Chapter 2

Bet hedging

In the previous chapter we considered models for short-term decisions, making a choice among the available options for what to do "right now". However many important decisions are at a longer time scale, where the choice is between doing one thing now, and another thing later – e.g., having more young this summer, versus being in better condition to survive the next winter and breed again next year. These longer term decisions are the subject of *life history theory*, which views the entire life cycle from the adaptationist perspective and seeks to answer questions such as:

- At what age should reproduction begin?
- Should reproduction be all-at-once (*semelparity*, meaning that there is a single reproductive event which is fatal to the parent) or spread out in time (*iteroparity*)?
- How many offspring should be produced each year?
- How should the parent allocate time and resources between maximizing offspring survival, and maximizing its own chances of living to breed again next year?

These questions are more difficult to study than short-term optimization, for at least two reasons. The first reason is the need to have some "common currency" to evaluate tradeoffs of survival against fecundity, or of fecundity now versus fecundity later. These different kinds of "payoff' must be combined into an overall measure of Darwinian fitness for the life cycle, using results from the theory of structured population models – so we'll come back to this later.

The second reason – and the subject of this chapter – is that there is enormous variation, from one year to the next, in the conditions under which decisions have to be made. – see Table 2.1. The world is very "noisy". For a forager trying to make it through the day, variation between one year and the next is not an issue, and we can reasonably model as if the current conditions were going to persist indefinitely (at least as a starting point). But when the structure of the entire life cycle is at issue, it is hard to justify

Long-lived adults	Range of variation	Diapausing seeds/eggs	Range of variation
Forest perennial plants	333	Chalk grassland annuals	1150
Desert perennial plants	4	Chapparal perennials	614
Marine inverebrates	591	Freshwater zooplankton	1150
Freshwater fish	706	Insects	31,600
Terrestrial vertebrates	38		
Birds	2200		

Table 2.1: Range of between-year variation in reproductive success (of long-lived adults or diapausing seeds or eggs), over years in which some reproduction occurred, based on Hairston et al. (1996). Hairston et al. (1996) compiled field studies that estimated per-capita reproductive success in a population population, in several different years at the same location. They computed for each study the ratio between the highest and lowest annual values, omitting years when reproduction failed completely. The values in this table are the highest such ratio for species within each group listed.

models based on average conditions, such as the average payoff from a unit of time or energy invested in reproduction.

In this chapter we consider some simple models for individual decision making in the face of uncertainty, starting with a review based on Ellner (1997). The specific question is how reproduction should be spread across the life cycle, in situations where trying to reproduce is risky or the payoff is highly unpredictable. So an attempt this year may be fatal and unsuccessful, where waiting and trying later might have succeeded – or the reverse might hold, and in advance one can't predict which.

2.1 It's not easy being a seed

The canonical example is Dan Cohen's (1966) theory for seed germination in a desert annual plant. A seed of a desert annual plant faces the problem of completing its life cycle and setting a seed crop within a short and unpredictable growing season that (in Israel, where Cohen worked) begins with rains in the winter and ends in the hot, dry summer. If a seed is going to germinate this year, it should do so at the first opportunity – the first good rain – but if that is not followed by sufficient rainfall later, the plant will die before setting seed.

Dan Cohen did his PhD studying the physiology of seed germination, and then did a postdoc studying decision theory and applying it to germination and other biological "decisions" under uncertainty. It was a very career move. As of this writing (2/7/07) his first resulting paper (Cohen 1966) has been cited 426 times, with 18 citations in 2006. The complete Introduction to that paper (whose brevity may not be unrelated to the paper's success) is as follows:

> Most living organisms are faced with considerable risk of failure when trying to reproduce. One obvious way to survive and reproduce in a risky environment is to spread the risk so

Figure 2.1: A seed-bearing dead skeleton of the Rose Of Jericho, *Anastatica hierochuntica*, in Algeria. Rainfall causes the skeleton to open partially, releasing some seeds to the soil where they can stay wet long enough to germinate. Lee Segel, one of the founders of modern mathematical biology, called this plant the ultimate Jewish Mother, who even after her death is keeping a tight grasp on her children and controlling their lives (*personal communication*). Image from *Botany Online: The Internet Hypertextbook* by Alice Bergfeld, Rolf Bergmann, and Peter von Sengbusch at `www.biologie.uni-hamburg.de/b-online`.

that one failure will not be decisively harmful.

"Spreading the risk" of germination means that the seeds produced by one parent in one year will germinate at a range of times spread out over many years, even if they experience exactly the same conditions. This is accomplished by a variety of proximate mechanisms. A common one in desert plants involves germination inhibitors that must leach out of a seed before it can germinate. If seeds produced by a parent vary in the amount of germination inhibitor in their seed coat, two good things happen: germination is spread out over time, and a large rainfall causes more seeds to germinate than a small one. Another mechanism is differences in the sturdiness of a water-tight seed coat. Weaker ones will tend to crack sooner and allow the seed within to germinate, while stronger or thicker ones stay intact longer. Some desert plants retain seeds on their skeleton after they die, with mechanical processes causing some fraction to be released each time it rains. (Figure 2.1). If this piques your interest, see Evenari et al. (1971).

Figure 2.2 summarizes his model. At the start of growing season t, there are $n(t)$ buried seeds or eggs, of which a fraction H "decide" to hatch [seeds don't hatch, they germinate – but we'll eventually apply this to copepod eggs so I'll call it the hatching fraction]. Seeds that hatch each produce Y_t new seeds that re-enter the seed bank and survive until the start of growing season $(t+1)$. We consider Y_t to be highly variable: in some years there is a lot of rain and Y is large, in other years there is little or no rain

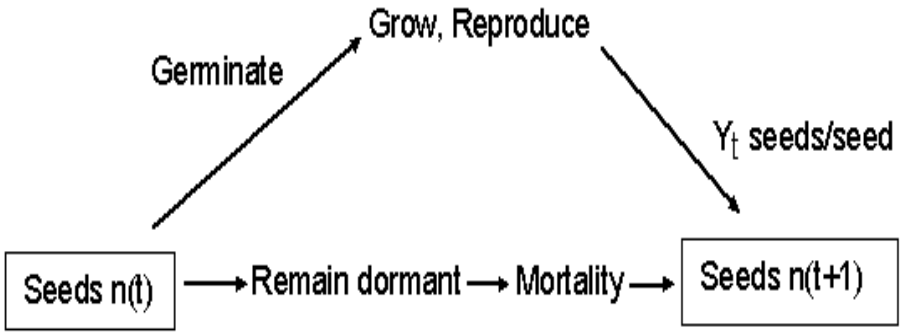

Figure 2.2: Cohen's (1966) model for optimal dormancy strategies in seeds of annual plants. At census times $t = 0, 1, 2, ...$ the population consists entirely of dormant seeds. Seeds that remain in dormancy have a constant risk of death, while seeds that germinate face a random and unpredictable set of conditions affecting their chances of completing the adult life cycle and producing a crop of new seeds.

and Y is small or 0. We also assume (for now) that Y_t is unpredictable: a seed has to germinate or not, without knowing if there will later be enough rain for it to grow, flower, and set seed. For seeds that don't hatch, life is predictable but unrewarding: a fraction $s < 1$ survive to time $t + 1$. The question is: should all seeds germinate, or should some "sit it out" until next year ($H < 1$)? And if so, how many?

We don't believe that seeds are literally making decisions about whether or not to germinate, but to build an adaptationist model we begin by assuming that evolution will find a way to let them behave as they "ought to" – then if this model fails we might ask whether seeds are under some constraints that keep them from utilizing the best possible decision rule.

Essentially the same model has recently been proposed (Stumpf et al. 2002) as an explanation for the *static latency* behavior of herpes viruses including herpes simplex HSV1 and HSV2, and varicella zoster virus (VZV) which is respondible for chickenpox and shingles. After an attack of chickenpox VZV lies dormant in nerve tissue. After many years it may re-emerge years to cause shingles. Shingles causes pain and discomfort that can last for months or even years, and an outbreak on the face may lead to permanently impaired hearing or sight. According to the US NIAID's FAQ page on shingles, roughly 20% of individuals in the US will get shingles at some time. Unlike the case of HIV, this is not an "active" latency resulting from host immune response – VZV has genes that actively maintain and regulate latency (Stumpf et al. 2002). Outbreak is more common in individuals with impaired immune function, but there is no known "trigger" for shingles to emerge from latency.

In the shingles model, the fluctuating environmental factor is the availability of susceptible hosts to infect, which they assume is unpredictable by the virus. In their model, an infected case can produce a certain number c_1 of infections immediately, and another number c_2 after a latent period of duration τ unless the host dies beforehand, each modified by a random variable r_t proportional to the susceptible

host density in year t:

$$N_{t+1} = r_t(c_1 N_t + c_2 m^\tau N_{t-\tau}).$$

Here m is the annual probability of host survival. Given enough variation in the abundance of susceptible hosts, Stumpf et al. (2002) show by simulation that large values of τ, similar to those observed for VZV, can be favored by natural selection – $\tau = 5$ years or higher for model parameters intended to represent preindustrial humans. This application illustrates the recent recognition that from the perspective of disease organisms **you are an ecosystem** and ecological models are often relevant to understanding the cat-and-mouse game between the immune system and infections, and how each of these evolves in response to the other.

Now back to seeds: we assume for now that H is constant (and it can be proved that if Y_t is unpredictable, there is no advantage to having H vary between years). Then if H is the annual fraction that hatch, the population dynamics of the seedbank are

$$n(t+1) = [HY_t + (1-H)s]\, n(t). \tag{2.1}$$

This may seem like an odd way to specify an evolutionary model: where are the genes, Mendel's Laws, and the fitness differentials that drive evolution? But equation (2.1) has all the necessary information, if we adopt the simplifying assumption that "like begets like": all offspring have the same H as their parent. This can be made somewhat respectable by calling it a haploid asexual model: selection among competing clones. Each clone obeys (2.1) with its own value of H, and the changes in relative clone abundance translate into changes in genotype frequencies. So under like-begets-like, the winning H (which we denote H^*) is the one that results in the highest long-run population growth rate. Later in this chapter we discuss the limitations of this approach – for now we can view it as a useful simplifying assumption for a first attack on the problem.

So we have two jobs to do: we have to compute the long-run growth rate of a population obeying (2.1), and then figure out which value of H maximizes it, in order to identify the conditions in which be "winning" H is < 1. So first we have to review some probability theory.

Exercise 2.1 (a) Give one unstated simplifying biological assumption that is necessary for (2.1) to be valid as a description of population growth for our hypothetical desert annual plant with a seed bank ("simplifying" meaning, in particular, that the assumption is not likely to be exactly true in reality). (b) Propose a more realistic assumption, and derive the resulting replacement for (2.1) that applies under the more realistic assumption.

2.2 Random variables review

The models we consider are based on viewing environmental conditions, and the birth and death rates that result from them (such as Y_t), as *random variables*: quantities whose value cannot be predicted in advance with certainty. Instead, there is a range of possible outcomes, and associated with each is a *probability* of its occurrence. Random variables come in two flavors, discrete and continuous.

A **discrete random variable** X has a list of possible values $\{x_1, x_2, x_3, \cdots\}$ and is characterized by its density function $p_k = p(x_k) = P[X = x_k]$.

A **continuous random variable** X can take any value in some interval (L, U) which may be finite or infinite in length, and is characterized by its density function $p(x)$ which has the property that

$$P[a \leqslant X \leqslant b] = \int_a^b p(x)dx \tag{2.2}$$

Some important continuous random variables are the **Uniform**, **Normal** (also called **Gaussian**) and **Exponential**.

- Uniform on $[a, b]$: $p(x) = 1/(b-a), a \leq x \leq b$.
- Exponential with rate λ: $p(x) = \lambda \exp(-\lambda x), x \geq 0$.
- Gaussian(μ, σ^2): $p(x) = \frac{1}{\sqrt{2\pi}\sigma} \exp(-x^2/(2\sigma^2))$

Two random variables X, Y are **independent** if

$$P[X \in A, Y \in B] = P[X \in A]P[Y \in B]. \tag{2.3}$$

This says that X and Y do not interact: the relative frequency of different outcomes for Y is unaffected by the value of X.

The **mean** or **expected value** of X, denoted by $E[X]$, μ_X, or $\bar{X}$, is computed as

$$E[X] = \sum_j p_j x_j \text{ (discrete)}, \ \text{E[X]} = \int_L^U xp(x)dx \text{ (continuous)} \tag{2.4}$$

The mean represents the long-term average over many repetitions of the same "random experiment".

The **variance** of X, denoted Var(X) or σ_X^2, is

$$\sigma_X^2 = E[(X - \mu_X)^2]. \tag{2.5}$$

The square root of the variance, σ_X, is called the **standard deviation**. These measure how far X tends to stray from its mean value. The standard deviation is on the same scale (same units) as the variable itself; the variance has the units of the variable squared.

Rules about Means and Variances

1. If c is a constant, $E[c] = c$ and $Var(c) = 0$.
2. $E[cX] = cE[X]$ for any constant c.
3. $E[X_1 + X_2 + \cdots X_n] = E[X_1] + E[X_2] + \cdots E[X_n]$ **always**.

4. $E[X_1X_2\cdots X_n] = E[X_1]E[X_2]\cdots E[X_n]$ **if the X_i are mutually independent.**
5. $Var(X) = E[X^2] - E[X]^2$ (so $E[X] = 0 \Longrightarrow Var(X) = E[X^2]$)
6. $Var(cX) = c^2Var(X)$ for any constant c
7. $Var[X_1+X_2+\cdots X_n] = Var[X_1]+Var[X_2]+\cdots Var[X_n]$ **if the $X_i$are mutually independent.**
8. $(X - \mu_X)/\sigma_X$ has mean=0, and variance=1.

The Law of the Unconscious Statistician: the random variable $f(X)$ has mean given by

$$E[f(X)] = \sum_j p_j f(x_j) \text{ (discrete)}, \; E[f(X)] = \int_L^U f(x)p(x)dx \text{ (continuous)} \tag{2.6}$$

We'll be applying this soon to the logarithm of population growth rate.

Small Variance Approximations: if $0 < \sigma_X \ll 1$, then

$$E[f(X)] \approx f(\mu_X) + \frac{1}{2}f''(\mu_X)\sigma_X^2 \tag{2.7}$$

and

$$Var(f(X)) \approx f'(\mu_X)\sigma_X^2 \tag{2.8}$$

Derivation: from Taylor Series expansion for f about μ_X:

$$f(x+\varepsilon) = f(x) + f'(x)\varepsilon + \frac{f''(x)}{2!}\varepsilon^2 + \cdots . \tag{2.9}$$

For a random variable X, let

$$z = (X - \mu_X)/\sigma_X .$$

Then we have $X = \mu_X + \sigma_X z$, and our rules for means and variances imply that z has mean 0, and variance 1. Then using the first 3 terms in the Taylor series with $x = \mu_X, \varepsilon = \sigma_X z$ we have

$$f(X) \doteq f(\mu_X) + f'(\mu_X)\sigma_X z + \frac{f''(\mu_X)}{2}\sigma_X^2 z^2. \tag{2.10}$$

Then to get (2.7) we take the mean of both sides of (2.10). Deriving (2.8) is even easier: keep only the first two terms in (2.9) and compute the variance of both sides.

Jensen's Inequality If $f''(x) \le 0$ for all possible values of X, then $E[f(X)] \le f[E(X)]$ (with strict inequality if $f'' < 0$ and $Var(X) > 0$)

The SVA says that Jensen's holds for small variance, but in fact it is always true and says that diminishing returns favors risk-averse behavior (constant reward $E[X]$ beats variable reward X).

Exercise 2.2 (a) Derive the small-variance approximations for $E[e^X]$ and $E[\sin(X)]$, where X is a Gaussian random variable with mean $E[X] = 1$ and variance σ^2. (b) Use simulations to see how well this works. That is, for a range of σ values going from "small" to "large", estimate $E[e^X]$ and $E[sin(X)]$ by the average over 50,000 randomly generated values of X (using **rnorm**), and explore how the error of the small variance approximation drops as σ goes down. A graphical summary of your results (with verbal interpretation and conclusions) will be especially effective.

2.2.1 Limit theorems for random variables

Let $\{X_1, X_2, \cdots\}$ be **independent, identically distributed** random variables with common mean μ and variance σ^2. Then our rules above give,

$$E\left[\frac{X_1 + X_2 + \cdots X_n}{n}\right] = \mu, \quad Var\left[\frac{X_1 + X_2 + \cdots X_n}{n}\right] = \sigma^2/n.$$

Note that the mean is constant, but the variance goes down, as more and more independent X's are added. As a result we have (though it takes more to prove them):

Strong Law of Large Numbers: $\left[\frac{X_1 + X_2 + \cdots X_n}{n}\right] \to \mu$ as $n \to \infty$.

Weak Law of Large Numbers: For any $\varepsilon > 0$, $P\left[\left|\frac{X_1+X_2+\cdots X_n}{n} - \mu\right| > \varepsilon\right] \to 0$ as $n \to \infty$.

We can also scale the sum so that the variance remains constant, in which case the variability of the sum tends to a limiting shape as we add more and more independent terms.

$$E\left[\frac{X_1 + X_2 + \cdots X_n - n\mu}{\sigma\sqrt{n}}\right] = 0, \quad Var\left[\frac{X_1 + X_2 + \cdots X_n - n\mu}{\sigma\sqrt{n}}\right] = 1. \tag{2.11}$$

Central Limit Theorem. As $n \to \infty$, the distribution of $\left[\frac{X_1+X_2+\cdots X_n-n\mu}{\sigma\sqrt{n}}\right]$ converges to $N(0,1)$,the Normal distribution with mean=0, variance=1.

These limit theorems can be summarized informally by writing

$$X_1 + X_2 + \cdots X_n \sim n\mu + \sqrt{n}\sigma Z \tag{2.12}$$

where Z is an Gaussian random variable with mean 0 and variance 1. The meaning of $\sim$ is that the relative difference between the two sides goes to zero; the *absolute* difference generally does not go to zero.

2.3 How to gamble if you're a seed

We can now return to our decision problem for dormant seeds. Denote $\lambda(t) = [HY_t + (1-H)s]$, so that

$$n(t+1) = \lambda(t)n(t). \tag{2.13}$$

Given $n(0) = n_0$ we then have

$$\begin{aligned} n(1) &= \lambda(0)n(0) \\ n(2) &= \lambda(1)n(1) = \lambda(1)\lambda(0)n(0) \\ &\vdots \\ n(t) &= \lambda(t-1)\lambda(t-2)\cdots\lambda(0)n(0) \end{aligned} \tag{2.14}$$

Since the Y's are independent the λ's are too. So if we define $\bar{\lambda} = E[\lambda]$ we have (by taking the mean of both sides)

$$E[n(t)] = \bar{\lambda}^t n_0 \tag{2.15}$$

This suggests that the key quantity for long-term population growth is the average annual growth rate $\bar{\lambda}$ – *but it isn't!* To see why we have to take logs in the last line of (2.14). The result is

$$\log n(t) = \log \lambda(t-1) + \log \lambda(t-2) \cdots + \log \lambda(0) + \log n(0) \tag{2.16}$$

Then dividing by t, we have

$$\frac{1}{t}\log(n(t)) = \frac{1}{t}\log(n(0)) + \frac{1}{t}\sum_{j=0}^{t-1}\log(\lambda(j)) \to E\log(\lambda(t)). \tag{2.17}$$

Thus $\log n(t) \sim \rho t$ where $\rho = E[\log \lambda(t)]$. A population will grow if $\rho > 0$, and decline to extinction if $\rho < 0$.

Note that if $E[\lambda] > 1$ but $E[\log \lambda] < 0$ (which is possible) the expected population size $E[n(t)]$ grows to infinity, despite the fact that the population converges to 0 since $\log n(t) \to -\infty$ (Lewontin and Cohen 1969). How is this possible? Think of "expected population size" as the average over "many" populations growing in separate patches with their own independent $\lambda_i(t)$. Eventually all of them go extinct ($n_i(t) \to 0$). But at any finite time, some of them are "lucky" and have very high population densities. The Weak Law of Large Numbers tells us that as time goes on, the fraction of "lucky" patches drops to zero. So the fact that the average keeps growing means that as time goes on, you have a smaller and smaller fraction of the populations doing weirder and weirder things, before crashing to 0 in the long run. Figure 2.3 shows an example, 250 independent runs where $\lambda(t)$ took the values 0.7 and 1.35 with equal probability, so $E[\lambda] = 1.025, E[\log \lambda] \doteq= -0.03$. The eventual tendency is downward, but at any given time a few oddball runs cause the mean to go up.

Exercise 2.3 (a) Use the Central Limit Theorem to show that the asymptotic (large-t distribution of $n(t)$ is lognormal: that is, $\log n(t) \sim N(\mu_t, \sigma_t^2)$ where N is a Normal (Gaussian) random variable with mean μ_t and variance σ_t^2 (the subscripts indicating that the mean and variance depend on time t). What are the formulas for μ_t and σ_t in terms of the model parameters and the initial conditions? (b) How would you go about testing this prediction in the field? (be optimistic – assume you can do any population sampling etc. required to get the data that you want – but limit yourself to the typical 3-year time span of a research grant).

2.3.1 Computing ρ

1. If $\lambda(t)$ is discrete: possible values $\lambda_1, \lambda_2, \cdots \lambda_n$ with probabilities $p_1, p_2, \cdots p_n$. Then

$$\begin{aligned} \rho &= E[\log \lambda] = \sum_i p_i \log \lambda_i \\ \text{hence } e^\rho &= e^{\sum_i p_i \log \lambda_i} = \prod_i e^{p_i \log \lambda_i} = \prod_i (e^{\log \lambda_i})^{p_i} = \prod_i \lambda_i^{p_i} \end{aligned} \tag{2.18}$$

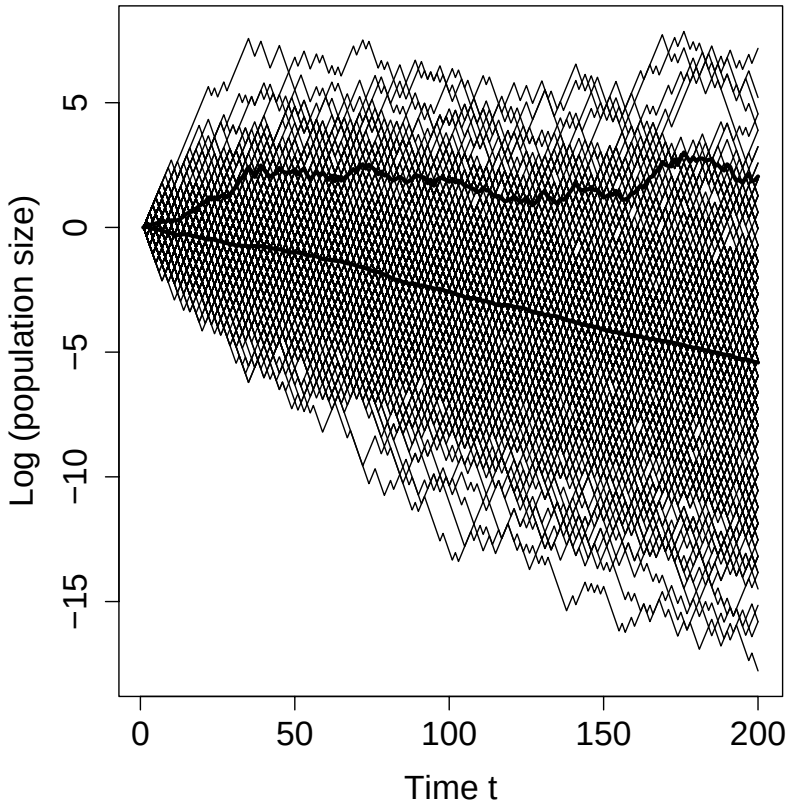

Figure 2.3: Random-environment model where $\lambda(t)$ took the values 0.7 and 1.35 with equal probability. The thin lines are $\log n(t)$ from 250 replicated simulations. The increasing thick line is the log of the average population size, the decreasing thick line is the average of the log population size.

This last quantity is known as the *geometric mean* of λ.

Example:

$$\lambda = \begin{array}{ll} \bar{\lambda}+\sigma & \text{with probability } 1/2 \\ \bar{\lambda}+\sigma & \text{with probability } 1/2 \end{array} . \tag{2.19}$$

In this case $E[\lambda] = \bar{\lambda}$, and $Var(\lambda) = \sigma^2$. Then

$$\begin{aligned} e^{\rho} &= (\bar{\lambda}+\sigma)^{1/2}(\bar{\lambda}-\sigma)^{1/2} = \sqrt{\lambda^2-\sigma^2} \\ \rho &= (1/2)\log(\lambda^2-\sigma^2) \end{aligned} \tag{2.20}$$

So a bad year hurts more than a good year helps, when their deviations from the mean are equal in magnitude. If the stock market goes up 10% one day and down 10% the next day (or vice versa), you have less money than you started with. You learned long ago that $(1+x)(1-x) = 1-x^2$, and now you

know what it means for plants.

2. If $\lambda(t)$ is continuous with density $p(\lambda)$: Then by the Law of the Unconscious Statistician,

$$\rho = \int_0^\infty \log(\lambda) p(\lambda) d\lambda. \tag{2.21}$$

Equation (2.21) is typically useless since you can't evaluate the integral. So one can resort to the Small Variance Approximation with

$$f(x) = \log x, f'(x) = 1/x, f''(x) = 1/x^2$$

which gives

$$E[\log \lambda] \doteq log(\bar{\lambda}) - \frac{\sigma^2}{2\bar{\lambda}^2}. \tag{2.22}$$

where σ^2 is the variance of λ. This is pretty crude (because it makes no use of the distribution of λ) but it conveys the essential information: *variance hurts*.

"Bet hedging" is the general term for strategies that reduce the variance of rewards, at the price of a reduction in the mean reward. Phillipi and Seger (1989) identify two categories of bet-hedging: conservative and diversified. A conservative strategy for a shrub to deal with drought, for example, would be to store up water when it's available rather than using it all for immediate seed production, even if there is a cost to storage that reduced the number of seeds that can be produced per unit of water taken up. A diversified strategy involves some variability of behavior at the individual level; at the level of the genotype (present in multiple individuals) this results in "not putting all your eggs in one basket".

2.3.2 Optimal seeds

Our seedbank/eggbank model is a diversified strategy: some gamble on this year being good, others sit it out and try again later. The general theory above says that the winning H is the one which maximizes

$$\rho = E \log \left[HY_t + (1-H)s \right]. \tag{2.23}$$

To see what H this is, we differentiate within the expectation and do some curve-sketching of ρ as a function of H for $0 < H < 1$. [The expectation is either a sum or an integral, and in either case it is legitimate to move differentiation inside the operation of summing/integrating].

$$\rho'(H) = E\left[\frac{Y_t - s}{HY_t + (1-H)s}\right] \qquad \rho''(H) = E\left[\frac{-(Y_t - s)^2}{(HY_t + (1-H)s)^2}\right] < 0 \tag{2.24}$$

Because of the uniformly negative second derivative, there there are only 3 possibilities (see Figure 2.4) which can be distiguished based on the signs of $\rho'(0)$ and $\rho'(1)$:

1. $\rho(H)$ is decreasing on [0,1]. This occurs if $\rho'(0) < 0$, i.e. if $E(Y_t) < s$, and the optimum strategy is $H = 0$ giving population growth rate $\rho(0) = \log(s) < 0$, implying that all types decrease to

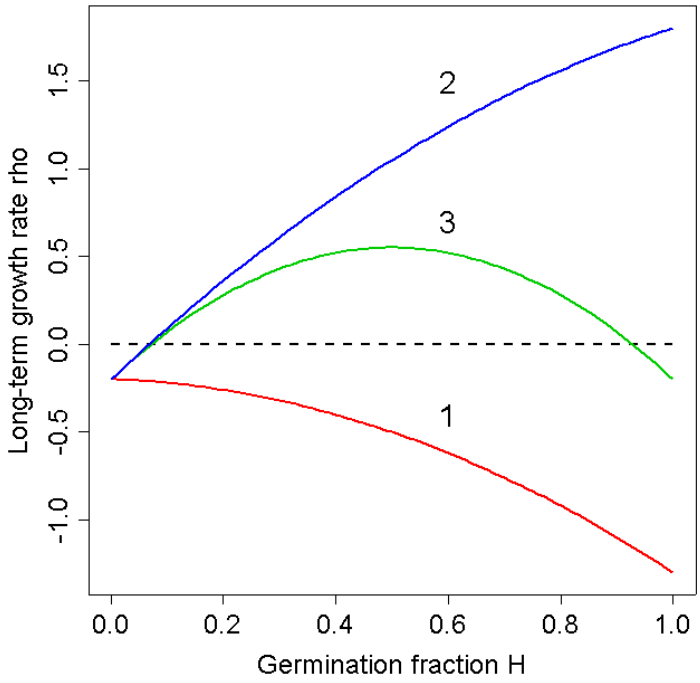

Figure 2.4: Curve-sketching $\rho(H)$ for Cohen's (1966) model. Because $\rho(0) = \log(s) < 0$ and $\rho''(H) < 0$, there are only three qualitative possibilities determined by the sign of ρ' at 0 and 1.

zero abundance. So this possibility is actually not possible, and any real population must have $\rho'(0) > 0$.

2. $\rho(H)$ is increasing on [0,1], in which case the optimum strategy is $H = 1$, all hatch at the first opportunity.

3. $\rho(H)$ has a maximum at some H^* between 0 and 1, so the optimal strategy is to bet-hedge: some hatch each year and some wait until later.

We can tell which of the two possibilities holds by looking at the sign of $\rho'(1)$: The optimum is at $H = 1$ if $\rho'(1) = 1 - sE[1/Y_t] \geq 0$ and at some H^* between 0 and 1 if $\rho'(1) < 0$. Thus we found when some seeds should "sit it out" each year:

$$H^* < 1 \text{ if and only if } sE[1/Y_t] > 1. \tag{2.25}$$

Having derived condition (2.25), we need to make sense of it. There are two special cases that usually help: *small variance*, and *good years/bad years*.

Exercise 2.4 Use the small variance approximation to derive that

$$E[1/Y_t] \doteq \frac{1}{\bar{Y}} + \frac{\sigma^2}{\bar{Y}^3} = (1/\bar{Y})[1 + (\sigma/\bar{Y})^2] \tag{2.26}$$

By substituting the result of the Exercise above into (2.25), we have the "small fluctuations" approximate condition for $H^* < 1$:

$$(s/\bar{Y})\left[1 + (\sigma/\bar{Y})^2\right] > 1. \tag{2.27}$$

$\bar{Y}$ must be > 1 (otherwise ρ would be negative for any H by Jensen's Inequality), so $(s/\bar{Y}) < 1$. The interpretation of (2.27) is therefore that all seeds should hatch each year if the variance in reproductive success is small, while if the variance is large some should sit it out. *Note the paradox here*: we derive the condition by assuming small variance, and interpret what it means when the variance is large. Don't let this bother you – we're going for qualitative insight, not mathematical rigor. As with any approximation, you check your insights on the computer to see if the model really acts like you expect it to.

The *good years/bad years* case is to suppose that $Y_t = M$ or m with probabilities p and $1-p$ respectively, where $m \ll 1 \ll M$. Then from (2.24) we calculate directly

$$\rho'(H) = p\left[\frac{M-s}{HM+(1-H)s}\right] + (1-p)\left[\frac{m-s}{Hm+(1-H)s}\right] \doteq \frac{p}{H} - \frac{1-p}{1-H} \tag{2.28}$$

using the assumption $m \ll s \ll M$ to simplify the fractions. Thus the optimum is approximately at $H = p$: the fraction of seeds that hatch should be approximately equal to the fraction of good years, p.

Since $\bar{Y} > 1$ but $s < 1$, the *expected* number of seeds next year (new plus survivors) is always maximized at $H = 1$. Thus the "decision" to have $H < 1$ when the variability in Y is large, really is a bet-hedging strategy: the mean payoff is reduced, but in the long run it pays off because it reduces the variance.

Exercise 2.5 Verify the statements above about the effect of H on the mean and variance of the population growth rate $\lambda(t) = HY(t) + (1-H)s$, i.e. show that the mean and variance of $\lambda(t)$ are both increasing functions of H [HINT: these can both be done using the rules on mean and variance listed in section 2.2].

2.4 Limits to Growth and Evolutionary Game Theory

One unrealistic aspect of Cohen's model is that population density increases without limit for many "losers" as well as the "winner" phenotype. This behavior comes from the simplifying assumption that per-capita fecundity Y is unaffected by crowding. One has to start somewhere and often a density-independent model is a good place to start. But given that there always are limits to population growth imposed by crowding, it's important to see if a model with such limits makes different predictions.

The traditional way of modeling limits to growth is to assume that each individual's per-capita reproductive success is affected by the overall density of competitors, say $Y_t = K_t F(N_t)$ where K_t is random and $N_t = \sum_i H_i n_i(t)$ is the total number of seeds emerging from dormancy (i running over all competing types in the population). The key simplifying assumption is that the population is well-mixed in space, i.e. spatial variation in density is small enough to ignore.

Because strategies are now pitted against each other, instead of an "optimal" strategy we seek a "evo-

lutionarily stable" strategy or **ESS**: a value H^* such that any competing type with a different H is at a disadvantage when type H^* dominates the population. Germination is then an *evolutionary game* in the sense of mathematical game theory: a contest where the payoff to each player depends not only on its own behavior, but also on what other players do. An ESS as defined above is closely related to the *Nash equilibrium* solution concept of mathematical game theory. A Nash equilibrium is a strategy (or set of strategies for all players) such that no player can do better by making a unilateral change of strategy.

Formally, let $\tilde{Y}_t$ denote the value of Y_t in a population consisting entirely of type-H individuals. Then the logic behind equations (2.13) and (2.17) still applies to give the growth rate of a rare type h invading an otherwise all-H population:

$$\rho(h|H) = E \log \left[h\tilde{Y}_t + (1-h)s \right] \tag{2.29}$$

This approach is a shortcut called an "invasibility analysis" that avoids having to analyze competition between two competing types. Instead we consider a "resident" tye H that has been established in the habitat for a long time, faced with a mutant "invader" h. We assume that the invader is at such low density that H goes on as if the mutant were not present: the resident affects the invader, but not vice-versa. If H is such that no invader can increase from its initial rare density, then H is called an **ESS** (evolutionarily stable strategy). This is much easier than analyzing competition, and usually gets the right answer(see Chesson and Warner 1981, Ellner 1985, Chesson and Ellner).

Note that $\rho(H|H) = 0$ (WHY? – no math needed, just a bit of insight). The condition for H to be an ESS is that

$$\rho(h|H) < 0 \text{ for } h \neq H,$$

hence H^* is an ESS if $\rho(h|H^*)$, considered as a function of h, has its maximum at $h = H^*$. That is: an ESS is defined by the property that it is the best invader of itself. As in Cohen's model we have

$$\frac{\partial \rho}{\partial h} = E\left[\frac{\tilde{Y}_t - s}{h\tilde{Y}_t + (1-h)s} \right], \quad \frac{\partial^2 \rho}{\partial h^2} < 0 \tag{2.30}$$

Because the second derivative is everywhere negative, for any H there is a single "best" invader. For a possible ESS $0 < H^* < 1$, the ESS property of being the best invader of one's self can be written as:

$$\partial\rho/\partial h = 0 \text{ at } h = H = H^*. \tag{2.31}$$

A more modest goal is to find when some dormancy is favored, i.e. under what conditions $H^* < 1$. As in the density-independent we do this by asking whether $H = 1$ can be invaded by $h < 1$, i.e., the condition for some dormancy to be favored is

$$\frac{\partial \rho}{\partial h} < 0 \text{ at } h = H = 1. \tag{2.32}$$

The formal calculations are the same as before and the result is equation (2.25) with Y_t replaced by the $\tilde{Y}_t$ for $H = 1$.

To proceed we will choose a convenient form of density dependence. In Cohen's model we assumed that the number of offspring in a given year was unlimited: each seed that germinates produces Y_t new seeds.

Twice as many parents will produce twice as many offspring. As an opposite extreme, we can assume instead that the number of offspring in each year is strictly limited, regardless of the number of parents trying to reproduce.

Let K_t be the number of seeds produced in year t. If the population consists of a single resident with $H = 1$, then the number of seeds germinating in year t is exactly the number produced last year, i.e. K_{t-1} and the number of offspring per germinating seed is then $\tilde{Y}_t = K_t/K_{t-1}$.

Substituting into (2.30) and setting $h = H = 1$, the condition becomes for dormancy to be advantageous becomes

$$sE[K_t]E[1/K_t] > 1. \tag{2.33}$$

Exercise 2.6 Verify that (2.33) is correct.

Exercise 2.7 Derive the small-variance approximation for (2.33) in order to express the condition in terms of the mean and variance of K, and interpret what it predicts qualitatively about the conditions in which dormancy is favored ($H^* < 1$).

Exercise 2.8 Derive a good years/bad years approximation for for (2.33), along the lines of what we did above for Cohen's model ($K_t = M$ or m with$m \ll M$, and keep only the dominant term(s)).

Exercise 2.9 Explain why the good years/bad years approximation leads to the prediction that $H^* = 1$ if good years are either frequent or very infrequent, while some dormancy is favored if good and bad years are both frequent. Why does this make sense? – give an *intuitive, nonmathematical* explanation from the viewpoint of a seed deciding whether to germinate "now" versus waiting for next year.

2.4.1 Proceed with caution

We know that evolution involves

- Gene frequency changes governed by Mendelian genetics in a finite population.
- Competition among a suite of genotypes for different trait values.

To do an ESS analysis we *pretend* that it involves

- "Like begets like": uniparental clonal reproduction.
- Pairwise competition between an established "resident" and a rare "invader", ignoring effects of the invader on the resident.

These gambits don't always succeed.

- "Like begets like" is especially dubious in situations involving conflicts among offspring or between parent and offspring, such as allocation of resources within the family. Any time the interests of relatives don't coincide, it is essential to explicitly model the power structure.

- An ESS can't be dislodged once it is established, but that doesn't guarantee that evolution will move the population to the ESS (e.g. Eshel and Motro 1981, Takada and Kegami 1991). A strategy x^* with the latter property is called a **CSS** (continuously stable strategy). For a single strategy parameter, a CSS is defined by the property that if x is near x^* and y is between x and x^*, then y can invade x. The CSS and other related stability concepts are reviewed by Levin and Mueller-Landau (2000).

- The focus on the expected long-term growth rate of an invading strategy can be misleading when populations are small. The "coin tossing" randomness of individual survival and breeding then becomes important, and models based on the probability of a rare allele becoming fixed in the population then do a better job of predicting evolutionary dynamics (Proulx and Day 2001).

Because of these possible difficulties, an essential step in an ESS analysis is to check that all the tacit shortcuts have not led you into error. The available checks are general theory, special cases and simulation. "General theory" is a set of results giving conditions under which an ESS analysis agrees with the outcome of evolution in a mechanistic genetic model (e.g., Taylor 1989, Charlesworth 1990, Day and Taylor 1996, 1998). Almost all of these rely on weak selection approximations, so they offer comfort but not certainty. Convenient special cases of the genetics can often be used to check the "like begets like" gambit by raising the likely complications in the simplest possible setting (e.g., a diploid model with a few loci, a few alleles, but strong selection). However the pairwise competition gambit usually has to be checked by simulation, because multi-type competition models are high-dimensional and there is no general theory to help you with that.

Project Exercise 2.10 Check your conclusions for the good-bad years case by writing a program to simulate competition among multiple (10-25) types with different H values; run it first at parameter values where $H = 1$ should win, and then at parameters where $H = 1$ should lose, according to the criterion under the good-bad years approximation.

2.5 Empirical tests

2.5.1 Desert seeds

Phillipi (1993a) tested 3 predictions from Cohen's model on 6 desert annual species in the Southwestern US:

1. Of the seeds that do not germinate in their first year (under normal field conditions with adequate water), some should germinate in their second year under identical conditions.

2. The fraction germinating the first and second years should be equal

3. If first-year germination is prevented by bad conditions (a "no water" treatment in his experiments), this should not affect the germination fraction in the next year.

To be more precise, in the following experimental design

	Year 1	**Year 2**
Treatment 1	Good	Good
Treatment 2	Bad	Good

the observed value of H should be the same in all three "Good" trials.

Results:

Prediction 1: yes in 6 of 6 species
Prediction 2: no (either lower or higher
Prediction 3: no (either lower or higher)

Phillipi (1993b) looked at one species *Lepidium lasiocarpum* across multiple sites, to assess the response of H to environmental conditions.

1. H should be higher where average rainfall is higher ("good" years more common): *Yes, but only a very weak effect, and germination rate remained nearly constant over a wide range of average rainfall.*
2. There is a stronger, negative correlation between H this year, and the rainfall in the year when the seeds were produced.
3. Seeds produced by large mother plants were less likely to germinate in their first year.

All of these tend to favor the "limits to growth" ESS models. The first one is consistent with those models, and not with the basic prediction $H \approx p$ in the density-independent model. Clauss and Venable (2000) note that in general, experimental studies have not uniformly supported the prediction H should be higher in seeds from populations with a higher fraction of "good" years. In fact, sometimes the trend is the reverse of the prediction.

One interpretation of Phillipi's second and third results is that seeds are responding to "cues" about the density of competitors. If last year was good, or if "your" mother was large and therefore produced many seeds, "you" are probably surrounded by a lot of other seeds, so it's a good idea to stay dormant until the crowd thins out. Models bear this out (but we need to move on, so you'll have to take this on trust): in a density-dependent setting where seeds can estimate how crowded it is, Phillipi's observations are properties of the ESS.

Clauss and Venable (2000) focused on the response of seeds to conditions at the time of germination. They extended Cohen's model by assuming that years come in 3 types: *No Rain, Good, Bad*. In a No Rain year, seeds just sit it out. In a Good year, early rains that allow germination are followed by sufficient late rain to allow seed set. In a Bad year, early rains are not followed by adequate late rain. The prediction that they derive for this model is

$$H^* = \frac{P(G|r)Y - s}{Y - s} \approx P(G|r)$$

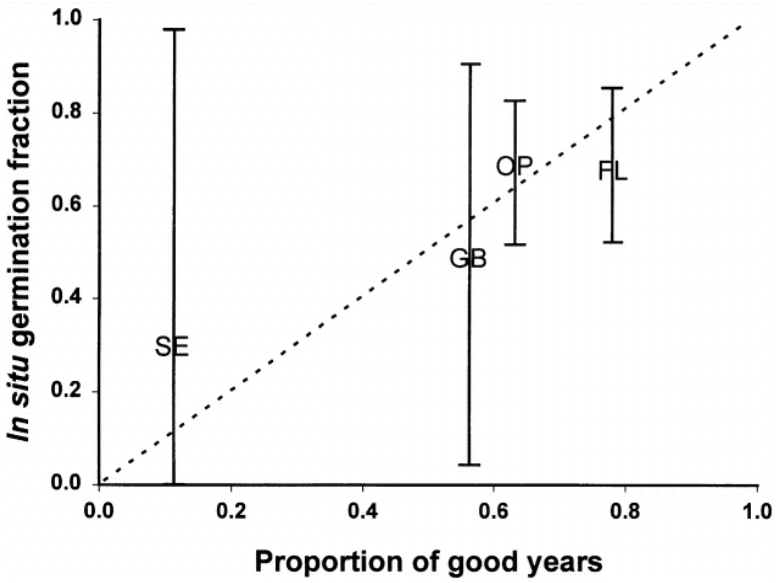

Figure 2.5: From Clauss and Venable (2000), comparing predicted values of H^* with actual mean germination rates in the field.

where $P(G|r)$ is the probability that the year is good, given that some early rains occur, and Y is the per-capita reproductive success in a good year.

Comparing this prediction with field-germination rates in the desert annual *Plantago insularis*, the results were quite good (Figure 2.5). Clauss and Venable (2000) reported that a density-dependent version of their model gave similar predictions to the density-independent model whose predictions are plotted, but don't go into details.

On the other hand, they found that under "common garden" conditions in the lab, there was **no** tendency for seeds from better (more good year) sites to have a higher annual germination rate. In fact, seeds from drier sites germinated under less restrictive conditions than seeds from wetter sites. So the pattern in Figure (2.5) resulted from the interaction of intrinsic seed properties with conditions in the field – laboratory tests of seeds' propensity to germinate may be totally misleading about germination rates in the field. Clauss and Venable (2000) suggest that this might account for the inconsistent results in lab-experiment tests of bet-hedging models.

2.5.2 Freshwater copepod eggs

There are (at least) two decision problems in the life-cycle of the freshwater copepod *Diaptomus sanguineus* (Figure 2.6). The life-cycle of *Diaptomus* in Bullhead Pond, RI, is summarized in Figure 2.7, based on experimental work over nearly two decades by Nelson Hairston, Jr. and collaborators (summarized in Ellner et al. 1999).

Figure 2.6: A female *Diaptomus sanguineus* carrying a clutch of eggs. This individual is about 1.5mm long. Picture courtesy of N.G. Hairston, Jr.

Eggs hatch in the Fall, mature, and lay eggs that will hatch immediately and produce a second adult cohort. Second-cohort females begin by doing the same, but switch in the Spring to making diapausing eggs that will remain dormant at least until the next Fall. Diapausing eggs are safe from predation by fish, which quickly intensifies when the pond warms up in Spring. It is not too inaccurate to imagine that fish suddenly becoming active on a single "catastrophe date" each year. The catastrophe date varies widely from year to year and there is no apparent way to predict it in advance.

So for *Diaptomus* females, a very early switch date is sure to yield at least some diapausing eggs before fish become active. But a late switch date is a gamble on when the catastrophe date will come this year. If it comes late, she keeps on pumping out eggs that hatch and produce many daughters and grand-daughters before predation intensifies. But if it comes early, the female and her (still active) offspring suffer high mortality and leave few descendents. Selection against those who guess wrong is strong enough to cause year-to-year changes in mean switch date – it shifts earlier after a year of low predation, and later after a high predation year.

We initially studied the system in order to understand the high levels of heritable (i.e. genetically determined) variation in switch date among females (Figure 2.8), which could not be explained by the then-current theory from population genetics (Hedrick 1986, Bull 1987, Karlin 1988, Turelli 1988, Barton and Turelli 1989, Frank and Slatkin 1990, Gillespie 1991). We developed models that could explain this (which is another story), and then to test our models made some predictions about differences in switch-date distribution between descendants of active females in the water column, and descendants of eggs buried in the pond sediments. Diapausing eggs that remain dormant are eventually covered by the accumulation of sediment at the bottom of the pond, and cannot hatch unless they are mixed up to the

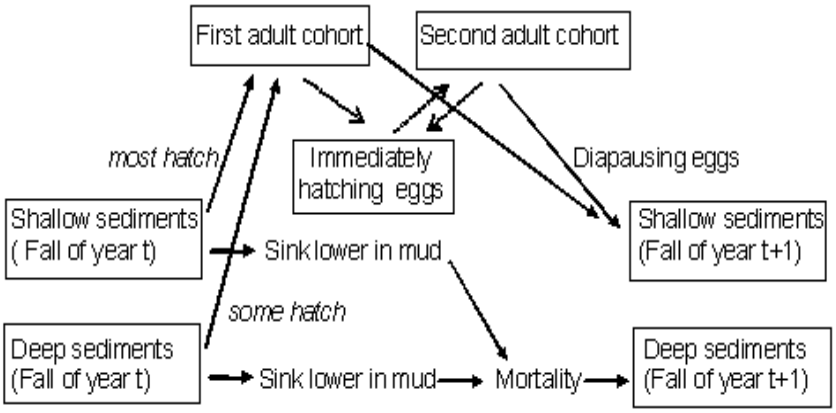

Figure 2.7: Schematic life cycle of Diaptomus sanguineus in Bullhead Pond, RI. Eggs hatch in late fall and there are typically two generations completed before predation wipes out the active population in the Spring. The population persists through the summer as diapausing eggs. Eggs that do not hatch the next Fall sink deeper into pond sediments and can only hatch in later years if they are mixed upwards to the surface of the sediment layer, e.g. by fish laying nests.

surface, e.g. by fish building nests or a tree limb falling into the pond and disturbing the sediments. Such buried eggs can survive for centuries: in the lab, 50% hatching success was obtained on eggs buried for 300 years, according to lead-210 dating of the sediments from which they were extracted (Figure 2.9).

Our models (leaving out some early ones that didn't work) are conceptually similar to the seedbank model, but consider two different decision variables: switch date (z) and first-year hatching rate H. Conceptually, the model is

$$n(t+1) = n(t)[HY(z,t) + (1-H)s] \tag{2.34}$$

which is the basic seedbank model except that reproductive success Y is partially determined by switch date z. In particular, we assumed that Y is determined jointly by z and the (unpredictable, random) "catastrophe date" $\theta(t)$) when predation commences in year t.

$$Y(z,t) \propto e^{(-c(z-\theta(t))^2)}$$

with the total number of eggs being higher in years when predation is late ($K(t) = Ke^{a\theta(t)}, \quad a > 0$). This fitness function is shown in Figure 2.10. We mostly used a "limits to growth" version of the model (with a cap on total population size), and for comparisons with data added other features (such as there being two generations per year) – but (2.34) is the gist of all the models.

With these assumptions we got the following predictions:

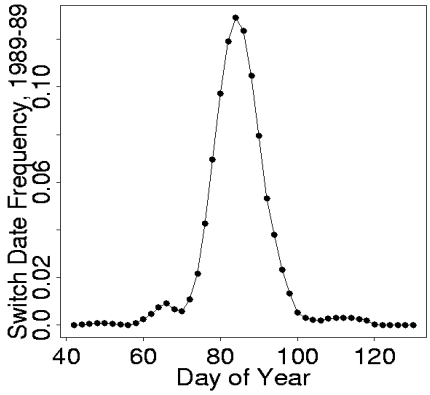

Figure 2.8: Average distribution of switch dates over 10 years in Bullhead Pond, RI, based on cohort sample data of Nelson G. Hairston, Jr.

1. When Var(θ) is low, the ESS is to have z at the average optimum switch date.

2. When Var(θ) is high, there is no ESS. Instead, the population should consist of a discrete set of types $(z_1, H_1), (z_2, H_2), \ldots, (z_n, H_n)$ having distinctly different switch dates and first-year hatching rates.

3. Unless Var(θ) is really, really high, two types coexist with a negative correlation between switch date and hatch rate.

The predicted negative correlation is easy to understand: types with a early early switch date never encounter predation, so every year is the same and they should have a high H. Late-switching types experience a highly variable environment and should have a low H.

We estimated (Ellner et al. 1999) that Var(θ) was high but not really, really high, so the prediction to test was the third one above. Figure 2.11 shows evidence for the existence of two egg types, based on the amount of incubation time required for hatching to occur in the lab, in conditions that would evoke hatching in the field. While suggestive, this is not quite enough, because the difference could simply be a bet-hedging strategy of variable diapause timing: we also need to check whether it lines up with differences in switch date. To do that, offspring of those eggs were assayed for what kinds of clutches they produce, under lab conditions mimicking those in the pond at the average switch date. The results (Figure 2.12) confirm the association: quick hatchers produce a mixture of clutch types, late hatchers

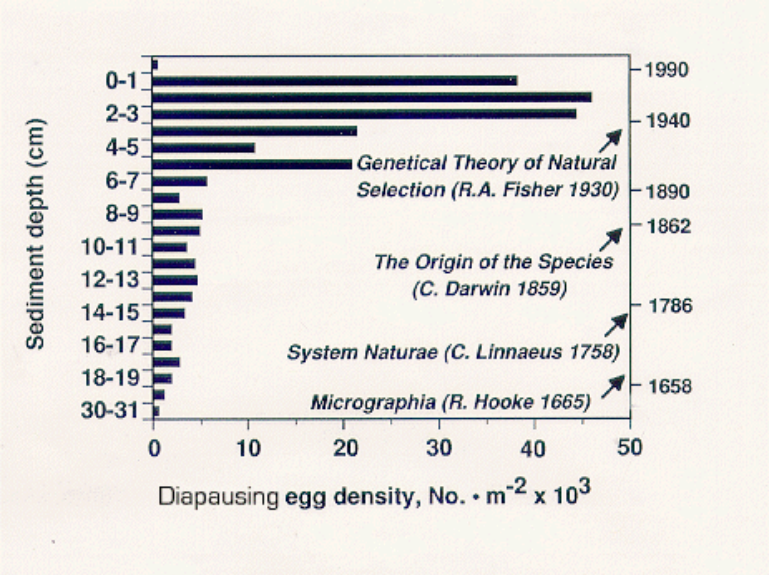

Figure 2.9: Distribution of diapausing eggs buried in the sediments of Bullhead Pond, RI and estimated ages based on lead-210 dating, compared to some landmarks in the history of biology. Figure courtesy of Nelson G. Hairston, Jr.

are overwhelmingly producing diapausing eggs (these figures use data from the experiments reported by Hairston et al. 1996b)

The overall picture is that the copepod population was a mix of genotypes that used different ways of coping with the risk of reproduction in the face of unpredictable predation, one "conservative" and one "diversified". Early-switch genotypes are conservative: as adults they accept the reliable payoff for producing diapausing eggs before things get risky, so they have no reason to bet-hedge as eggs and therefore hatch readily. Late-switch genotypes gamble as adults on the big payoff that comes in years of low predation risk, and therefore play it safe by bet-hedging as eggs, spreading their hatching over many years. A larger lesson from this is that how safe or risky the world is, is not necessarily an intrinsic property: a lot can depends on whether the organism avoids or seeks risky opportunities.

2.6 Reminder: the big picture

As we leave the topic of individual-level decision making, it's again a good time to expand our focus. Foraging theory introduced classical models that ignore variability. The assumed goals involve long-term average rates, and ignore short-term accidents. Dynamic state variable models incorporate variability during the lifetime of an individual, or on the time-scale of daily decision-making. Bet-hedging theory takes account of variability on longer time scales, such that successive generations, or one individual over the course of its lifetime, face conditions that vary unpredictably. To develop the theory we had to learn about population growth under temporally varying conditions, mathematical techniques for

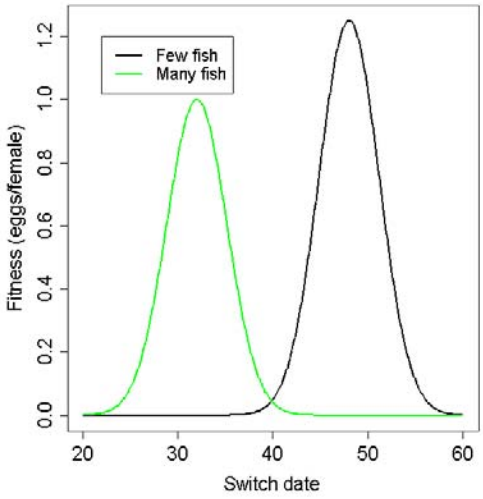

Figure 2.10: Fitness function (eggs per female as a function of switch date) in model for bet-hedging by *Diaptomus sanguineus*. Under intense predation, it pays to have an early switch date; under weak predation it pays to have a late switch date, and the potential reproduction is greater.

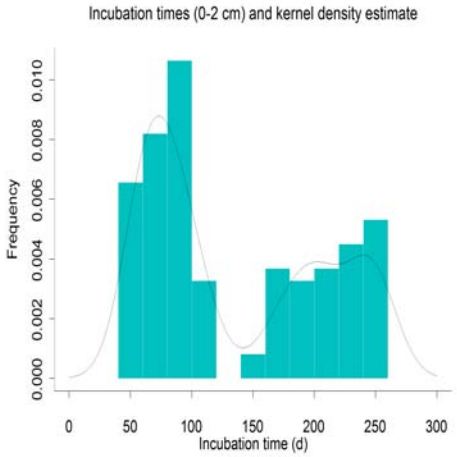

Figure 2.11: Distribution of incubation time (days) required for hatching to occur.

approximating stochastic growth models. Taking account of limits to growth forced us to learn about invasibility analysis and evolutionarily stable strategies (ESS).

All of these general ideas and methods have much broader application. In particular, the ESS approach of evolutionary game theory is the fundamental tool for models of animal behavior whenever interactions

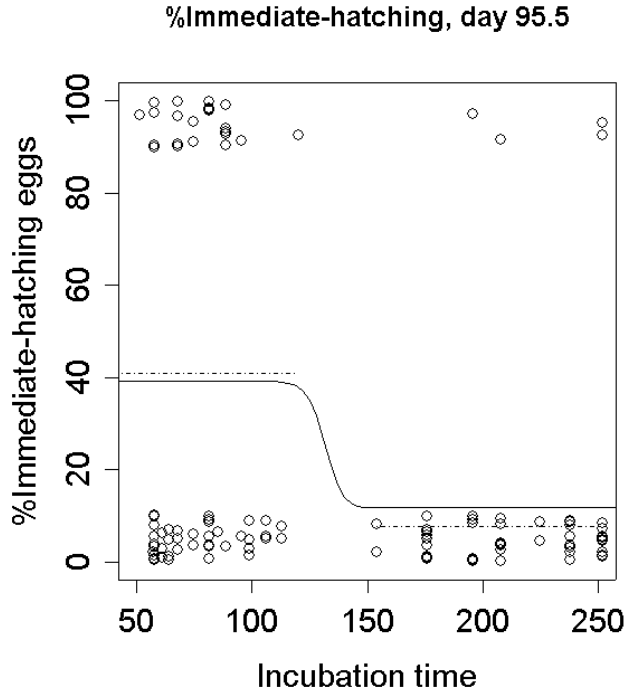

Figure 2.12: Association between incubation time and the fraction of immediately-hatching egg clutches, under lab conditions mimicking those in the field on Julian day 95.5

between individuals are involved. Examples include: contests for food, territory or mates; signaling; coordination of group activities (e.g. sentinel/forage decisions); division of labor in social insects and allocation of reproductive output among individuals; cooperation among unrelated individuals. The book edited by Dugatkin and Reeve (1998) reviews the wide scope of game theory models in the study of animal behavior.

Another major application area is life history theory, as mentioned at the start of this chapter:

- Body size: when to stop growing.
- Age at maturity: when to start allocating resources to reproduction rather than just growth and survival.
- Reproductive lifespan: continuous breeding over many years, versus a single suicidal "spawning".
- Offspring size and number: many small or few large offspring at each breeding?
- Aging: patterns of change in fecundity and survival over time.

As in our models for short-term decision making, these are viewed as solutions that optimize some assumed goal, that is a proxy for long-term representation in the gene pool. As always, optimization

is subject to constraints and tradeoffs, e.g., putting more resources into breeding decreases growth or survival. In a historical development similar to that of foraging theory, life history theory has progressed from classical models that ignore variability, to state-dependent models that take account of within- and between-generation variability in conditions. So although the applications are different, the tools and concepts are the ones that you've seen in this and the previous chapter. Stearns (1992, 2000) and Roff (2001) are good recent reviews of life theory and its applications.

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