by countless discussions in elementary mathematics courses. At $a = 3$ the system bifurcates, to give a cycle of period 2, which is stable for $1+\sqrt{6} > a > 3$. As $a$ increases beyond this, successive bifurcations give rise to a cascade of period doublings, producing cycles of periods 2, 4, 8, 16, ..., $2^n$ for $a$ in the range $3.570.. > a > 3$. Beyond the point of accumulation of this cascade, $4 > a > 3.570..$, there lies an apparently chaotic régime, in which trajectories look like the sample functions of random processes. In detail, the apparently chaotic régime comprises infinitely many tiny windows of $a$-values, in which basic cycles of period $k$ are born stable (accompanied by unstable twins), cascade down through their period-doublings to give stable harmonics of periods $k \times 2^n$, and become unstable; this sequence of events recapitulates the process seen more clearly for the basic fixed point of period 1. The details of these processes, and catalogues of the various basic $k$-cycles, have been given independently several times and are reviewed by May (1976), Collet & Eckmann (1980), and others.

The nature of the chaotic régime for such 'maps of the interval' is often misunderstood. In detail, the chaotic régime is largely a mosaic of stable cycles, one giving way to another with kaleidoscopic rapidity as $a$ increases. But for essentially all practical applications, the chaotic region has the effectively random character that superficial inspection or numerical simulations suggest. This point is exemplified by the 'Lyapunov exponent' that is often computed as an index of chaotic behaviour. These exponents are analogous to the eigenvalues that characterize the stability properties of simpler systems. They are typically calculated by iterating difference equations, such as (2.1), and calculating the geometric average value of the slope of the map at each iterate: that is, for the difference equation

$$x_{t+1} = F(x_t), \tag{2.2}$$

the Lyapunov exponent $\lambda$ is given by

$$\ln \lambda = \lim_{n\to\infty} \left\{ \frac{1}{n} \sum_{t=0}^{n} \ln \left( \mathrm{d}F(x_t)/\mathrm{d}x \right) \right\}. \tag{2.3}$$

For generically quadratic maps, there are unique attractors for most values of $a$ in the chaotic régime. Therefore this calculation, if carried out exactly, or if the

iterations are carried on long enough, will give values of $\lambda$ less then unity ($\ln\lambda$ negative); an exact plot of the Lyapunov exponent for increasing $a$ in the chaotic régime would be a hopeless jumble of ink lines, connecting the negative values (which arise for most values of $a$) to the set of positive values (which do have positive measure). But if only several tens of thousands of iterates are taken in numerical studies, $\ln\lambda$ is typically found to be positive in the chaotic régime (because transients take enormous times to die away for the very high-order cycles that predominate). Although inexact in a strictly mathematical sense, the 'chaotic' impression ($\ln\lambda > 0$) given by these numerical studies is probably more accurate for practical application than exact calculations ($\ln\lambda < 0$) would be! Thus the work on Lyapunov exponents reviewed by Olsen & Degn (1985) may be mathematically inaccurate (as pointed out by Gambaudo & Tresser (1983) and Kloeden & Mees (1985)), but it is usually correct in spirit.

The properties of exhibiting a stable point, or a cascade of period-doublings, or apparently chaotic dynamics are not peculiar to the quadratic map of (2.1), but are general to essentially all maps with one hump. Table 1 catalogues several such

TABLE 1. SOME FIRST-ORDER DIFFERENCE EQUATIONS, $x_{t+1} = F(x_t)$, TAKEN FROM THE BIOLOGICAL LITERATURE, WHICH CAN EXHIBIT CHAOTIC DYNAMICS

| $F(x)$ | source |
|---|---|
| $x\exp[r(1-x)]$ | Moran (1950), Ricker (1954), Macfadyen (1963), Cook (1965), Pacala & Silander (1985) |
| $x[1+r(1-x)]$ | Maynard Smith (1986), May (1972), Li & Yorke (1975) |
| $\lambda x$, if $x<1$<br>$\lambda x^{1-b}$, if $x>1$ | Haldane (1953), Varley *et al.* (1973) and references therein |
| $\lambda x/(1+ax^b)$ | Maynard Smith (1974), Bellows (1981) |
| $\lambda x(1+ax)^{-b}$ | Hassell (1974) |
| $x[1/(a+bx)-\sigma]$ | Utida (1957) |
| $\lambda_+ x$ if $x<1$<br>$\lambda_- x$, if $x>1$ | Williamson (1974), with $\lambda_+ > 1$, $\lambda_- < 1$ |
| $\lambda x[1-I(x)]$ | May (1985), with $I(x)$ given by $1-I = \exp(-Ix)$ |
| $\lambda x\,e^{-x}\sum_{i=0}^{\infty}\frac{x^i}{i!(1+\alpha i)}$ | Pacala & Silander (1985), Crawley & May (1987) |
| $\frac{\lambda x}{(1+ax)^b+cx}$ | Watkinson (1980) |

first-order difference equations that have been proposed, in various theoretical and empirical contexts, as descriptions of biological populations. The basic mechanisms producing this array of behaviour in one-dimensional maps can, moreover, be understood in a very simple way, by using geometrical, combinatorial, or other approaches (May 1976). In particular, the generic process whereby period-doubling occurs, with a stable orbit of period $k\times 2^{n+1}$ appearing as the basic $k$-cycle harmonic of period $k\times 2^n$ becomes unstable, can be understood by a simple geometrical argument. This argument also gives an analytical estimate of the

Feigenbaum ratio as $\delta = 2(\sqrt{2}+1) \approx 4.83$ (obtained by approximating the period-$k \times 2^{n+1}$ map by a cubic in the neighbourhood of the fixed points of period $k \times 2^n$ at their transition from stability to instability: May & Oster (1980)). This is in good agreement (up to terms of relative order $\delta^{-2}$) with the exact numerical computation $\delta = 4.669\ldots$.

The above remarks apply to maps that are generically quadratic in the sense that they have negative Schwarzian derivative (Singer 1978). In the absence of this restriction, we could have one-hump maps with, for example, a narrow range of attraction around the fixed point, but with the rest of the map generating very long chaotic transients which perpetuated until, by chance, an iterate fell within the range of attraction and settled to the fixed point. Such a system essentially has two different states, one stable state and one labile state (often called monostable); the example might be called monostable chaos. Alternatively, as discussed in more detail by Olsen & Degn (1985), it can be that the slope of the map in the neighbourhood of the fixed point is slightly below $-1$, with most of the rest of the map generating chaotic trajectories. The fixed point is then only weakly unstable, so that it takes many iterations to leave its neighbourhood; once the iterate has, however, left this neighbourhood, it can abruptly become fully chaotic, only to get caught in the quasistable neighbourhood of the fixed point again, sooner or later. Such alternation between an almost stationary state and chaotic fluctuations, repeated at apparently random intervals, is called intermittency. Intermittency does not arise for any of the maps listed in table 1, nor have population biologists given much thought to the phenomena. Its possible relevance in ecological contexts deserves more attention.

### 2.2. *A speculation about the history of the subject*

Given that simple equations, which arise naturally in many contexts, generate such surprising dynamics, it is interesting to ask why it took so long for chaos to move to centre stage the way it has over the past ten years or so. I think the answer is partly that widespread appreciation of the significance of chaos had to wait until it was found by people looking at systems simple enough for generalities to be perceived, in contexts with practical applications in mind, and in a time when computers made numerical studies easy.

Individually, the first two of these conditions were met long ago. Thus Poincaré found strange attractors in his studies of planetary dynamics, and he appreciated their significance, but these applications were sufficiently complicated that each could appear *sui generis*. Many concluded that, in nonlinear systems, each application is special, with no general messages. Even Lorenz's (1963) beautiful example of chaos in a simple system of three ordinary differential equations is complicated enough to have resisted a fairly full analysis until relatively recently. First-order difference equations, such as those in table 1, are indeed simple enough for a fairly complete understanding of their range of behaviour to be obtained, and several people (starting with Myrberg (1962) and Sharkovsky (1964)) did just that. But these earlier investigators were primarily interested in the exquisite mathematics, and do not seem to have had any messianic sense of the wider implications of their work.

In the 1940s and 1950s, several population biologists studied simple difference equations as models for practical problems: Moran (1950) in a entomological context; Ricker (1954) as a model for recruitment in fisheries. Their numerical studies uncovered stable points, stable cycles and even chaos. These people, however, were mainly interested in stable solutions, and they did not pursue the chaotic dynamics they found. Perhaps they distrusted the chaotic trajectories, as possible artifacts of their mechanical calculators.

In the studies of Moran, Ricker and other population biologists one had the conjunction of simple systems being studied with practical problems in view, but not my third conjectured ingredient of fast and reliable computers. All three ingredients did come together in the early 1970s, in work motivated by simple problems in population biology.

### 2.3. *A 'completely chaotic' example*

One particular example, which might have stimulated work on chaotic dynamics at an earlier date had it been studied earlier, arises as a simple and natural model for an insect population with discrete, non-overlapping generations that is regulated by a lethal pathogen which spreads in epidemic fashion through each generation, before reproduction. This system has recently been studied by May (1985) (see also Rogers *et al.* 1986). If the population increases by a factor $\lambda$ from generation to generation in the absence of the pathogen, the population in generation $t+1$, $N_{t+1}$, is related to that in generation $t$ by

$$N_{t+1} = \lambda N_t[1-I(N_t)]. \tag{2.4}$$

Here $I(N_t)$ is the fraction infected, and thus killed before reproducing. The Kermack–McKendrick (1927) equation may be used to find the total fraction infected when an epidemic spreads through a population of magnitude $N_t$:

$$1-I = \exp(-IN_t/N_T). \tag{2.5}$$

Here $N_T$ is the threshold population size, which depends on the transmissibility and virulence of the pathogen: if $N_t < N_T$, the epidemic cannot spread, and $I = 0$; if $N_t > N_T$, the epidemic can spread, so that $I \neq 0$ and the effective reproductive rate is below $\lambda$. The one-dimensional map generated by this model has no stable points, and no stable cycles. As illustrated in figure 1, the system is 'completely chaotic', with an invariant measure for all values of $\lambda$ ($\lambda > 1$). For a more detailed discussion, see May (1985) and Rogers *et al.* (1986).

The models for regulation of host insects by parasitoids, which are discussed below in §2.6, were developed in the 1920s and 1930s. They are very similar in spirit to (2.4) for regulation by a pathogen, except they do possess stable points and/or simple cycles among their possible dynamical behaviour. I have previously speculated that, had (2.4) been studied earlier, its completely chaotic dynamics might have forced population biologists to acknowledge the existence of chaotic dynamics much sooner.

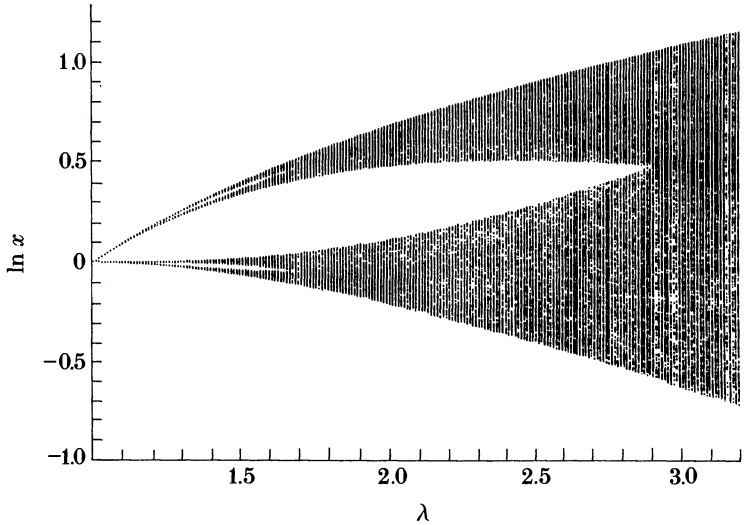


FIGURE 1. Plot of population values (on a logarithmic scale, $\ln(N/N_T)$) generated by iterating (2.4) many times, for each of a sequence of $\lambda$-values. The diagram gives an impression of the probability distribution of population values generated by this purely deterministic difference equation (after May 1985).

### 2.4. *Higher-dimension systems of difference equations*

The first-order difference equations listed in table 1 are metaphors for underlying density-dependencies in mechanisms regulating natural populations. Such regulatory effects, however, will often themselves contain time lags. This leads us to consider, as one of the simplest examples of such effects, the time-delayed logistic equation (Maynard Smith 1968):

$$x_{t+1} = \lambda x_t(1-x_{t-1}). \tag{2.6}$$

Such an equation can be reformulated as a pair of first-order difference equations:

$$\left.\begin{aligned} x_{t+1} &= \lambda x_t(1-y_t), \\ y_{t+1} &= x_t. \end{aligned}\right\} \tag{2.7}$$

In this formulation, the dynamics may be followed by plotting pairs of points $(x_t, y_t)$ in a two-dimensional phase plane; in the example (2.7), sensible trajectories are restricted to lie within the unit square (negative values $x_t$ or $y_t$ being taken to correspond to extinction). The system defined by (2.7) is a special case of more general classes of prey–predator relationships.

Equations (2.6) and (2.7) have been studied by Pounder & Rogers (1980), Aronson *et al.* (1982) and Rogers & Clarke (1981). Pounder & Rogers show the trajectories of points $x_t$, $y_t$ are attracted to an invariant curve, which has an extremely complicated shape. Very briefly, this curve has infinitely many loops or folds issuing from the origin; the loops are successive images of the bottom arc of the curve. Once $\lambda$ exceeds a critical value, the bottom arc of the curve can cross the $x$-axis, enabling the system to 'escape' to negative values (corresponding,

biologically, to extinction). Interior loops of the invariant curve can also lead to 'escaping' trajectories, and extinction. The net result is that, for a range of $\lambda$-values, there are initial values which lead to extinction (possibly after very long times), while others can lead to cycles of high period or to chaotic fluctuations; the phase plane has a complicated filamentary structure, in which arbitrarily close initial points can undergo qualitatively different fates (exhibiting what Yorke has called 'fractal basin boundaries').

Rogers (1981) gives a review of this work, and Lauwerier (1986*b*) has presented an exceedingly lucid analysis of a range of related two-dimensional systems, including a generalized form of (2.7):

$$\left.\begin{aligned} x_{t+1} &= \lambda x_t[1-by_t-(1-b)x_t], \\ y_{t+1} &= x_t. \end{aligned}\right\} \tag{2.8}$$

Not surprisingly, these more general forms can have an even richer spectrum of dynamical behaviour than (2.7).

Such models lead into other two-dimensional systems, corresponding to interacting prey and predator populations with discrete, non-overlapping generations. One of the earliests such systems was propounded by the parasitologist Crofton (1971) as a description of certain kinds of host–parasite interactions:

$$\left.\begin{aligned} x_{t+1} &= \lambda x_t[1+y_t]^{-k}, \\ y_{t+1} &= x_t y_t[1+y_t]^{-k-1}. \end{aligned}\right\} \tag{2.9}$$

Preliminary analytical and numerical studies of this system by May (1979) reveal a fixed point, which attracts initial points lying along 'spiral arms' around it in phase space, with other initial points leading to high-order cycles or to apparent extinction. The above-described analyses of the delayed logistic equation help explain these earlier observations. Lauwerier's (1986*b*) review extends to a variety of discrete prey–predator systems of this general kind, and he shows the complex dynamics and extreme sensitivity to initial conditions that can ensue.

### 2.5. *Host–parasitoid interactions*

Roughly 10% of all metazoan species are insect parasitoids. These hymenopteran or dipteran species oviposit on or in their hosts (usually the egg, larval, pupal or adult stage of an insect, often a lepidopteran). Mathematical studies of the dynamics of such systems have gone hand-in-hand with empirical studies since the early work of Nicholson and Bailey in the 1930s (Hassell 1978). This is, in part, because each host produces either another host or a parasitoid in the next generation, which means that relatively realistic models have a simple structure

$$x_{t+1} = \lambda x_t f(y_t), \tag{2.10a}$$

$$y_{t+1} = x_t - x_{t+1}/\lambda. \tag{2.10b}$$

Here $x$ and $y$ represent the population densities of hosts and parasitoids, respectively. Each host either escapes parasitoid attack (with probability $f$, here assumed to depend only on $y$) and then produces $\lambda$ progeny, or else is parasitized to produce one parasitoid in the next generation. A variety of forms have been

propounded, in the entomological literature, for the 'search function' $f(y)$; some of these are reviewed by Lauwerier (1986*b*), and to his list should be added the 'negative binomial' form $f(y) = [1+ay/k]^{-k}$.

The dynamics of the system (2.10) will depend on $\lambda$ and on one or more other parameters characterizing $f(y)$. In general, there can be a stable fixed point, which can undergo Hopf bifurcation to produce stable cycles. An interesting result which is little known among biological workers in this area, yet which explains the results of many numerical studies, is that the functional form of (2.10) generally implies the stable periodic solutions have approximate periods of at least six generations. This result is presented by Lauwerier (1986*b*), and illustrated with numerical studies of several such systems (some of which have very complicated dynamics, including, for example, a stable inner seven-point cycle and an unstable outer cycle surrounding the unstable fixed point). Lauwerier's derivation, however, contains a minor error; Appendix A sketches the analysis.

### 2.6. *More general systems*

More generally, models for single populations can involve many discrete, but overlapping, age classes (see, for example, Levin & Goodyear 1980; Sparrow 1980; Hassell & May 1987). These eventually shade into situations where populations undergo continuous growth, but with time delays in recruitment or other processes, thus obeying time-delayed differential equations. The standard equations used by the International Whaling Commission to set quotas are of this kind, and can exhibit a rich range of period-doublings and chaotic behaviour as time lags lengthen and nonlinearities become steeper (although the parameters pertaining to real whale population produce only stable points). For reviews of this material, see May (1983) and Olsen & Degn (1985).

In short, simple and natural models for various kinds of biological populations exhibit cyclic and chaotic dynamics. We now ask whether population parameters will typically have values leading to such interesting dynamics.

## 3. Chaos and the dynamics of real populations

### 3.1. *Analysis of laboratory populations*

Single populations that are subject to density-dependent regulation, in a deterministic and homogeneous environment, are likely to be found only in the artificial setting of the laboratory. In this printed version of my lecture I shall not recapitulate my recent review of a variety of such laboratory studies (May 1986), several of which do indeed seem to show transitions from stable points to cycles, and possibly to chaos, as factors affecting demographic parameters are altered. Many of these laboratory investigations are, moreover, accompanied by explicit mathematical models for the dynamical behaviour.

Such laboratory studies are seen by some ecologists as unsatisfactory, in at least two respects. On the one hand, their artificiality may be argued to give them the status of living computers (conforming to unnaturally simple regulatory mechanisms, of little relevance to the dynamics of natural populations). On the other hand, for all the artificiality there remain many biological sources of noise, so that

we do not in fact see period-doubling or other crisp transitions in dynamical régimes, but instead see at best fuzzy changes from constancy to cycles of increasing amplitude and/or irregularity, as biological parameters are varied.

### 3.2. *Natural populations: understanding the dynamics*

Interactions with other species mean that natural populations are usually governed by higher-order systems of equations. These complications are, of course, compounded by environmental noise and spatial heterogeneity. Thus, although broad patterns may be understandable (four-year cycles in many populations of small mammals in extremely seasonal environments may be an example), most work on the dynamics of natural populations is concerned just with trying to tease out density-dependent signals from a confusing background of density-independent noise, rather than with nonlinear details of the density-dependent signal as such.

The population dynamics of viral, bacterial, protozoan and helminth infections constitute one class of possible exceptions to this gloomy view. For one thing, the transmission of such organisms among hosts may be described more simply than is the case for the complex numerical and functional responses characterizing the population biology of most vertebrate prey–predator associations. For another, public health records afford long runs of data. Recent work on the nonlinear dynamics of host–pathogen associations seeks to explain the persistent and non-seasonal oscillations in the reported incidence of many childhood infections of humans in developed countries (measles, pertussis, rubella), and to predict temporal changes in incidence of infection following the implementation of specific vaccination programmes. These confrontations between nonlinear models for the dynamics, and population data, are also reviewed in my Croonian Lecture (May 1986). Period-doubling and chaos play a part in much of this work on non-seasonal periodicities (see, for example, Aron & Schwartz 1984; Grossman 1980).

### 3.3. *Natural populations: phenomenological analysis*

A very different approach to the analysis of population data has been pioneered by Schaffer & Kot (1985, 1986). The approach uses methods developed for physical problems by Packard *et al.* (1980), Takens (1981), and others, for situations where only one variable can be measured in a system possessing many independent variables. If some multidimensional attractor underlies the observed time series, it may be reconstructed (without any understanding of the fundamental mechanism that generates it) by choosing some fixed time lag, $T$, and plotting values of the variables $x(t)$, $x(t+T)$, $x(t+2T)$, ..., $x(t+[m-1]T)$ in $m$-dimensional space; the value chosen for $T$ is not critical. The value of $m$ is selected so that increasing its value by unity does not apparently result in any additional structure.

Schaffer & Kot (1985, 1986) have applied these techniques to the recorded numbers of cases of chickenpox, mumps and measles per month, $N(t)$, in New York and Baltimore before mass vaccination. They construct three-dimensional phase plots of $N(t)$, $N(t+T)$, $N(t+2T)$, with $T$ fixed around two to three months. For all three-phase plots, Poincaré cross sections suggest the flows are indeed confined to

a nearly two-dimensional conical surface, corresponding to some nearly one-dimensional map. Schaffer & Kot compute the Lyapunov exponents for these phenomenologically constructed one-dimensional maps, and find them all to be positive. Olsen & Degn (1985) review this work, and give a parallel but independent analysis of measles data from Copenhagen, which yields a one-dimensional humped map almost identical to those found for measles by Schaffer & Kot. Schaffer (1984) has also given a similar analysis of the Canadian data on apparent cycles in lynx abundance, arguing that this system also is chaotic and governed by a nearly one-dimensional map.

This phenomenological approach is clearly different in spirit from conventional approaches which seek to understand dynamical behaviour in terms of specific models based on underlying biological mechanisms. The approach holds the promise of providing new insights; its main problem is that it needs longer runs of data than are commonly available to population biologists.

## 4. Nonlinear effects and the analysis of population data

A growing amount of literature deals with the interplay between environmental noise and the intrinsic dynamics of nonlinear systems of the kinds discussed above. From a population biologist's point of view, one problem is that environmental fluctuations in reality affect individual organisms, and not population-level parameters as such. A fundamental analysis of how to extract density-dependent signals from environmental noise in population data therefore requires that we first understand how parameters characterizing the dynamics of a population derive from the behaviour of individuals.

Hassell (Hassell 1986; Hassell & May 1985) has recently explored these issues, both in illustrative but abstract models, and in relation to explicit data for populations of whitefly, *Aleurotrachelus jelinekii*, on viburnum bushes in England (Hassell *et al.* 1987). These theoretical and empirical studies exemplify the conjunction of three factors, which will influence the dynamics of most natural populations: in each generation, the overall population is distributed (often in a very non-uniform way) among many different patches; in each patch, density-dependent mechanisms affect the population dynamics (differently at different densities in different patches); and environmental fluctuations influence individual behaviour and thence population dynamics (again, possibly differently in different patches).

To begin, environmental fluctuations are ignored, and specific assumptions are made (or deduced from detailed observations) about the statistical distribution of the total population of reproductive adults among $m$ distinct patches. Suppose next that each adult produces $F$ offspring, and that the chance of each offspring surviving to the prereproductive dispersal stage, from a patch with $i$ adults and thus $iF$ offspring, is characterized by some density-dependent survival function $s(iF)$. The total population of reproductive adults in the next generation, $N_{t+1}$, is given by

$$N_{t+1} = m\{\sum_i p(i; N_t/m)\, s(iF)\, iF\}. \tag{4.1}$$

In this way we arrive at a first-order difference equation relating $N_{t+1}$ to $N_t$, but now the parameters are those characterizing the distribution $p(\cdot)$ and survival $s(\cdot)$ at the patch and individual level. Note that regulatory effects are likely to be a combination of inter- and intragenerational effects, with some regulation occurring within each generation owing to density dependence acting differently at the different densities in various patches, and other regulatory effects deriving from between-generation differences in average population densities.

Such mathematical models can be used to generate pseudo-data (which can be noisy if the dynamics are chaotic), against which to test standard techniques of data analysis. Such studies show that conventional $k$-factor analysis, which in this case essentially plots changes in the total egg-to-adult mortality (the $k$-value on the $y$-axis) against initial adult density (plotted logarithmically on the $x$-axis), does reveal the density-dependent regulatory effects deriving from nonlinearities in the survival function $s(\cdot)$. This is true even though the analysis involves only average densities in successive generations, whereas much of the regulation occurs among patches within each generation.

The picture changes, however, when stochastic fluctuations are incorporated in the clutch size $F$, or in the parameters characterizing the dispersal and survivorship functions $p(\cdot)$ and $s(\cdot)$, respectively. Analysis of this theoretically generated pseudo-data by conventional $k$-factor analysis, applied to averaged densities in successive generations, in some cases still does reveal the underlying density-dependent effects, but in other cases does not. Whether or not the density-dependence that is actually present – though often predominantly acting within each generation – is revealed by such analysis depends on the magnitude of the stochastic fluctuations thus introduced (which is understandable), but also upon which parameter has been made noisy. May (1986) gives a much more explicit review of this recent work, complete with application to a model based on the whitefly-viburnum data.

The essential point is that when a population is distributed non-uniformly among many patches, with patches of different densities capable of exhibiting dynamical patterns ranging from stable points, through stable cycles, to chaos, the disentangling of density-dependent regulatory effects from superimposed environmental noise can be very difficult. The task may, indeed, often not be possible using conventional methods applied to overall average densities in successive generations. These questions go to the heart of the subject, calling for a reappraisal of conventional methods of gathering and analysing data.

## 5. Population genetics and chaotic polymorphisms

However complex the dynamics in simple population models, things can get messier when population genetics is combined with population biology in models where fitness functions are frequency- or density-dependent.

The simplest such models deal with a single diallelic locus, with $p_t$ and $q_t$ being the relative proportions (or 'frequencies', $p+q=1$) of the two alleles A and a, respectively, in generation $t$. In a diploid population with random mating, the

frequency of A in generation $t+1$, $p_{t+1}$, is related to that in generation $t$ by a first-order difference equation

$$p_{t+1} = \frac{(p_t^2 W_{AA} + p_t q_t W_{Aa})}{(p_t^2 W_{AA} + 2p_t q_t W_{Aa} + q_t^2 W_{aa})}. \tag{5.1}$$

The quantities $W_{ij}$ represent the fitnesses, or relative reproductive successes, of the three genotypes. If these fitnesses themselves depend on the gene frequency $p_t$, as can happen in a variety of biologically reasonable situations, we can have a highly nonlinear map, in the unit square, relating $p_{t+1}$ to $p_t$.

In particular, May & Anderson (1983) have studied aspects of the coevolution of host-pathogen associations, using fitness functions derived from the kind of epidemiological considerations sketched in §2.3. Specifically, they assume in the simplest case that each of the three genotypes is susceptible to a particular pathogen (to which the other two are resistant), which spreads in epidemic fashion as described in §2.3. The fitness of genotype ij (ij ≡ AA, Aa, aa) in generation $t$ is then

$$W_{ij} = \lambda_{ij}[1 - \gamma_{ij} I(N_{ij})]. \tag{5.2}$$

Here $\lambda_{AA}$ is the fitness, or relative productive success, of genotype AA in the absence of disease; $\gamma_{AA}$ is the proportion of those infected who die; and $N_{AA} = N_t p_t^2$, with $N_t$ the total density of the population in generation $t$. Similar definitions apply to the corresponding quantities for the genotypes Aa and aa. In each case, the Kermack–McKendrick (1927) relation gives the implicit expression (2.5) for $I(\cdot)$:

$$1 - I_{ij} = \exp[-I_{ij} N_{ij}/N_T]. \tag{5.3}$$

Here, as before, $N_T$ is the threshold density for transmission of the infection.

Two cases can now be distinguished.

If overall population density in each generation is held constant by other ecological constraints, $N_t = K$, the proportion of each of the three genotypes to be infected – and thence, via (5.2), the fitness functions $W_{ij}$ – depend only on the gene frequency, $p_t$. For such frequency-dependent (but not density-dependent) selection, gene frequencies in successive generations obey (5.1) with frequency-dependent $W_{ij}$ from (5.2) and (5.3). The map has obvious fixed points at (0,0) and (1,1), but in general also has an interior fixed point by virtue of the propensity of $p_t$ to increase from low values and decrease from high values (because disease spreads less effectively among rare genotypes and more effectively among common ones). The result can be a stable polymorphism, but for plausible values of the epidemiological parameters there can alternatively be cyclic or chaotic fluctuations in gene frequency.

If overall population density is itself regulated by the different diseases afflicting the three different genotypes, then both total population density and gene frequency can fluctuate cyclically or chaotically. Indeed, if all three diseases are lethal ($\gamma_{ij} = 1$ in all cases), it follows from §2.3 that the total population, and consequently the relative proportions of the genes A and a, have chaotic dynamics. Figure 2 illustrates such chaotic fluctuations in gene frequencies.

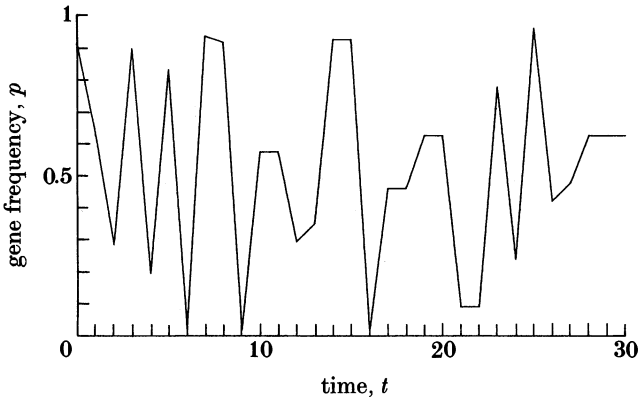


FIGURE 2. Illustrating the highly chaotic dynamical behaviour of the gene frequency $p$ that can arise once the selective forces exerted by the pathogens are both frequency- and density-dependent. The figure plots successive iterates of $p$, as generated by the first-order difference equation (4.1) with (4.2) and (4.3) defining the fitness functions. After May & Anderson (1983), where details are given.

The above is a very brief summary of results presented in detail in May & Anderson (1983). The results have intrinsic mathematical interest, but their greater significance is that – like the results outlined in the previous section – they suggest we should think again about empirical aspects of biological studies. Given that many polymorphisms are thought to be maintained by frequency- or density-dependent mechanisms, it must be recognized that such maintenance is not necessarily at constant proportions of A and a; these nonlinear mechanisms can readily generate cyclic or chaotic fluctuations in gene frequencies. Whether such fluctuations are observed in natural populations is not known, because most studies have not reckoned with the possibility that gene frequencies may be continually changing, driven by their own chaotic dynamics.

## 6. DISCUSSION

In part, this review has aimed to present a range of models which provide deliberately oversimplified descriptions of the dynamics of natural populations of plants and animals. These simple, yet naturally derived, models can exhibit an astonishing array of dynamical behaviour.

I have argued that such behaviour does give qualitative insights into many population phenomena in the natural world, and that in some cases (dynamics of infectious diseases, for example) reasonably detailed understanding of nonlinear dynamical effects is emerging. But I doubt that phenomena like period-doubling or inverse Hopf bifurcations will be seen even in the cleanest population data, as they arguably are in physiological or biochemical contexts.

Recognition that density-dependent mechanisms can produce cyclic and chaotic behaviour in natural populations does, however, have important implications for the way certain kinds of data are analysed by ecologists. As clearly illustrated by applying conventional techniques of analysis to pseudo-data generated by models

in which nonlinear dynamics, spatial heterogeneity, and environmental stochasticity roil together, the claim that a given set of data shows no evidence for density-dependence may often be a statement about the method of analysis, or about how the data were collected, and not about the biology of the system. Similarly, simple models suggest that cyclic or chaotic fluctuations in gene frequency can easily be maintained by selective forces whose magnitudes depend on gene frequencies or population densities. Again, there is need for a fresh look at gene frequencies in field populations, in the light of these results.

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## Appendix A

This appendix establishes the result that host–parasitoid systems, as described by (2.10), are unlikely to have stable periodic orbits of order below 6.

Equation (2.10) has a fixed point at $y = y^*$, $x = x^* = y^*\lambda/(\lambda-1)$, where $y^*$ is given by $\lambda f(y^*) = 1$. A linear stability analysis of this fixed point leads to a quadratic equation for the stability-determining eigenvalues $\sigma$:

$$\sigma^2 - \sigma(1+\alpha) + \lambda\alpha = 0. \tag{A 1}$$

Here I have, for convenience, defined

$$\alpha \equiv -x^*(\mathrm{d}f/\mathrm{d}y)^*. \tag{A 2}$$

As $f$ should be monotonically decreasing for increasing $y$, we should have $\alpha > 0$. The fixed point $x^*$, $y^*$, will be unstable as $\sigma$ crosses the unit circle. At this Hopf bifurcation, we write $\sigma = \mathrm{e}^{\mathrm{i}\theta}$, and the imaginary part of (A 1) then gives

$$\sin(2\theta) - (1+\alpha)\sin\theta = 0. \tag{A 3}$$

That is, at the Hopf bifurcation from a stable point to a stable cycle, the phase angle is given by

$$\cos\theta = \tfrac{1}{2}(1+\alpha). \tag{A 4}$$

At the bifurcation, the parameters $\alpha$ and $\lambda$ are related by $\lambda\alpha = 1$ (which can be obtained from the real part of (A 1) with $\sigma = \mathrm{e}^{\mathrm{i}\theta}$, or directly from the Schur–Cohn criterion). Using this to express $\alpha$ in terms of the more biologically familiar $\lambda$ in (A 4), we finally arrive at

$$\cos\theta = \tfrac{1}{2}(1+1/\lambda). \tag{A 5}$$

As $\lambda$ increases from around unity to very large values, the phase angle $\theta$ increases from 0° to 60°. We can thus obtain a six-point cycle in the limit $\lambda \to \infty$, but more generally a cycle of order roughly seven is the lowest likely to be found. This accords with several numerical studies presented by Olsen & Degn (1985), and with the earlier numerical work of Beddington *et al.* (1975). (Olsen & Degn's (1985) discussion has a sign wrong, to get $(1-1/\lambda)$ in (A 5); this leads them to conclude the phase angle $\theta$ lies between 60° and 90°, which would give cycles of approximate period between 4 and 6, rather than the correct 6 or more.)

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## *Discussion*

D. M. G. Wishart (*University of Birmingham, U.K.*). One of the most frequently analysed sets of data is the lynx–hare set. Is Professor May suggesting that we should, perhaps, declare a moratorium on this activity?

R. M. May. The lynx–hare data, compiled for the trading records of the Hudson Bay Trading Co., constitute one of the few long-term series available to population