

Chapter 6

Structured populations: continuous state and time

The goal of this chapter is to develop some important aspects of current approaches to modeling structured populations in continuous time. We begin with some classical models that every theoretical ecologist should know about, and then move to more contemporary work.

As in the last chapter, the goal of these models is to understand the consequences of variability among individuals in their “state”: age, size, or any other attribute or combination of attributes that has implications for birth and death rates or for the attributes of offspring. Once we move to continuous time, it becomes natural to also imagine that the state variables characterizing individuals also change continuously, rather than having a discrete set of possible states as in a matrix model.

The combination of continuous state and continuous time puts us in the realm of partial differential equations, which are both numerically and analytically harder to deal with than the models we’ve dealt with so far. Ways to reduce model complexity, while retaining as much biological realism as possible, are therefore very useful. The approach that we will be emphasizing originated in attempts to understand the experimental results shown in Figure 6.1. Both of these are laboratory insect populations, limited by the rate of food supply. These were grown under constant conditions, so the oscillations are internally generated.

A striking feature of the blowfly data is the emergence of nearly discrete generations, with each new cohort of flies generated by a separate burst of egg production. In this experiment the limiting factor was the food supply to adults – food for larvae was provided in excess. Note that eggs were only produced when adult density is low. When there are too many adults relative to the food supply, they can eat enough to survive but aren’t able to reproduce. The period of the population cycles is roughly 2-3 times the maturation time (which is roughly constant, because immatures have as much food as they want).

In the *Plodia* data, the cycles are more irregular, and their dominant period is close to the generation

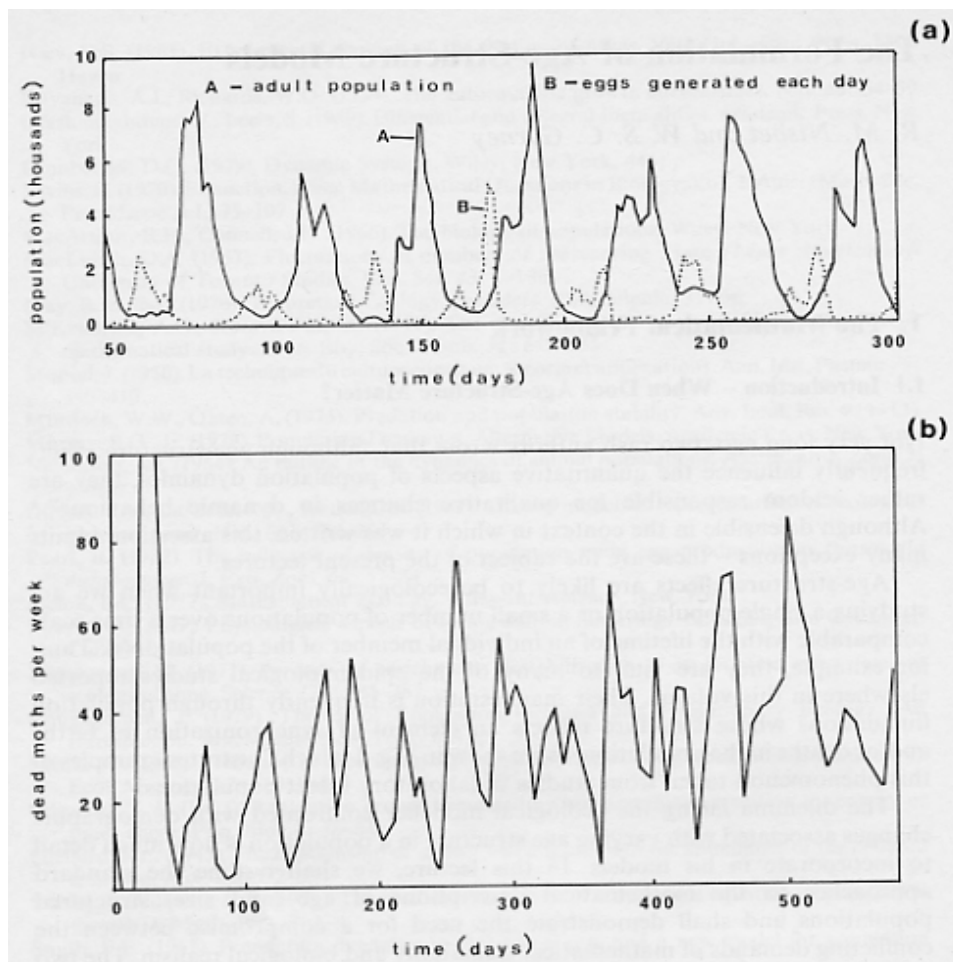


Figure 6.1: Experimental data on insect population fluctuations under constant conditions resulting from age-structure effects. (a) Nicholson's (1954,1957) blowflies. The solid curve is the total adult population, and the dotted curve is the rate of egg production. These data first appeared as Figure 3 in Nicholson (1954) and are from an experiment where the population was "governed by the daily supply of 0.5g of ground liver for the adults" (Nicholson 1954 p. 21). (b) Lawton's experiments on *Plodia interpunctella* (Indian meal moth) with the population regulated by limited food supply for larvae. The data are the number of dead adult moths, which is a proxy for the adult population. This figure is taken from Nisbet and Gurney (1986).

time. In this experiment population was limited by the food used by larvae, while food for adults was supplied as needed.

So there are two things in need of explanation:

1. How can discrete generations can emerge spontaneously in a continuously breeding organism growing under constant conditions?

2. How do different mechanisms of population regulation result in very different relations between the period of population cycles and the generation length of the organism?

Both of these questions have to be addressed using models where time and individual state are both continuous. In a discrete time model, by specifying a discrete time interval we are imposing the *a priori* restriction that cycle periods must be multiples of the model's time step. So to fully understand these patterns we have to start with a model where time is continuous, so temporal patterns cannot be artifacts of an arbitrary choice of time step. Continuous state is needed so that quantities like "maturation time" can be well defined in the model. In a model with discrete stages (e.g. egg-larva-pupa-adults), individuals within a stage at a given time necessarily all have the same probability of moving up to the next stage.

6.1 Age structure in continuous time

The simplest starting point is the continuous-time analog of the Leslie matrix, in which vital rates depend on individual age (a). As in the Leslie model we ignore (to begin with) effects of population density and environmental factors, consider a single species, and count only females. The state of the population is then characterized by the *age distribution* $n(a, t)$ that we can loosely think of as being the number of age- a individuals at time t . Technically n is the probability density of the age distribution, and

$$\int_a^b n(s, t) ds = \text{number of individuals of age } a \text{ to } b. \quad (6.1)$$

However if $n(s, t) \approx n(a, t)$ over the whole interval $a \leq s \leq a + 1$, then $n(a, t)$ will be approximately the number of individuals between age a and age $a + 1$.

The dynamics of $n(a, t)$ are generated by the age-specific per-capita birth rate $b(a)$ and death rate $\mu(a)$ – these are the “primitives” of the model. The basic balance law is that in order to be age $a > 0$ at time t , an individual must have been age $a - dt$ at time $t - dt$, and not survived from time $t - dt$ to time t . That is:

$$n(a, t) = n(a - dt, t - dt) (1 - \mu(a - dt)dt). \quad (6.2)$$

With a bit of algebra this rearranges to

$$\frac{n(a, t) - n(a - dt, t - dt)}{dt} = -\mu(a - dt)n(a - dt, t - dt). \quad (6.3)$$

The left-hand side of (6.3) is a difference quotient, so letting $dt \rightarrow 0$ we get

$$\frac{\partial n}{\partial t} + \frac{\partial n}{\partial a} = -\mu(a)n(a, t) \quad (6.4)$$

which is the dynamic equation for $n(a, t)$. To complete the model we only need to supply the *boundary conditions*

$$n(0, t) = \int_0^{\text{infy}} b(a)n(a, t)da. \quad (6.5)$$

To study model solutions and their properties we need to calculate the survival curve $l(a)$, the fraction of individuals that survive from birth (at age 0) to age a . Starting with N_0 age-0 individuals at time 0, let $N(t)$ be the number alive at time t . The instantaneous per-capita mortality rate for this cohort is $\mu(t)$, because all individuals are age t at time t . So

$$\frac{dN}{dt} = -\mu(t)N(t)$$

and therefore

$$\frac{d}{dt} \log N(t) = -\mu(t).$$

Integrating both sides of the last equation, we get

$$\log N(t) = \int_0^t -\mu(s)ds + C$$

where C is a constant depending on initial conditions. Setting $t = 0$ we see that $C = \log N(0)$, so

$$N(t) = N_0 e^{-\int_0^t \mu(s)ds}$$

and therefore

$$l(a) = e^{-\int_0^a \mu(s)ds}. \quad (6.6)$$

Sometimes $l(a, b)$ is used to denote the survival from age a to age b , and then $l(a) = l(0, a)$. A derivation like that above gives

$$l(a, b) = e^{-\int_a^b \mu(s)ds}. \quad (6.7)$$

For any $b > a$ we have $l(0, b) = l(0, a)l(a, b)$, so $l(a, b) = l(b)/l(a)$.

Exercise 6.1 Explain in words why $l(0, b) = l(0, a)l(a, b)$ whenever $0 < a < b$.

Exercise 6.2 If X is a non-negative random variable, and $F(x)$ is its cumulative distribution function $F(x) = \text{Prob}(X \leq x)$, then

$$E[X] = \int_0^\infty (1 - F(x))dx.$$

Using this fact, show that the life expectancy (i.e. the mean age at death) for an individual is $\int_0^\infty l(a)da$.

6.1.1 Solving the age-structure model

The solution to the age-structure model $n(a, t)$ can be expressed in terms of the survival function $l(a, b)$, and the initial population $n(a, 0)$. The form of the solution depends on whether or not $a > t$. If so, any age- a individuals must have been alive at time 0. If not, age- a individuals were born at $t - a > 0$.

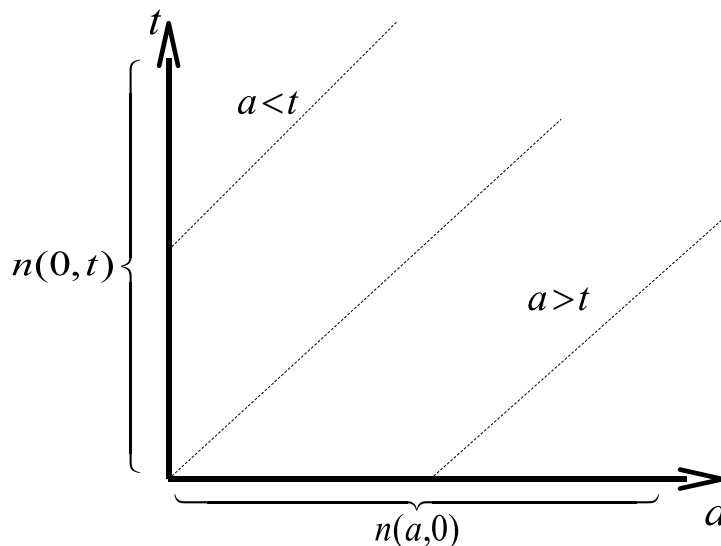


Figure 6.2: The so-called Lexis Diagram representing the solution (6.8) to the age-structure model.

In either case, $n(a, t)$ is the number of those “founders” who survive up to time t . So the form of the solution is

$$\begin{aligned} a < t : \quad n(a, t) &= n(0, t - a)l(0, a) \\ a > t : \quad n(a, t) &= n(a - t, 0)l(a - t, a) \end{aligned} \tag{6.8}$$

See Figure 6.2; following back lines of slope 1 (representing time and age changing at the same rate), points with $a < t$ trace back to the t axis, representing newborns (age 0) at time $t - a > 0$. Points with $a > t$ trace back to the a axis, representing individuals who were alive (and age $a - t$) at time 0.

This is not really a complete solution to the model, because the number of births $n(0, t - a)$ is not something we know – it has to be calculated from (6.5). Nonetheless it helps us determine the model’s longterm behavior. In the long run we expect to see exponential growth or decline at some rate r ,

$$n(a, t) \sim Cn^*(a)e^{rt} \tag{6.9}$$

where $n^*(a)$ is the *stable age distribution* and C is a constant depending on initial conditions. Like any eigenvector (pause a moment until that makes sense to you, or ask your instructor) n^* is only defined up to a multiplication by a constant. It is convenient to scale the stable age distribution so that $n^*(0) = 1$, so $n^*(a)$ is then the number of age- a individuals relative to the number of age-0 individuals.

For t large, we can substitute the second line of (6.8) into (6.5), getting

$$n(0, t) = \int_0^{\infty} b(a)n(0, t-a)l(a)da. \quad (6.10)$$

Then substituting (6.9) on both sides of this equation:

$$\begin{aligned} Ce^{rt} &= \int_0^{\infty} b(a)Ce^{r(t-a)}l(a)da \\ \implies 1 &= \int_0^{\infty} e^{-ra}l(a)b(a)da. \end{aligned} \quad (6.11)$$

The last line above is the famous Euler-Lotka equation. Because the right-hand side is a monotonic decreasing function of r , with limiting values $+\infty$ as $r \rightarrow -\infty$ and 0 as $r \rightarrow \infty$, there is always a unique solution.

Exercise 6.3 Define

$$R_0 = \int_0^{\infty} l(a)b(a)da. \quad (6.12)$$

R_0 can be interpreted as the average lifetime number of (female) offspring to a (female) newborn individual. Show that the asymptotic population growth rate r , which is the solution to the Euler-Lotka equation, is positive if $R_0 > 1$, and negative if $R_0 < 1$.

We can also derive the form of the stable age distribution using (6.9). For t large

$$\begin{aligned} n(a, t) &= n(0, t-a)l(a) \\ Ce^{rt}n^*(a) &= Ce^{r(t-a)}n^*(0)l(a) = Ce^{r(t-a)}l(a) \\ n^*(a) &= e^{-ra}l(a) \end{aligned} \quad (6.13)$$

There are several different ways that people define the *generation time*, all denoted T . These are different answers to the question: what is the average time between successive generations in the population, once it reaches stable age distribution? The simplest is to define T by the equation

$$e^{rT} = R_0$$

giving $T = \log(R_0)/r$. The motivation for this definition is that in each generation, a mother replaces herself by (on average) R_0 female offspring. When each mother has been replaced by her R_0 offspring, the population has grown (or shrunk) by a factor R_0 . T as defined above is the amount of time it takes (at stable age distribution) for the population to grow or shrink by that amount.

Another way of defining T is to take all the females born at some moment, and look at their average age at reproduction (i.e. each time one of them has a child, write down the mother's age; when all the females being tracked have died, average all the recorded ages at childbirth). This gives

$$T = \frac{1}{R_0} \int_0^{\infty} al(a)b(a)da$$

(remember, the last two equations are two *different* ways of defining T , and will generally give different values of T).

Exercise 6.4 Another way of defining T is as the mean age of the mother, for all of the children born “now” (at some large time t , when the population has reached stable age distribution). What is the formula for T under this definition?

6.2 Size structure in continuous time

It is not always sufficient or desirable to use age as the sole variable distinguishing individuals. An approach similar to the age-structured model can be developed for individuals classified by any continuous state variable m that changes as an individual develops over time. We will think of m as “size”, but it could really be any trait that changes deterministically over the course of an individual’s lifetime.

The state of the population is given by $n(m, t)$, the size-distribution of individuals at time t . The formal meaning of n is that $\int_a^b n(m, t) dm$ is the total number of individuals with size between a and b at time t . More useful are the approximate consequences of this definition:

The number of individuals in the size range $[m, m + h]$ is $n(m, t)h$

The number of individuals in the size range $[m, m - h]$ is $n(m, t)h$

These are approximations that hold as $h \rightarrow 0$, with error proportional to h^2 , but you generally won’t go wrong by thinking of them as being exactly true for h small.

In matrix models for size-structured populations we could easily allow changes in size to be random: some individuals of a given size-class now grow, while others shrink. In continuous time this is more difficult, so we will begin by only considering *deterministic* size dynamics. That is, we assume that all individuals of size m have size-dependent growth rate $g(m)$. The size of an individual therefore obeys the differential equation

$$dm/dt = g(m), \quad m(0) = m_0 \quad (6.14)$$

where m_0 is the individual’s size at birth. Size at birth can be allowed to vary in this model (i.e. not everyone will have the same m_0), but the growth function $g(m)$ is assumed to be the same for all individuals. Similarly, we assume that all size- m individuals have the same per capita mortality rate $\mu(m)$.

As in the age-structured case, we can derive a partial differential equation for the changes in $n(m, t)$ by tracking how many the individuals enter and leave a particular small category – in this case a size range $[m_1, m_2]$ – in a short interval of time of duration $\tau \ll 1$. Starting at some time t , Figure 6.3 leads to the following balance equation:

$$\begin{aligned} n(m, t + \tau)(m_2 - m_1) &= n(m, t)(m_2 - m_1) \\ &+ n(m_1, t)g(m_1)\tau - n(m_2, t)g(m_2)\tau \\ &- \mu(m)n(m, t)(m_2 - m_1)\tau \end{aligned} \quad (6.15)$$

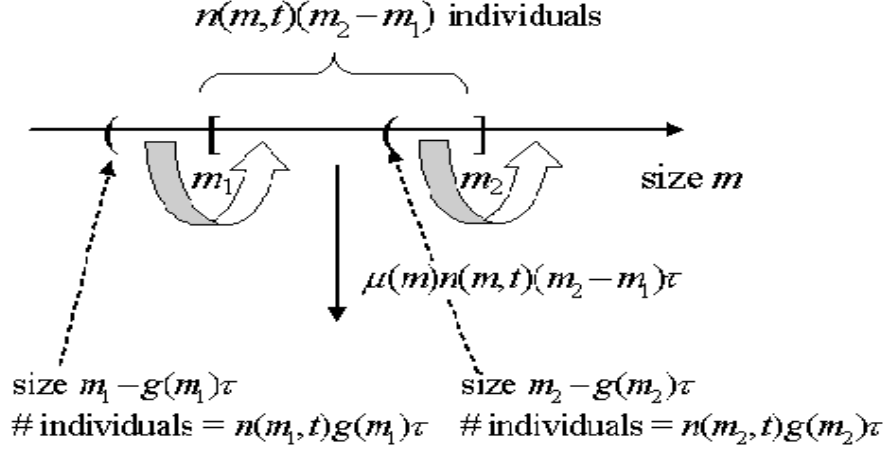


Figure 6.3: Population flows between times t and $t + \tau$ affecting the number of individuals in the size interval $[m_1, m_2]$, with m denoting a “typical” size in that interval. Individuals enter the size interval by growth from sizes $< m_1$ and leave it by growing to sizes $> m_2$ or by death.

where m is a “typical” size in $[m_1, m_2]$. We can be vague about m because we will soon let $m_2 \rightarrow m_1$, so that $m \rightarrow m_1$ also. The $ng\tau$ terms represent individuals growing into and out of the size range $[m_1, m_2]$. In each of these $g\tau$ is the “ h ” multiplying n and giving the number of individuals in the relevant size range: those smaller than m_1 who are big enough to grow into the interval in time period τ , and those in the interval who are big enough to grow out of it.

We now divide through by $(m_2 - m_1)$ in (6.15) and then let $m_2 \rightarrow m_1$; the result is

$$n(m_1, t + \tau) = n(m_1, t) - \frac{\partial(gn)}{\partial m}(m_1, t)\tau - \mu(m)n(m, t)\tau. \quad (6.16)$$

Bringing $n(m_1, t)$ over to the left-hand side, dividing through by τ and letting $\tau \rightarrow 0$ we get the final form of the model:

$$\frac{\partial n}{\partial t} + \frac{\partial(gn)}{\partial m} = -\mu n \quad (6.17)$$

This is called the *McKendrick-vonFoerster equation* after the people who first applied it to structured population dynamics. But it is actually identical to – and derived in exactly the same way as – the equation for advective particle transport in fluid mechanics (with m being particle location and g the location-dependent flow velocity). Taking advantage of this analogy, it can be shown that, exactly as in fluid transport, changes in size with a random component can be modeled by adding a diffusion term (involving second derivatives of n with respect to m) to the right-hand side of the equation. In the simplest case, where the variance in growth rate is independent of size, the equation becomes

$$\frac{\partial n}{\partial t} + \frac{\partial(gn)}{\partial m} = -\mu n + D \frac{\partial^2 n}{\partial m^2} \quad (6.18)$$

with $D > 0$ the *diffusion coefficient*. At the level of the basic mathematical description, it doesn't matter whether m refers to the size of an individual or its location in space, so long as $g(m)$ gives the rate of change for all individuals currently "at m ". This is one of the reasons why a degree in applied mathematics (or theoretical physics) is good preparation for a career in anything.

To put this into more general perspective: the "primitives" of these structured population models are

- A set of state variables (m , L and W , or whatever) describing the state of the individual (so-called i -state variables)
- A model for how the i -state variables of each individual change over time

From these we derive a "macroscopic" model that tracks the total population in terms of the frequency distribution of i -state variables. At the macroscopic level we are no longer tracking individuals one-by-one, but instead looking at the frequency distribution of the i -state variable across the population as a whole. But the model is derived by making assumptions at the individual level, not at the population level.

Exercise 6.5 Suppose that individuals are classified by two variables, length L and weight W , with growth rate functions $g_L(L, W)$ and $g_W(L, W)$. By generalizing the derivation above (using a box in the (L, W) plane rather than an interval on the m line) show that the population distribution $n(L, W, t)$ satisfies the partial differential equation

$$\frac{\partial n}{\partial t} + \frac{\partial(g_L n)}{\partial L} + \frac{\partial(g_W n)}{\partial W} = -\mu n. \quad (6.19)$$

6.3 Dynamics of stage classes

The general PDEs for age or size-structure dynamics are not simple as they stand. And to understand population dynamics, we have to make things even more complicated by adding density-dependence, interspecific interactions, or other forms of nonlinearity. If we do this and leave the models at the current level of generality, it is hard to derive anything at all about their behavior, and it is not even simple to solve them numerically.

To get around this problem, Gurney, Nisbet and Lawton (1983) proposed simplifying the models by assuming stage-specific vital rate functions. This is similar to, but less extreme than, the assumption in a matrix model that all individuals within a stage are identical. The distinction is this: we *will* assume that all juveniles have the same growth rate, possibly as a function of external variables such as food supply. We *will not* assume (as a matrix model would) that all juveniles are the same size, so (for example) smaller juveniles might have a lower probability of maturing into the adult class.

The simplest case is when stages correspond to age classes. As an example to illustrate what kind of model is produced by assuming stage-specific vital rate functions, consider the following simple model that was proposed for Nicholson's blowly experiments with adult food limitation:

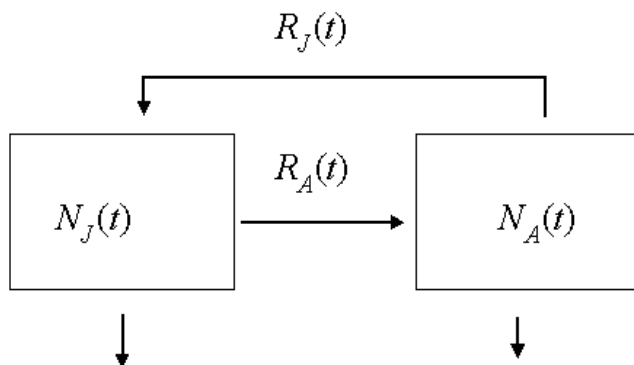


Figure 6.4: Age-structured model in continuous time with a Juvenile stage of fixed duration τ , and an adult stage with constant mortality. R_J and R_A denote the rates of recruitment into the Juvenile and Adult stages, respectively.

- Ages 0 to τ are Juveniles, with constant mortality rate $\mu(a, t) = \mu_J$ and birth rate $b(a, t) = 0$.
- Ages τ and above are Adults, with constant mortality rate $\mu(a, t) = \mu_A$ and birth rate $b(a, t) = qe^{-cN_A(t)}$ where $N_A(t)$ is the total number of adults at time t .

Under these assumptions, differential equations can be derived for the total numbers of Juveniles and Adults, $N_A(t)$ and $N_J(t)$. In the original article (and earlier renditions of these lectures) those equations were derived formally from the definitions

$$N_J(t) = \int_0^{\tau} n(a, t) da, \quad N_A(t) = \int_{\tau}^{\infty} n(a, t) da.$$

You differentiate with respect to t , pull the derivative inside the integrals, use the differential equation

$$\frac{\partial n}{\partial t} + \frac{\partial n}{\partial a} = -\mu(a)n(a, t)$$

to replace the t -derivative with an a -derivative, integrate by parts, \dots . It's not pretty.

Fortunately none of that is necessary. The stage structure of the population, and the rates of transfer between stages, are shown in Figure 6.4. Formally, we have the system of differential equations for the total numbers in each age class

$$\begin{aligned} dN_J/dt &= R_J(t) - R_A(t) - \mu_J N_J(t) \\ dN_A/dt &= R_A(t) - \mu_A N_A(t) \end{aligned} \tag{6.20}$$

The remaining task is to specify R_J and R_A . By assumption we have

$$R_J(t) = qN_A(t)e^{-cN_A(t)}.$$

Moreover, since the juvenile stage lasts exactly τ time units and the mortality rate is constant,

$$\begin{aligned} R_A(t) &= R_J(t - \tau) \times \text{survival through the juvenile stage} \\ &= R_J(t - \tau)e^{-\tau\mu_J} \end{aligned} \tag{6.21}$$

To simplify notation define $S_J = e^{-\tau\mu_J}$; we then have

$$R_A(t) = S_J q N_A(t - \tau) e^{-cN_A(t - \tau)}. \tag{6.22}$$

Since this depends only on N_A we can write a “standalone” equation for the adult population:

$$dN_A(t)/dt = S_J q N_A(t - \tau) e^{-cN_A(t - \tau)} - \mu_A N_A(t). \tag{6.23}$$

To reach this simple model we had to assume that all juveniles have the same rate functions. However, they are still allowed to differ in state: some are nearly mature and will soon become adults, others are recently born and will not become mature for some time. Although the final model only involves the total numbers in each class, its structure reflects the fact that newborns all wait τ time-units before maturing into Adults. The presence of the time delay τ in (6.23) is the price we pay for allowing individuals within stages to differ in state.

6.3.1 Modeling Nicholson’s blowflies with adult food-limitation

Gurney et al. (1983) were able to use Nicholson’s data to estimate the parameters for model (6.23):

- using the duration of each stage and the stage-specific mortality to estimate the egg-to-adult delay time $\tau \doteq 15.6$ and survival $S_J \doteq 0.91$.
- estimating the egg-production rate by combining data on egg production versus food supply with the assumption that food is divided evenly among adults, to get $b(N_A) \doteq 8.5e^{-N_A/600}$ for the experiments being modeled.
- using the rate of decline in adult population when no recruitment is occurring to estimate the adult mortality rate $\mu_A \doteq 0.27/d$.

With these estimates, the model produces sustained cycles with a period of about 37 days (compared to an average observed period of about 38 days), and adult population varying between a minimum of 150 and a maximum of 5400 (compared to observed mins and maxes of 270 ± 120 and 7500 ± 500) – pretty good for a model with *zero* free parameters adjusted to fit the adult population data. Moreover, model solutions exhibit the “double peak” that usually occurred in the data.

To simulate the model – without having to face the difficulties of solving delay-differential equations – we can express it as an age-structured model. Because the maturation time is 15.6 days, it is convenient

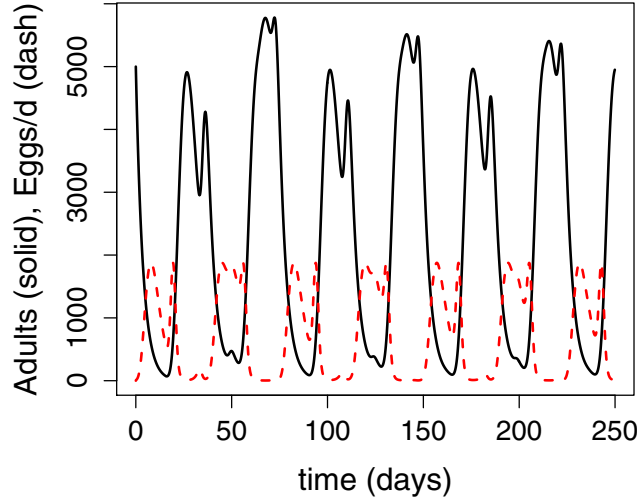


Figure 6.5: Simulation of the blowfly model with adult food limitation, by expressing it as an age-structured model with time and age increments of 0.1 days. The solid line shows the total adult population, and the dashed line is the rate of egg production (eggs/d)

to use time and age increments of 0.1 days. Then individuals of ages 0-156 are juveniles, with zero fecundity and survival probability $S_J^{1/157}$ per time increment. For adults we need only a single age-class: their fecundity per time step is $b(N_A)/10$ and survival probability (remaining in the adult class) $e^{-0.027}$ per time step. These rates specify a density-dependent Lefkovich matrix of size 158×158 . That's larger than what you usually see in the literature, but with a bit of attention to vectorizing a 500-day simulation (Figure 6.5 takes about 1 second in R).

It is instructive to contrast this model with one having discrete stage structure – a continuous-time analog of the stage-based matrix models in the previous chapter. The defining feature of such models is that individuals within a stage are assumed to be identical in *all* respects, including their odds of moving to the next-larger stage between one population census and the next. This is true for adults in (6.23), but juveniles differ in state. The closest possible analog to (6.23) with discrete stage structure would have juveniles leaving the juvenile stage at rate $1/\tau$ – so that the mean duration of the juvenile stage is τ – and a fraction S_J of those who exit the stage mature into adults while the remainder die.

$$\begin{aligned} dN_J/dt &= qN_A \exp(-cN_A) - N_J/\tau \\ dN_A/dt &= S_J N_J/\tau - \mu_A N_A \end{aligned} \tag{6.24}$$

This model can be rescaled into the form

$$\dot{J} = QAe^{-A} - J \quad \dot{A} = J - \delta A \tag{6.25}$$

and then analyzed by the methods of chapter 4. The conclusion is that there is a positive steady state so long as $Q > \delta$, it is always stable.

So regardless of parameter values, the model with discrete stage structure cannot possibly explain the observed population cycles. The difference in behavior between (6.23) and (6.24) illustrates the principle that a fixed time delay is usually destabilizing – here, relative to a model with an exponential distribution of the maturation time.

Exercise 6.6 Verify the statements made above about (6.25) by finding the steady state and computing the trace and determinant of the Jacobian matrix. Find the conditions on parameters under which the steady state is a stable spiral. For parameters in that range, use simulations of the model to show that cycles damp down very quickly onto the steady state, so that the sustained cycles observed in Nicholson's experiments cannot be explained by the transient behavior of model (6.25).

6.3.2 Modeling *Plodia*

Plodia requires a different model because larvae were food limited rather than adults. It was assumed that egg and pupa stages were short enough to ignore, so the model structure is the same as figure (6.4) except that the J compartment is now labeled A . Because adults are not food-limited, the model assumes constant per-capita mortality and fecundity for adults. Thus the rate of loss from the Adult compartment is $\mu_A A(t)$ and the recruitment rate of new larvae is $R_L(t) = qA(t)$.

The food limitation on larvae was modeled by assuming density-dependent larval mortality,

$$\mu_L(t) = \alpha L(t).$$

This also complicates the calculation of the recruitment into the adult class. We can write

$$R_A(t) = R_L(t - \tau) \times (\text{larval survival from } t - \tau \text{ to } t).$$

The first term on the right-hand side is $qA(t - \tau)$. The second is

$$S_L(t) \equiv e^{-\int_{t-\tau}^t \mu_L(s) ds} = e^{-\alpha \int_{t-\tau}^t L(s) ds}. \quad (6.26)$$

So we seem to have a *delay nonlinear integrodifferential equation model* on our hands. Fortunately we don't, thanks to a new trick: writing a differential equation for $S(t)$.

$$\frac{dS_L}{dt} = S_L(t) [-\alpha(L(t) - L(t - \tau))] = \alpha S_L(t) [L(t - \tau) - L(t)]. \quad (6.27)$$

Combining this with the balance equations for L and A (below) gives a complete model:

$$\begin{aligned} \frac{dA}{dt} &= qA(t - \tau)S_L(t) - \mu_A A(t) \\ \frac{dL}{dt} &= qA(t) - \alpha L(t)^2 - qA(t - \tau)S_L(t) \end{aligned} \quad (6.28)$$

The initial conditions for this model are somewhat tricky. To start the model running at $t = 0$ we need $A(t)$ and $L(t)$ from times $-\tau$ to 0. Gurney et al. suggest modeling inoculation of the population with new larvae: $A(t) = L(t) = 0$ for $-\tau \leq t \leq 0$ followed by

$$\frac{dL}{dt} = qA(t) + I(t) - \alpha L(t)^2, 0 < t < \tau$$

where $I(t)$ is positive and constant for short time period $0 < t < \epsilon$, and zero thereafter.

Gurney et al. also considered two variants of the model. The first modification is to assume that adults live for a fixed lifespan of τ_A days, and then die. The loss term $\mu_A A(t)$ is then replaced by $R_A(t - \tau_A)$ – deaths now are those individuals who became adults τ_A days ago. The second variant is to assume “cohort competition” among larvae: rather than competing with all larvae for food, larvae compete only with other larvae of the same age. This second variant is equivalent to the blowfly model, because a reduction in the survival of larvae of a given age has exactly the same effect as a reduction in the adult fecundity at their moment of birth.

The “target” that the *Plodia* model aims to hit is the cycle period being nearly the same as the generation length, roughly 42 days (Figure 6.6). The results are pretty good (Figure 6.7); even though the period is not predicted very closely, the qualitative result is right: the larval competition in the *Plodia* model leads to a much shorter cycle period (relative to the lifespan of the organism) than the adult competition in the blowfly model, matching the experimental findings.

6.4 Characteristic cycle periods

The blowfly and *Plodia* models suggest that different modes of population regulation lead to different characteristic values for the ratio between cycle period and maturation time. This appears to be true, as an “empirical fact” in the sense that it holds in a range of different models. The essential difference, first identified by Gurney et al. (1985) is whether the self-regulatory mechanism acts directly on numbers in the regulated stage (e.g., larval density causes higher larval mortality, hence the numbers of larvae are affected) or acts on the numbers in a later stage (e.g. adult density affects adult fecundity and hence the numbers of individuals in the egg/larval stages, or larval density affects how fecund those individuals will be when they mature).

Figure 6.8 summarizes their findings for 4 models (2 each of immediate/delayed). The basis for these results is an audacious approximation, validated by simulation. The approximation is to use linear stability analysis to compute the period of oscillation at the point where cycles arise by a Hopf bifurcation from a stable equilibrium. The cycle period of the linear system is determined by the point on the imaginary axis where the real part of the eigenvalues (a complex conjugate pair) crosses from negative to positive. Just into the unstable region, the cycles amplitude is small (proportional to the square-root of the real part of the eigenvalue, generically), so the cycle period is approximately that of the linear system. There is no reason why cycle periods must remain more or less the same once the system is in the range of large-scale oscillations, but in fact this is often what happens in population models that undergo a Hopf

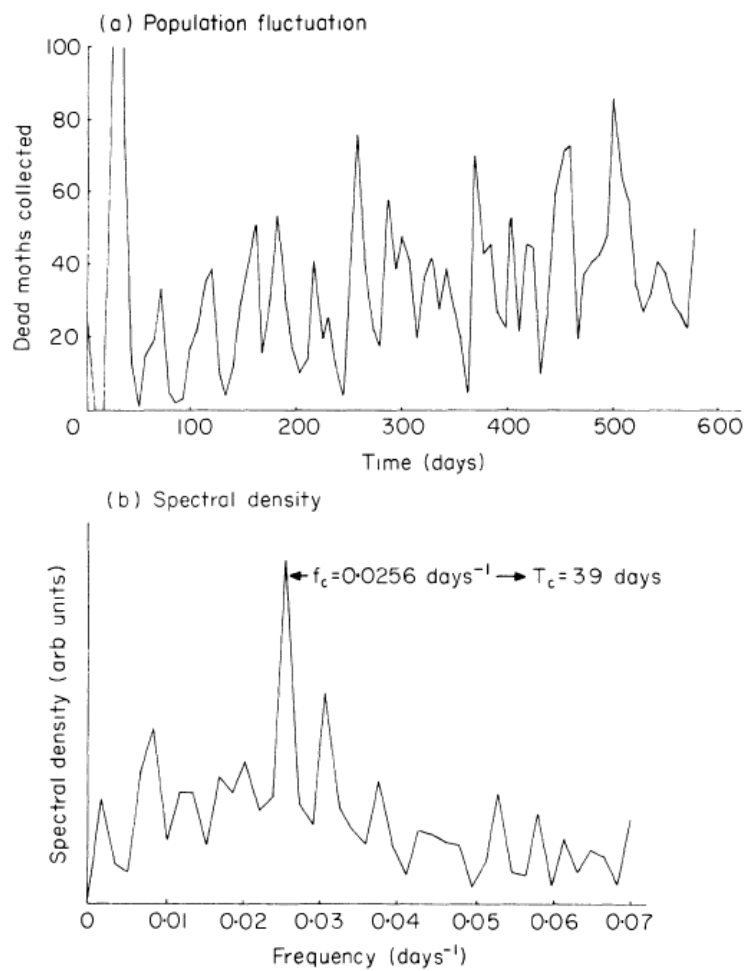


FIG. 5. (a) Weekly counts of dead adult moths in long-term cultures of *Plodia interpunctella*.

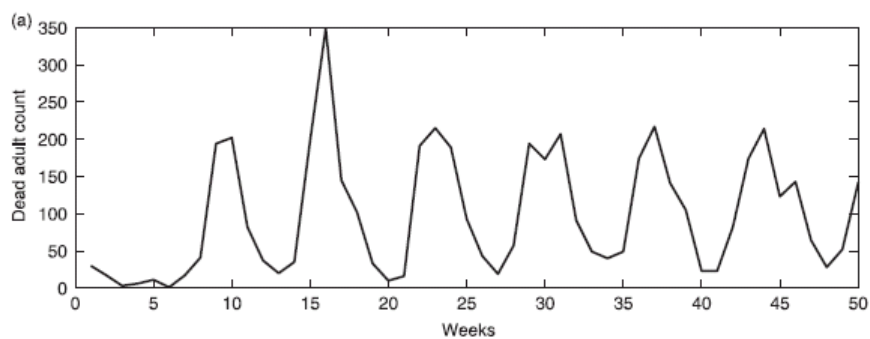


Figure 6.6: Data on laboratory populations of *Plodia interpunctella* with larval food limitation. Top panels are from Gurney et al. (1983), bottom from Wearing et al. (2004).

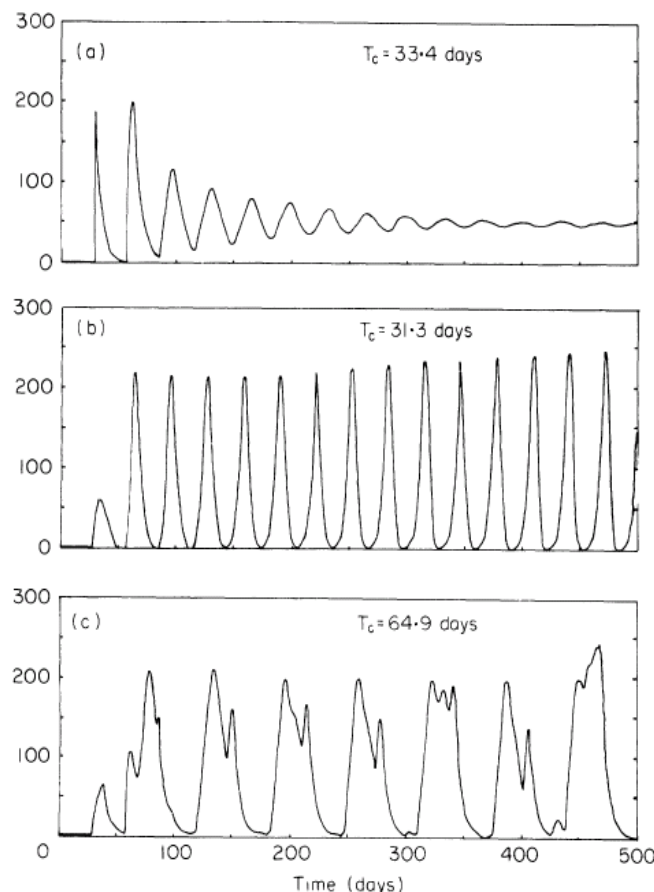


FIG. 4. Numerical solutions of the larval competition model with parameters appropriate to JHL's cultures of *Plodia interpunctella*: (a) uniform competition model with constant adult death rate (Variant (i)); (b) uniform competition model with fixed adult lifetime (Variant (ii)); (c) cohort competition model.

Figure 6.7: Simulations of the three variants of the *Plodia* model, from Gurney et al. 1983.

bifurcation giving rise to limit cycles. As a mathematical exercise it is easy to construct examples where cycle period changes quickly when cycle amplitude increases, but “empirically” in structured population models the cycle period at a Hopf bifurcation point is a good approximation to the period well beyond the bifurcation point.

The difference between immediate and delayed feedback can be understood intuitively, as follows (see Figure 6.9). In each case, we assume that there is a “limiting stage” in the life cycle whose density has a detrimental affect on survival. With direct feedback the limiting stage affects itself; with delayed feedback it affects another stage later in the life cycle.

1. With direct feedback, suppose there is a big burst of recruitment into the limiting stage at time t . There is then high mortality, killing off everyone else in the stage (and most of the new recruits, but not all of them, because they outnumber everyone else). This continues until these recruits

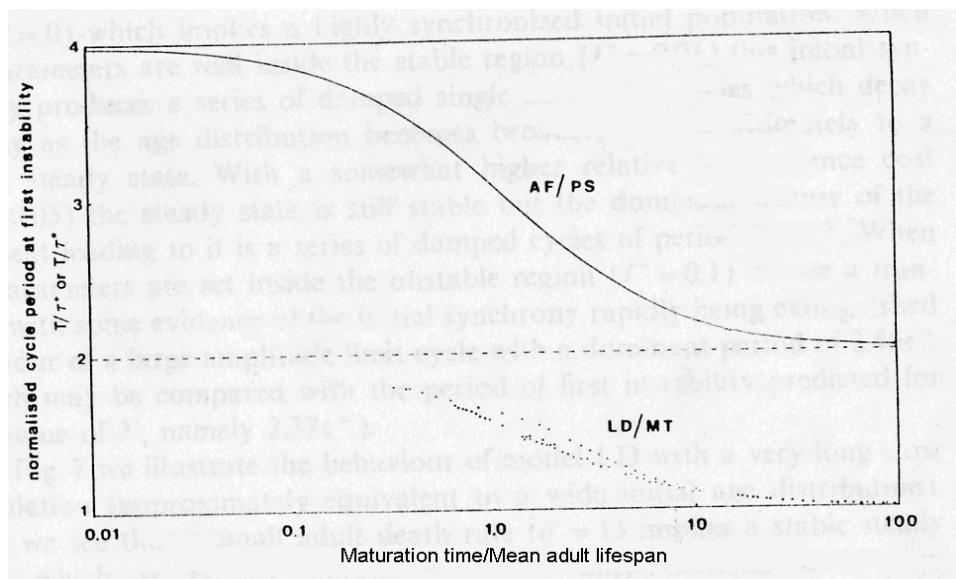


Figure 6.8: Scaled cycle period (relative to the maturation time) in continuous delay-differential models for age-structured populations with competition for limiting food. The x -axis is the ratio between the maturation time and the mean adult lifespan. In the “LD/MT” models the effects of food limitation act directly on the food-limited stage of the life cycle (e.g., food-deprived larvae have higher mortality, or a longer maturation time and hence lower survival to the next stage); these result in cycle periods “1 and a bit” times the maturation time. In the “AF/PS” models the effects of food limitation on the number of individuals is delayed to a later stage in the life cycle (food-deprived larvae grow up to be adults with lower fecundity, or pupae with lower survival); these result in cycle periods “2 and a bit” times the maturation time, up to 4 times the maturation time if mean adult lifespan is much longer than the maturation time.

mature out of the limiting stage. So a dominant cohort “clears out” everyone else within an age band whose width is twice the duration of the limiting stage. If that’s everyone (limiting stage is long, rest of the life cycle is short), the result is one cycle per generation – because the next big burst of recruitment into the limiting stage has to be the offspring of the dominant cohort. If the limiting stage is shorter, one can get shorter periods (e.g., two bursts of recruitment per generation time).

2. With delayed feedback, let τ be the “maturation time” – the time elapsed between leaving the affected stage and entering the limiting stage. If there are many individuals in the limiting stage “now”, that clears out the affected stage “now”, so there will be few in the limiting stage after the “maturation time” τ . That will allow high survival in the affected stage at time τ , and so there will again be many in the limiting stage after another generation time has passed. So the period of cycles is about 2τ , twice the “maturation time”. If we assume that adults are the limiting class and younger juveniles are affected, then τ really is the maturation time, and it will be approximately the generation time.

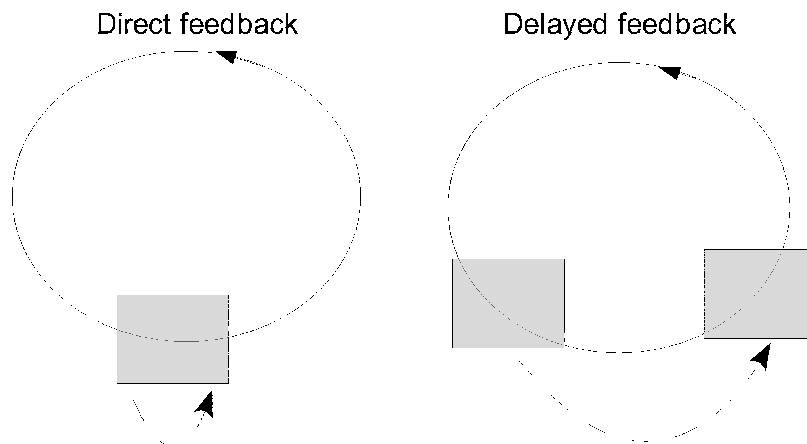


Figure 6.9: Diagrams of life cycles with direct versus delayed feedback on population growth.

6.4.1 Characteristic period for the blowfly model

Here we will use the blowfly model to illustrate how the characteristic cycle period for a given class of structured models can be derived analytically. [Some day soon, maybe].

6.5 Selected other applications

This chapter has introduced continuous-time structured population models by way of theoretical ecology's (and the author's) longstanding fascination with population cycles. Of course this is only one out of many, many applications of structured population models. The books edited by Metz and Diekmann (1986) and Tuljapurkar and Caswell (1997) contain a number of examples and reviews of such applications, along with additional aspects of the theory. Here we mention just a few examples to indicate the range of possibilities.

Levin and Paine (1974) proposed a model, motivated by rocky intertidal communities, in which a landscape is structured by external disturbances that create patches of open space. Patch creation is assumed to be followed by a deterministic local within-patch succession process. The local community is then viewed as a mosaic of patches in different stages of succession. In their model patches are classified by

age a and size ξ , so the state variable is $n(\xi, a, t)$ and the model structure is

$$\frac{\partial n}{\partial t} + \frac{\partial n}{\partial a} + \frac{\partial(gn)}{\partial z} = -\mu(\xi, a)n(\xi, a, t)$$

(SAL may still have the T-shirt with this equation on it presented by his graduate students for his 35th birthday). Paine and Levin (1981) parameterized the model based on Paine's studies of mussel beds on Tatoosh Island, Washington from 1970-1979. In that system patches are created when mussels are dislodged by external disturbances, creating new patches of various sizes. The mortality term μ represents patch disappearance. Small and medium-size patches disappear through incursions by surrounding mussels, while larger patches fill in through recruitment and establishment of larval mussels. With empirically based estimates of patch birth, growth, and disappearance rate functions, the model was able to make accurate predictions of total patch area. Predictions of the age \times size distribution were good for larger patches, but small patches were influenced strongly by stochastic effects that are not included in the model.

Murdoch et al. (1996) used stage-structured models for host-parasitoid dynamics to explain the competitive displacement of one parasitoid by another, both having been introduced in order to control red scale in California. The original control agent was unsuccessful. But another member of the same genus, once introduced, displaced the original control agent and provided successful control of red scale. Murdoch et al. (1996) show that this could be explained by a simple difference between the two introduced parasitoids: whichever parasitoid is able to produce female offspring on smaller host individuals is predicted to be the winner in competition and the more successful control agent.

Keeling and Grenfell (1997) examined the failure of conventional epidemic models to explain the "critical community size" (CCS) for measles in Great Britain. The CCS is a phenomenon first noted in the 1950's by Bartlett. Comparing case reports from cities of different sizes, one finds that in cities above a threshold size (about 250,000 to 400,000) the disease is always present: in every weekly reporting period, there are new cases. In cities below the threshold, there are fadeouts (defined by Keeling and Grenfell as 3 consecutive weeks with no case reports, which is long enough for anyone who caught the disease before the fadeout to have recovered from it). Similar patterns have been observed in the US and elsewhere.

Standard epidemic models use a discrete classification by disease state: individuals are either Susceptible, Latent, Infective, or Recovered. Consequently, the standard models do not have the property that individuals who have been sick for some time will recover sooner than recently-infected individuals. Despite numerous elaborations, including age structured disease transmission and spatial and social structure, these models uniformly overpredict the occurrence of fadeouts, and predict a CCS of about 1 million. Keeling and Grenfell (1997) extended an existing model by incorporating time-within-state, so that individuals are structured both disease state and also by how long they have been in that state, e.g. latent individuals (infected but not yet symptomatic) are represented by $n_L(a, t)$, where a is the time since the individual was infected by the disease. Compared to the standard models, their model has a lower between-individual variance for the time spent in each disease state. As a result, its dynamics are less irregular than that of the standard models, and it predicts fewer fadeouts. Simulations of the model show that it can account accurately not only for the observed CCS in Great Britain, but also for

how the frequency of fadeouts goes up as city size decreases below the CCS.

Size structure is also known to be important for predator-prey interactions between fish species, because of gape limitation: a fish can't eat another fish that is too big for its jaws. Small differences in timing – in predator abundance, how quickly juveniles grow, or whether climatic conditions give them a “head start” in outgrowing potential predators – can therefore be magnified into large differences in juvenile survival (e.g., Persson et al. 1996). As a result, size-structured models are important for predicting the survival of juvenile cohorts in prey species through their period of high vulnerability to predation.

Exercise 6.7 Consider the basic size-structured McKendrick-vonFoerster model

$$\frac{\partial n}{\partial t} + \frac{\partial(gn)}{\partial m} = -\mu n \quad (6.29)$$

that holds when individuals are all born at some minimum size m_0 . Suppose instead that individuals give birth only through *fission* – splitting into two individuals of size αm where m is the size of the “parent” and $0 < \alpha \leq \frac{1}{2}$. Let $\phi(m)$ be the rate (splits/individual/time) at which individuals undergo fission. The range of possible sizes for this model is $(0, \infty)$. Derive the modified equation that applies for this model. Note that when an individual undergoes fission, one individual of size m vanishes, and two individuals of size αm appear. You can pattern your derivation on the one for the basic model, but remember that fission adds a new way to exit, and to enter, any given size range.

Exercise 6.8 Consider the “*Plodia*” model where larval competition causes there to be an increased death rate of larvae. Suppose instead that larval death rate (per unit time) is constant, but that the transition from juvenile to adult occurs at a certain size rather than a certain age, so the duration of the juvenile stage can vary over time. This gives a stage-structured rather than an age-structured model. Specifically, suppose that

- Individuals of size 0 to 1 are *juveniles* with zero reproduction, and constant per-capita mortality rate $\mu_J > 0$.
- Individuals of size ≥ 1 are *adults*, with constant mortality rate $\mu_A > 0$ and constant per-capita fecundity $q > 0$.
- Juveniles grow at rate $g(t) = e^{-cN_J(t)}$; thus the duration of the juvenile stage for individuals maturing to adulthood at time t , which we will call $\tau(t)$, is implicitly defined by

$$\int_{t-\tau(t)}^t g(s)ds = 1. \quad (6.30)$$

In this model the effect of crowding is to slow the growth of juveniles, resulting in fewer of them surviving to adulthood.

(a) By differentiating both sides of (6.30) with respect to t , show that $\frac{d\tau}{dt} = 1 - \frac{g(t)}{g(t-\tau)}$.

(b) Show that $\frac{dN_A}{dt} = qN_A(t-\tau)e^{-\mu_J\tau(t)}\frac{g(t)}{g(t-\tau)} - \mu_A N_A(t)$.

(c) Derive the corresponding equation for $\frac{dN_J}{dt}$.

It is not a good idea to try deriving these results from the McKendrick-vonFoerster equation. Think in terms of Figure 6.4 and the rates at which individuals enter and exit each stage. Remember that the juveniles who become adults between times t and $t+dt$ are those whose size at time t is between $1-g(t)dt$ and 1. How much time elapsed between the birth times of the smallest and largest such individuals?

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