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ABSTRACT

THE THEORY OF PHYSIOLOGICAL AGE

DISTRIBUTIONS IN BIOLOGICAL POPULATIONS

By

John Van Sickle

For many species of plants and animals, chronological age is neither convenient nor useful to the population biologist as a measure of individual maturity. A physiological feature of an organism, such as its body size or chemical composition, provides an attractive alternative to chronological age as a frame of reference for discussing life history phenomena. Unfortunately, most demographic population models have been structured on the basis of chronological age.

This thesis is a study of a dynamic population model in which individuals are classified by physiological age. The physiological age profile of the population is represented by a number density function which obeys a first-order partial differential equation.

Solutions of the differential equation are presented. The asymptotic behavior of the model is discussed and well-known stability results for chronological age distributions are generalized to physiological age distributions. The shape of the stable physiological age distribution is shown to be a function of individual growth as well as mortality and fecundity.

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the model is applied to some problems in fish population dynamics. Equations for estimating biomass production are derived from the dynamical equations for the size distribution. A steady-state population size distribution is defined and applied to an exploited fish population previously modeled on the basis of chronological age. A third application shows how the stable size distribution predicted by the model can be used to compute mortality rates from empirical population size-frequency curves and a knowledge of individual growth rates.

Finally, time-varying growth rates are discussed, still in the context of fish populations. Fish are known to respond to changes in available resources by altering their growth rates. A stability analysis and simulations demonstrate how individual growth in fish can regulate population growth in a limited environment.

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THE THEORY OF PHYSIOLOGICAL AGE

DISTRIBUTIONS IN BIOLOGICAL POPULATIONS

By

John $\overset{Allan}{}$ Van Sickle

A DISSERTATION

Submitted to
Michigan State University
in partial fulfillment of the requirements
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DOCTOR OF PHILOSOPHY

Department of Electrical Engineering and Systems Science

1975

DEDICATION

To Jane, with love.

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CHAPTER 1

INTRODUCTION

Quantitative models with a single dynamic variable are inadequate
for a realistic description of most populations. If functional or time
lag terms are included, ordinary differential or difference equations for
total numbers can reproduce many observed population trajectories.
However, such complex equations usually can not be interpreted in a
biologically useful manner.

Much of the problem lies in the assumption of these models that
all members of the population are identical. Numerous field and labora-
tory populations have demonstrated how the heterogeneous composition of
a population profoundly affects its dynamics. Population models must
begin to account for this hetereogeneity.

Oster and Takahashi [1974, p. 483] express this in a different
way by stating that ". . .populations are intrinsically distributed
parameter systems." For populations whose members are differentiated
by chronological age, a highly developed set of distributed parameter
models is available which includes integral equation, partial differ-
ential equation, and discrete matrix forms. Using this set of models
as a theoretical base, demographers have developed a comprehensive
methodology for age-specific parameter estimation and prediction of
future age distributions [Keyfitz, 1968].

There are many ways to classify individuals other than age. Such

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features as individual body size, dietary requirements, or chemical composition might strongly influence how individual birth and death phenomena "sum up" to give population birth and death rates.

Alternate methods of differentiating among individuals become especially important when describing non-human populations. Chronological age is difficult to assess for a great many species of plants and animals, and it is usually more convenient to keep track of other physiological characteristics of individuals.

Even if chronological age can be determined, it is often an unreliable indicator of an organism's sexual maturity, fertility, or its chances for future survival. For most species of invertebrates, plants, and fish, these vital parameters of population growth are strongly dependent on environmental factors such as available food or ambient temperature. For such populations, a physiological, rather than chronological, measure of age or maturity may lead to more realistic and useful models of population dynamics.

This thesis is a study of a distributed parameter model in which individuals are classified by physiological age. It is assumed that these individuals live in a spatially homogeneous population and that immigration and emigration are negligible. Sex differences among individuals are also ignored, since sex ratios tend to remain fairly constant in most populations.

The study has two principal objectives. The first objective is to define and analyze a continuous-time dynamical system for the physiological age distribution of a population. Various forms of the system equations which will be used have already been applied to specific physiological characteristics of specific populations, but the system

will be analyzed in the more general context of an arbitrary measure of physiological age. Chapter 2 of the thesis defines the model and gives a brief review of its previous applications to biological populations. Solutions of the system equations in their autonomous form are presented and interpreted.

Chapter 3 examines the asymptotic (in time) behavior of the autonomous model. A. J. Lotka's familiar stability theory of chronological age distributions is generalized to apply to physiological age structures.

The second main objective is to thoroughly understand the crucial role played by growth in population dynamics. In this thesis, "growth" refers to the dynamic relationship between an individual's physiological age and its chronological age. Individual growth rates become vital parameters along with mortality and fecundity rates. In addition, individual growth is the mechanism through which many population systems are coupled to their environment.

In Chapters 2 and 3, we see how time-invariant growth rates affect the rate of increase of a population and the shape of its physiological age distribution.

Chapter 4 is an application of the theory of Chapters 2 and 3 to fish populations. Individual growth in length or weight has been long recognized as a key factor in fish population dynamics, and body size is employed as a particular measure of physiological age in applying the model to fish. The chapter briefly discusses growth in fish and then goes on to attack three quite different problems of observing and modeling fish populations which are assumed to have time-invariant individual growth rates.

Chapter 5 retains the general context of fish populations classi-
fied by body size, but the emphasis shifts to time-varying growth rates.
Specifically, we study the population in relation to environment by
assuming individual growth is a function of population density. This
growth response to limited food or space is felt by many fishery
biologists to be a powerful regulator of fish population densities. A
perturbation analysis and computer simulations of the model show the
regulatory properties of a flexible growth response.

The arguments of this thesis will show that physiological age
models provide the added dimension of individual growth to population
dynamics at little or no cost in mathematical tractability or ease of
interpretation.

CHAPTER 2

THE MODEL: DEFINITION AND BACKGROUND

2.1 Physiological Age

Suppose z represents some measure of maturity or physiological development of individuals in a population. The variable z could be chronological age, body size, chemical composition, or any other physiological feature having a bearing on individual reproduction or mortality.

The physiological age, z, of an individual is dynamically described by a differential equation:

$$\frac{dz}{dt} \overset{\Delta}{=} g(z,t). \tag{2.1-1}$$

Here, $g(z,t)$ is the growth rate of the physiological age variable, z. The term "physiological age" will be abbreviated by "p-age", and hereafter z will be referred to as a p-age variable.

Entomologists, notably Hughes [1962] and Lefkovitch [1965], were among the first to use stages of maturation, rather than chronological age, in structuring their models. This approach was partly motivated, no doubt, by the discrete, easily recognizable, life stages seen in many insect species.

Other researchers have been attracted to the concept of p-age by the relative ease with which p-age, as opposed to chronological age, can be determined in many organisms. Examples are the early fish population model of Baranov [1918] and the model of Usher [1966] for forest management.

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More recently, the need for generalized age-structured models,
together with the rise of computer simulation as a tool of the population
biologist, has fostered the theoretical development and implementation
of continuous [Striefer and Istock, 1973; Streifer, 1974; Weiss, 1968]
and discrete [Boling, 1973; Coulman, Reice, and Tummala, 1972] models
employing "critical" state variables based on p-age.

2.2 The Population Balance Model

We now introduce a number density function, $N(z,t)$ with the meaning
that $\int_{z_2}^{z_1} N(\tilde{z},t)d\tilde{z}$ is the total number of individuals in a population
between the p-ages of z_1 and z_2 at time t. $N(z,t)$ is thus a dynamic,
p-age number distribution.

Its equation of motion obeys the conservation law:

$$\frac{\partial N(z,t)}{\partial t} + \frac{\partial}{\partial z}\left[g(z,t)N(z,t)\right] = -\mu(z,t)N(z,t). \qquad (2.2\text{-}1)$$

Here, $g(z,t)$ is defined by (2.1-1), and $\mu(z,t)$ is the instantaneous
loss rate of individuals from the population due to predation and
natural mortality.

Equation (2.2-1) is a "balance" equation. It says that the rate
of growth of individuals through p-age z, given by $\frac{\partial}{\partial z}\left[g(z,t)N(z,t)\right]$,
and the loss rate of individuals of p-age z, given by $[\mu(z,t)N(z,t)]$,
are balanced by the net rate of change of numbers at p-age z, expressed
by $\frac{\partial N(z,t)}{\partial t}$.

To describe the dynamics of a reproducing population, Equation
(2.2-1) must be accompanied by a reproductive feedback which will appear
as a boundary condition. Let the birth rate of individuals of p-age z

at time t be denoted by $B(z,t)$. Then, the birth rate can be defined
as the functional:

$$B(z,t) = \int_{z_b}^{z_b+\alpha} b(z,\tilde{z},t) \, N(\tilde{z},t) \, d\tilde{z}. \tag{2.2-2}$$

In this equation, $b(z,\tilde{z},t)$ is the rate at which individuals of p-age $\tilde{z}$
give birth to neonates of p-age z. z_b is the p-age at which individuals
first reproduce, and α is the width of the breeding interval.

Equation (2.2-2) is the most general form for a reproductive
feedback, but when it is coupled with (2.2-1) it appears as a distributed
boundary condition, and the resulting system equations yield little to
analysis. For most biological populations, with the notable exception
of cell populations, a restricted form of (2.2-2) will entail only a
minor loss of realism. We will hereafter assume that all neonates have
the same p-age, denoted by z_0. Then (2.2-2) becomes

$$B(t) \stackrel{\Delta}{=} \int_{z_b}^{z_b+\alpha} b(\tilde{z},t) \, N(\tilde{z},t) \, d\tilde{z}. \tag{2.2-3}$$

Now $B(t)$ is defined as the total instantaneous birth rate, and $b(z,t)$ is
the p-age-specific individual fecundity.

The connection between the reproductive functional, $B(t)$, and a
boundary condition for (2.2-1) is found by integrating (2.2-1) over all
p-ages to get

$$\frac{\partial}{\partial t}\left[\int_{z_0}^{\infty} N(\tilde{z},t)\,d\tilde{z}\right] + \left[g(\infty,t)N(\infty,t) - g(z_0,t)N(z_0,t)\right] =$$

$$- \int_{z_0}^{\infty} \mu(\tilde{z},t)N(\tilde{z},t)\,d\tilde{z}. \tag{2.2-4}$$

Define $N_t(t) \overset{\Delta}{=} \int_{z_0}^{\infty} N(\tilde{z},t)d\tilde{z}$ as the total number in the population. Also, assume that no organisms survive past some upper limit in p-age so that $g(\infty,t) \, N(\infty,t) = 0$. Then (2.2-4) becomes

$$\frac{dN_t(t)}{dt} = g(z_0,t)N(z_0,t) - \int_{z_0}^{\infty} \mu(\tilde{z},t)N(\tilde{z},t)d\tilde{z}. \qquad (2.2-5)$$

Since $\frac{dN_t}{dt} = B(t) - D(t)$, where $B(t)$ and $D(t)$ are population birth and death rates, respectively, we must have

$$g(z_0,t) \, N(z_0,t) = B(t). \qquad (2.2-6)$$

Using (2.2-3) the boundary condition is also written as

$$g(z_0,t) \, N(z_0,t) = \int_{z_b}^{z_b+\alpha} b(\tilde{z},t) \, N(\tilde{z},t)d\tilde{z}. \qquad (2.2-7)$$

With reasonable restrictions on $g(z,t)$, to be discussed shortly, and an initial condition, $N(z,0) = N_I(z)$, the system (2.2-1), (2.2-7) prescribes a unique solution surface above the (p-age)x(time) plane for the number density $N(z,t)$.

Figure 2.1 is a sketch of a p-age distribution, $N(z,t_1)$, at an instant in time, t_1, showing the relationships implied by (2.2-1) and (2.2-7). Similar sketches are drawn by Oster and Takahashi [1974]. Derivations of various forms of the balance Equation (2.2-1) in the context of populations can be found in several papers [Von Foerster, 1957; Sinko and Streifer, 1967; Weiss, 1968; Levin and Paine, 1975; Lee, et al., 1975].

For the sake of tractability, an important restriction must be made on the growth rate in (2.2-1). It is necessary that $g(z,t)$ be
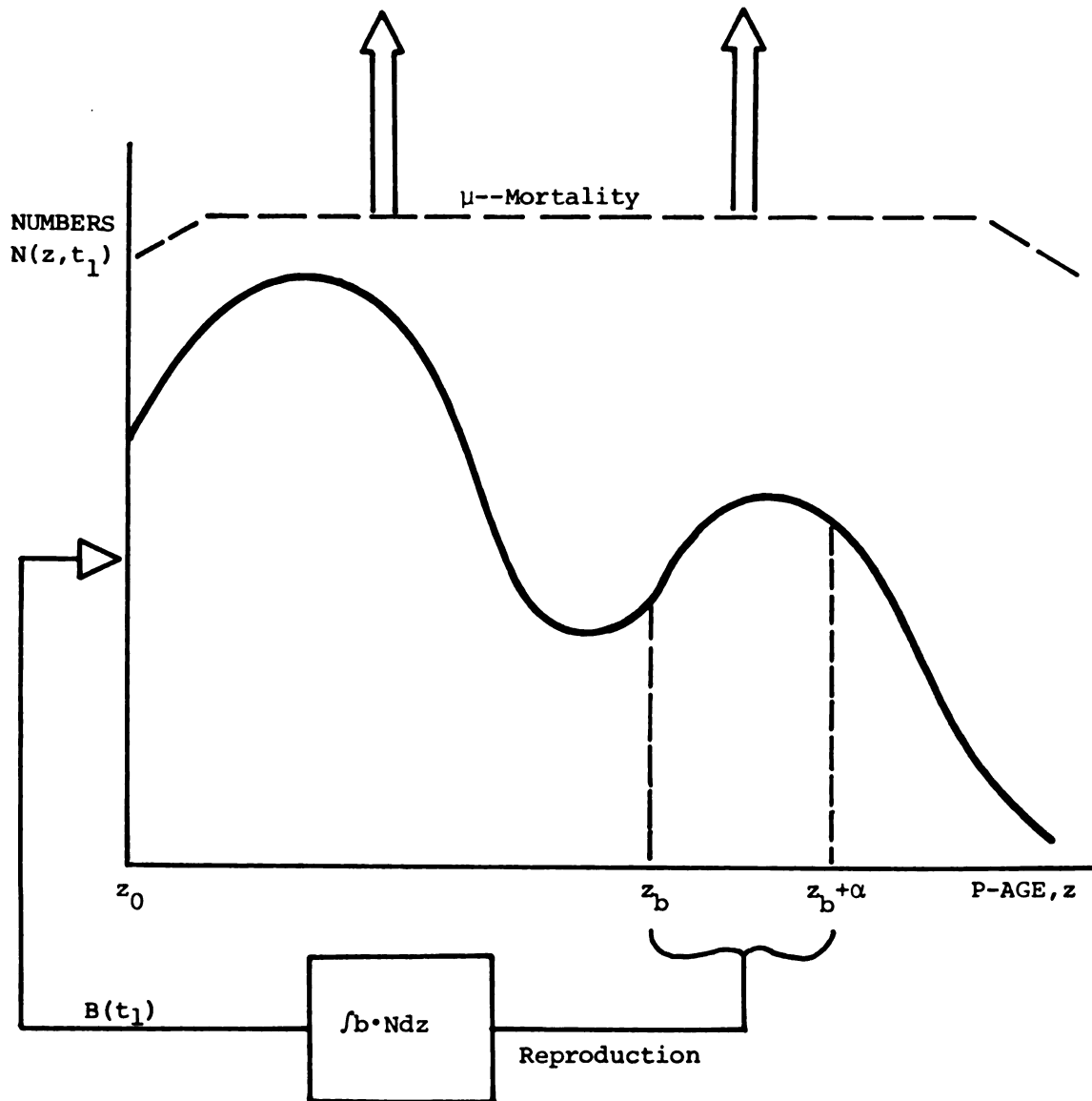
Figure 2.1. Physiological age (p-age) profile at a point in time, t_1, showing reproductive feedback and mortality loss.

everywhere positive, so that an individual's p-age is continually

increasing. This restriction, together with the restricted description

of reproduction seen in (2.2-3), results in a one-to-one correspondence

between any individual's p-age and its chronological age. This cor-

respondence is necessary for the validity of the analytical solutions

to (2.2-1) which are discussed later in this chapter. The restriction

that individual growth must be positive results in a less realistic

model, especially if z represents body weight. Fortunately, the

restriction is necessary only for analytical solutions. Most detailed

applications of the model for predicting population trajectories would

require simulation, in which case negative growth could be easily

handled.

The vital statistics g, b, and μ are in general dependent upon

both the internal p-age structure of the population and external environ-

mental factors. The effects of intraspecific competition can be expressed

by allowing g, b, and μ to vary with the state function, $N(z,t)$. The

inclusion of density-dependence in (2.2-1) and (2.2-7) is discussed

in Chapter 5.

It is also possible, in special cases, to factor out the explicit

time dependence of g, b, and μ. For example, developmental rates in

insects and other poikilotherms are closely tied to temperature, and

this has prompted entomologists to discuss growth relative to a physio-

logical time scale based on degree days or "accumulated heat units."

Hughes [1962] used this concept to model the instar or life-stage

structure of an aphid population. Lee, et al. [1975] model the cereal

leaf beetle and its parasites using an equation of the same form as

(2.2-1). They base their growth, mortality, and reproductive parameters on a physiological time scale, so that, for example, the equation for growth, (2.1-1), appears in their model as $\frac{dz}{d\hat{t}} = g(z)$, where $\hat{t}$ represents degree-days.

2.3 Relations to Other Models

The balance model is easily generalized to handle characterization of individuals by more than one feature. In this case, z becomes a vector, $\bar{z}$, with

$$\bar{z} = \left[z_1, \text{age}; \ z_2, \text{length}; \ z_3, \text{weight}; \ldots z_m \right].$$

Equation (2.2-1) is expanded to the form

$$\frac{\partial N(\bar{z},t)}{\partial t} + \sum_{i=1}^{m} \frac{\partial}{\partial z_i} \left[g_i(\bar{z},t) N(\bar{z},t) \right] = -\mu(\bar{z},t) N(\bar{z},t). \quad (2.3-1)$$

Here, $g_i(\bar{z},t)$ is the growth rate of characteristic $\bar{z}_i$ [Oster and Takahashi, 1974].

Sinko and Streifer [1967; 1969; 1971] discuss the model (2.3-1) with z_1 = chronological age and z_2 = body mass, and apply it to populations of Daphnia and the planarian worm Dugesia tigrina. In a model of shrimp in an estuary, Billups, Wilson, and Pike [1971] apply (2.3-1) to shrimp classified by body length and three spatial coordinates, resulting in a four-dimensional state vector, N.

An interesting application of (2.3-1) to a quite different ecological system is proposed by Levin and Paine [1974; 1975]. They use the density function $N(\bar{z},t)$ to represent component islands, or "patches"

within a heterogeneous natural community. Each patch is assumed to be

homogeneous with respect to biological properties, e.g., species com-

position, and the growth rate of patches is a function of intra- and

inter-patch interactions and localized environmental disturbances.

In this fashion, Levin and Paine hope to examine the long-ignored role

of spatial heterogeneity in structuring communities.

If, in (2.1-1), we let p-age, z, be equivalent to chronological

age, a, then $\frac{da}{dt} = 1 = g$. Hereafter, the abbreviation "c-age" will be

used for chronological age, a. Now (2.2-1) reduces to a simpler

equation:

$$\frac{\partial N(a,t)}{\partial t} + \frac{\partial N(a,t)}{\partial a} = -\mu(a,t)N(a,t). \qquad (2.3-2)$$

This form of (2.2-1) is known as von Foerster's equation, after H.

von Foerster [1959] who used it to model cell populations. Since von

Foerster's work, balance models have been studied extensively in the

context of cell population dynamics. Trucco [1965] and Nooney [1967]

investigate (2.2-1) in some detail. Weiss [1968] and Bell and Anderson

[1967] present more generalized models of the form (2.3-1) for cell

populations. Bell and Anderson, for example, classify cells according

to the c-age and volume. For mitotic cell populations with individuals

classified by size, distributed boundary conditions must be used with

balance models since daughter cells at birth are assumed to be one-half

the size of their progenitors. This fact, together with the common

assumption by cell modelers that cells experience no natural mortality

before division, results in partial differential equation models whose

dynamics are quite different from those of the system (2.2-1), (2.2-7).

An excellent application of von Foerster's equation to host-parasite systems has been presented by Oster and his coworkers [Oster and Takahashi, 1974; Auslander, Oster and Huffaker, 1974]. A variety of system dynamics are discussed in relation to such parametric properties as synchronous maturation delays in host and parasite populations and periodic time functions for mortality and fecundity.

A more complete review of applications of distributed parameter models to various populations is found in Streifer [1974]. Because of the complexity of the generalized model (2.3-1), a detailed analysis of its dynamics will not be attempted here.

If the c-age/time continuum is discretized, then the well-known matrix methods developed by Lewis [1942] and Leslie [1945] are applicable. Sinko [1969] shows the equivalence, in the limit as $\Delta t \rightarrow 0$, of the matrix methods and the von Foerster equation.

Discrete models based on p-age are a more recent development. Lefkovitch [1965] subdivided an insect population into life stages and defined a vector $\bar{n}(t)$ whose component $\bar{n}_i(t)$ gives the number of individuals in the ith stage at time t. He then described a projection matrix M; the (i,j) element of M is the proportional contribution of the numbers in the ith stage at time t to the numbers in the jth stage at time (t + Δt). Thus, $\bar{n}(t + \Delta t) = M \cdot \bar{n}(t)$. Lefkovitch recognized that the numbers in the matrix M represented the effects on $\bar{n}$ of mortality during the time interval [t, t + Δt], confounded with the effects of growth during the same period. Because of this complication, he was unable to give a clear biological interpretation of the matrix entries.

A more complex discrete p-age model was developed by Coulman,

Reice and Tummala [1972]. In this model, stage-specific growth and mortality rates are considered separately, as is done continuously in (2.2-1). The equivalence in the limit as $\Delta t \rightarrow 0$ is shown in Kharkar, Coulman and Barr [1974].

A stochastic version of (2.2-1) permits individual differences in growth rate due to genetic variability or to microenvironmental variations arising from the spatial distribution of organisms. A term is added to (2.2-1) to reflect the dispersion in p-age of individuals, so that the model takes the form

$$\frac{\partial N(z,t)}{\partial t} + \frac{\partial [g(z,t)N(z,t)]}{\partial z} - \frac{1}{2}\frac{\partial^2}{\partial z^2}[v(z,t)N(z,t)] =$$

$$-\mu(z,t)N(z,t). \tag{2.3-3}$$

In (2.3-3) the individual growth rate is a random variable with mean $g(z,t)$ and variance $v(z,t)$. Higher moments of the probability distribution associated with growth are assumed negligible in their effects on population dynamics. The diffusion equation (2.3-3) and its application to populations is discussed by Bailey [1964]. A derivation can be found in Kharkar [1973] or Lee, et al. [1975].

2.4 Solutions for Time-Invariant Parameters

As a first step, assume that (2.2-1) and (2.2-7) describe a population whose growth, mortality and fecundity rates are independent of time. The system (2.2-1), (2.2-7) is then autonomous and, for specific functions g, μ, and b, its solution depends only on an initial p-age distribution, $N_I(z)$.

Equation (2.2-1), upon application of the product rule for

differentiation, becomes

$$\frac{\partial N(z,t)}{\partial t} + g(z) \frac{\partial N(z,t)}{\partial z} = - [g'(z) + \mu(z)] N(z,t), \quad (2.4-1)$$

where:

$$g'(z) \triangleq \frac{dg(z)}{dz} .$$

By the method of characteristics [Courant and Hilbert, 1966], (2.4-1) is equivalent to the set of differential equations

$$dt = \frac{dz}{g(z)} = \frac{dN(z,t) \cdot}{-[g'(z)+\mu(z)]} . \quad (2.4-2)$$

To facilitate writing solutions to (2.4-2), define

$$\tau(z_2,z_1) \triangleq \int_{z_1}^{z_2} \frac{d\tilde{z}}{g(\tilde{z})} . \quad (2.4-3)$$

Then $\tau(z_2,z_1)$ is the time required to grow from p-age z_1 to p-age z_2. Since we assumed that all neonates are of p-age z_0, the quantity $\tau(z,z_0)$ is simply the chronological age, a, of individuals of p-age z.

It was required that $g(z) > 0$ for all values of z, so $\tau(z_2,z_1)$ is a monotonically decreasing function in z_1 for any fixed z_2, and it thus has an inverse function in z_1, defined as

$$\hat{z}(z_2,\tau) \triangleq \tau^{-1}(z_2,z_1). \quad (2.4-4)$$

This definition means that $\hat{z}(z_2,\tau)$ is the p-age at which an organism must be at time $(t - \tau)$ in order to grow to p-age z_2 at time t.

Now, using the functions $\tau,\hat{z}$, and the initial condition $N(z,0) = N_I(z)$, we have the solution to (2.4-2):

$$N(z,t) = N_I(\hat{z}(z,t)) \exp\left[-\int_{\hat{z}(z,t)}^z \left(\frac{g'(\tilde{z})+\mu(\tilde{z})}{g(\tilde{z})}\right) d\tilde{z}\right] \quad (2.4\text{-}4a)$$

for all p-ages, z, such that $\tau(z,z_0) \geq t$, and

$$N(z,t) = N(z_0,t-\tau(z,z_0)) \exp\left[-\int_{z_0}^z \left(\frac{g'(\tilde{z})+\mu(\tilde{z})}{g(z)}\right) d\tilde{z}\right] (2.4\text{-}4b)$$

for all p-ages, z, such that $\tau(z,z_0) < t$.

The solution thus has two parts. The first part, (2.4-4a), describes that portion of the p-age distribution, $N(z,t)$, whose members are survivors of the initial population, $N_I(z)$. The second equation, (2.4-4b), characterizes the portion of the distribution whose members were born after time $t = 0$, and entered the distribution via the boundary, $N(z_0,\cdot)$.

The exponential terms in Equations (2.4-4) can be simplified as follows:

$$\exp\left[-\int_{z_1}^{z_2} \frac{g'(\tilde{z})}{g(\tilde{z})} d\tilde{z} - \int_{z_1}^{z_2} \frac{\mu(\tilde{z})}{g(\tilde{z})} d\tilde{z}\right] =$$

$$\frac{g(z_1)}{g(z_2)} \exp\left[-\int_{z_1}^{z_2} \frac{\mu(\tilde{z})}{g(\tilde{z})} d\tilde{z}\right]. \quad (2.4\text{-}5)$$

Now, the exponential factor on the right-hand side of (2.4-5) will be redefined as the p-age-specific survivorship. That is,

$$\exp\left[-\int_{z_1}^{z_2} \frac{\mu(\tilde{z})}{g(\tilde{z})} d\tilde{z}\right] \overset{\Delta}{=} \ell(z_2,z_1). \quad (2.4\text{-}6)$$

where $\ell(z_2,z_1)$ is the proportion of organisms alive at p-age z_1 which survive to reach p-age z_2 after being exposed to the mortality rate $\mu(z)$. Using this simpler notation, the solution (2.4-4) becomes

$$N(z,t) = N_I(\hat{z}(z,t)) \frac{g(\hat{z})}{g(z)} \ell(z,\hat{z}),$$ (2.4-7a)

for $\tau(z,z_0) \geq t$, and

$$N(z,t) = N(z_0, t - \tau(z,z_0)) \frac{g(z_0)}{g(z)} \ell(z,z_0)$$ (2.4-7b)

for $\tau(z,z_0) < t$.

To illustrate the solution equations, take the simple case of constant growth rate, i.e., $g(z) = \hat{g}$. Then

$$\tau(z_2,z_1) = \int_{z_1}^{z_2} \frac{1}{\hat{g}} d\tilde{z} = \frac{z_2 - z_1}{\hat{g}},$$

and $\hat{z}(z,t) = z - \hat{g}t$, since $\hat{g}t$ is the increment in p-age during the time interval $(0,t)$.

The solution (2.4-7) then becomes

$$N(z,t) = N_I(z - \hat{g}t) \; \ell(z, z - \hat{g}t),$$ (2.4-8a)

for $\dfrac{z - z_0}{g} \geq t$, and

$$N(z,t) = N(z_0, t - \left[\frac{z-z_0}{g}\right]) \; \ell(z,z_0),$$ (2.4-8b)

for $\dfrac{z - z_0}{g} < t$.

If $z_0 = 0$, this case of constant growth rate is equivalent to a c-age model which uses time units of $\hat{g}t$ instead of t, and the Equations (2.4-8) become identical to those given for the von Foerster equations by Trucco [1965]. If $\mu = 0$, the Equations (2.4-8) are identical to the results in Lee, et al. [1975].

2.5 Interpretation of Solutions

To better understand the nature of the solutions (2.4-4) we will sketch the characteristic curves, defined by (2.4-2), for two cases. Figure 2.2, reproduced from Skellam [1967], shows a typical family of trajectories for the von Foerster equation where $z = a = c$-age. In this case, the solution is given by (2.4-8) with $\hat{g} = 1$ and $z_0 = 0$. The Equation (2.4-2) reduces to

$$dt = da = \frac{dN}{-\mu(a)} \qquad (2.5-1)$$

In Figure 2.2, the family of oblique lines in the age-time plane of slope $\frac{da}{dt} = 1$ are life-history trajectories, and they are also the characteristic curves of the partial differential Equation (2.3-2). This family of 45° lines in the age-time plane is known to demographers as a Lexis diagram [Keyfitz, 1968]. Along a life history trajectory, the number density N decreases in accordance with the equation $\frac{dN}{-\mu(a)} = da$. A pulse-wave type initial condition is shown along the (t = 0)-age axis, and a sinusoidal birth rate, B(t), provides the boundary condition along the (a = 0)-time axis.

Let us contrast the curves in Figure 2.2 with a common, but more complex, function for g. Suppose the p-age variable z represents body weight. Growth in body weight is described, for many organisms, by a sigmoidal or logistic weight-for-age function, where body weight z tends toward an upper limit, z_∞, as $t \to \infty$ [Thompson, 1942]. Figure 2.3a shows a family of sigmoidal growth curves. The growth function $g(z) = \frac{dz}{dt}$ gives the slope of any curve at a particular body weight, z. It can be seen that g(a) must have the shape shown in Figure 2.3b. It is generally
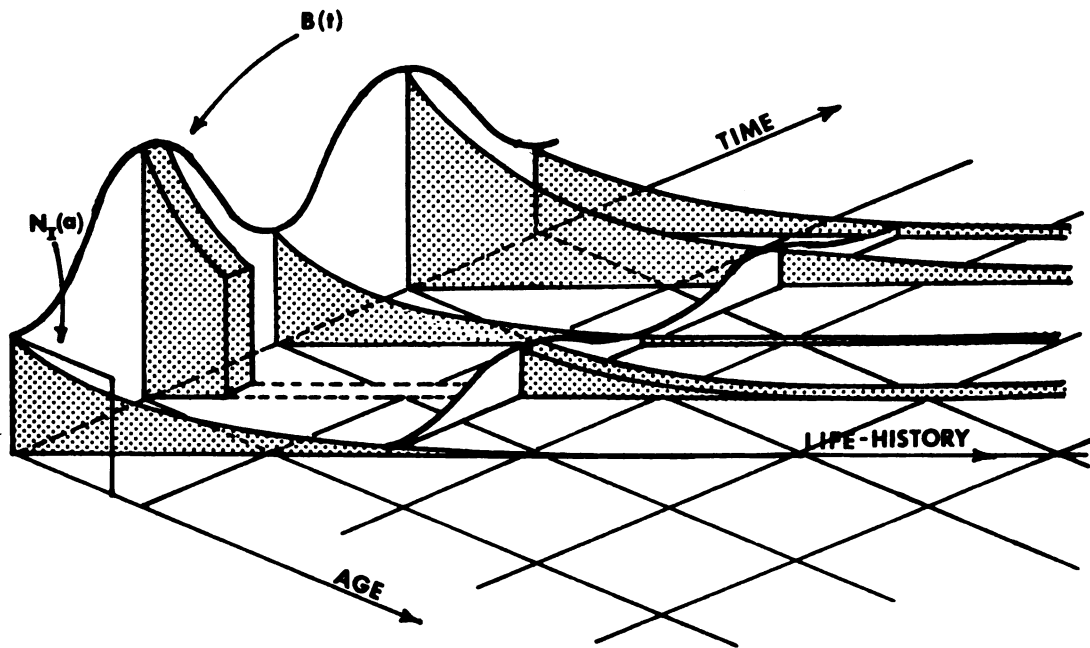
Figure 2.2. Trajectories for the von Foerster equation. From Skellam [1967].
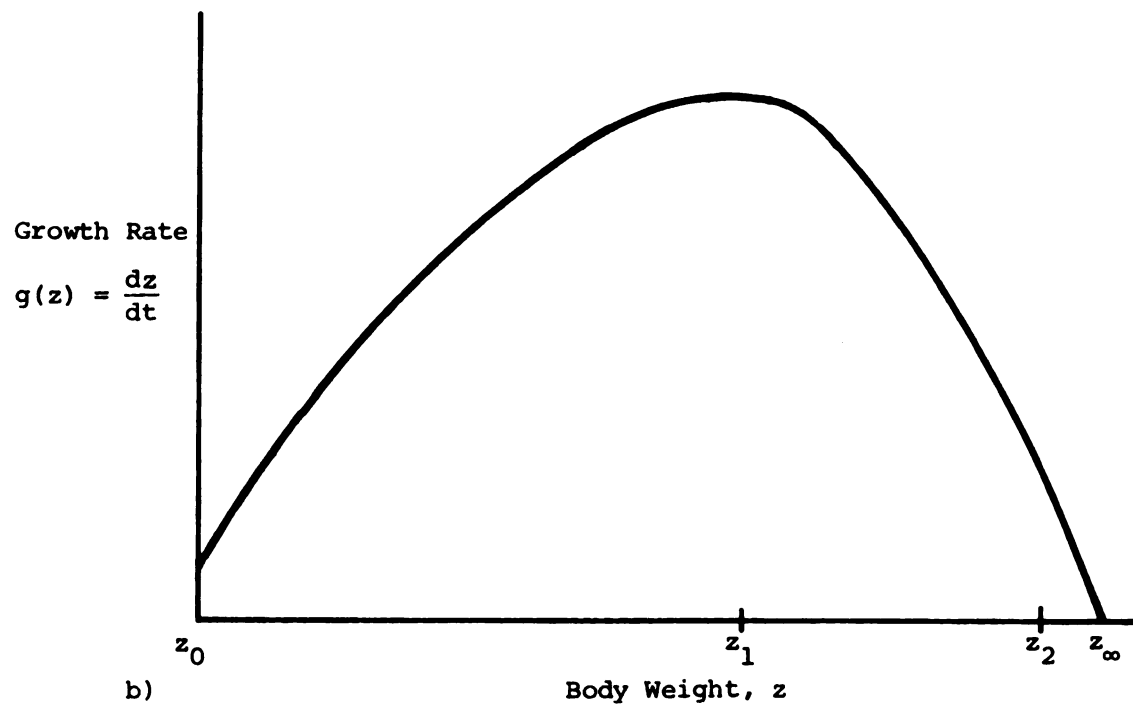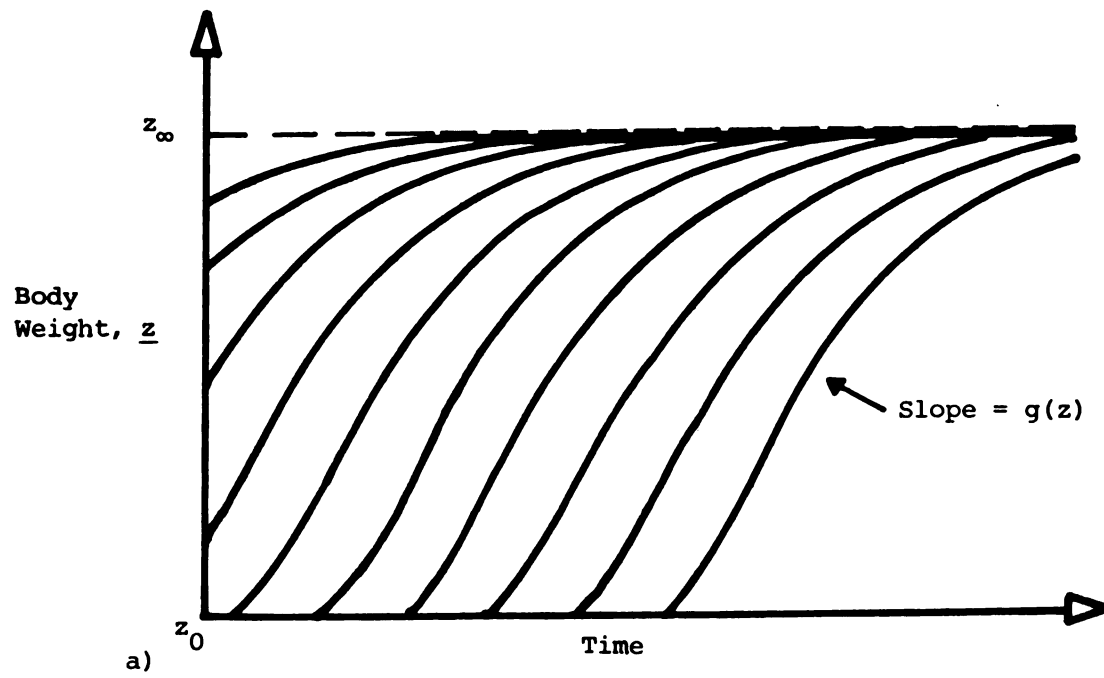
Figure 2.3. a) Characteristic curves for sigmoidal growth.
b) Growth rate curve for sigmoidal growth.

a concave function of z, first increasing, then decreasing to zero

as $z \to z_\infty$.

The family of curves in Figure 2.3a is analogous to the set of

oblique straight lines in the age-time plane of Figure 2.2. The curves

in Figure 2.3a are the characteristic curves defined by Equation (2.4-2)

employing a concave growth function $g(z)$.

In order to solve (2.4-1) when it contains a growth function $g(z)$

which approaches zero for a finite value z_∞, it is necessary that

$[g'(z) + \mu(z)] > 0$ as $z \to z_\infty$ so that the exponential terms in the solu-

tion (2.4-4) do not become unbounded as $z \to z_\infty$.

Figure 2.2 illustrates the dynamics of $N(z,t)$ for the simple case

$g = 1$ and $z = a$. What dynamics might be seen for $N(z,t)$ if a concave

function like that in Figure 2.3b is used for $g(z)$? Let us examine the

progress of a simple idealized population density, $N(z,t)$, through

a p-age distribution. Assume that z represents body weight, $g(z)$ is

described by Figure 2.3b, and $\mu(z) = \hat{\mu}$ a constant.

Imagine starting with a single cohort of individuals in the

weight range z_0 to $(z_0 + s)$ at time t_0 (Figure 2.4a). The total number

of individuals in the population is equivalent to the area under the

rectangular number density curve. Assume that s is small enough so that

the number density of the cohort can be approximated by an average value

over the width of the cohort. Two cases for the fate of the cohort

will be described.

CASE 1 - High mortality, μ, over the full weight range (Figure 2.4a).

As individuals increase in weight, up to z_1, the larger individuals

in the cohort grow at a faster rate than the smaller ones. The result
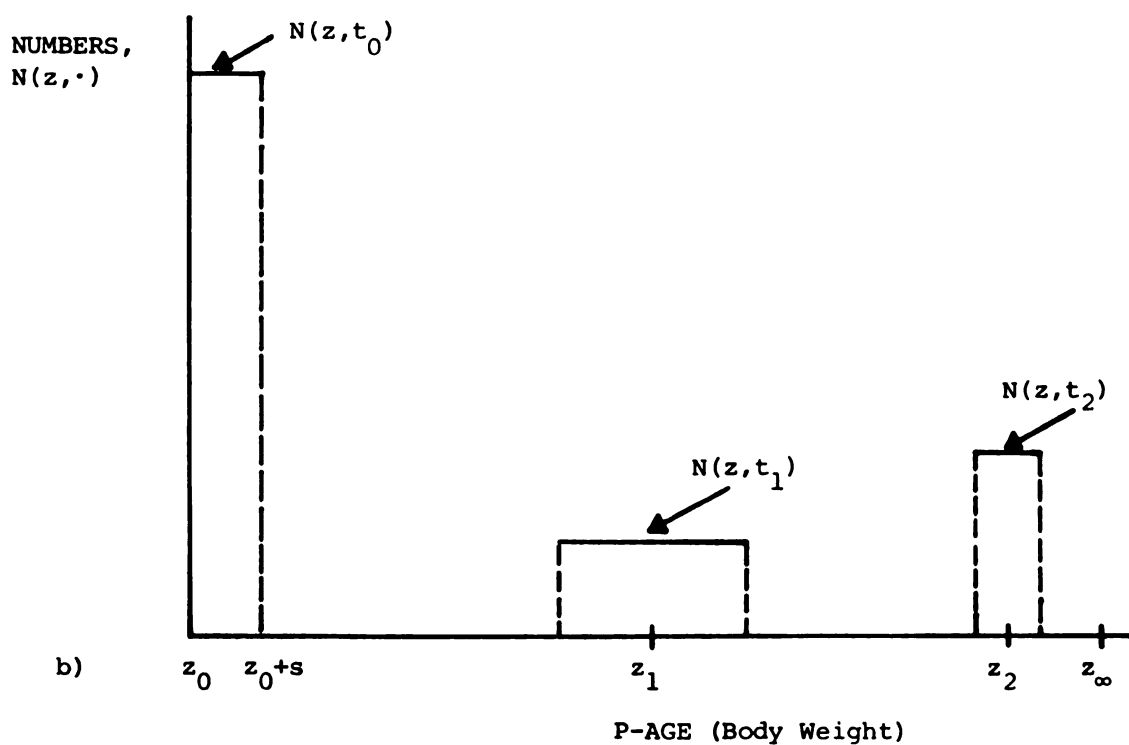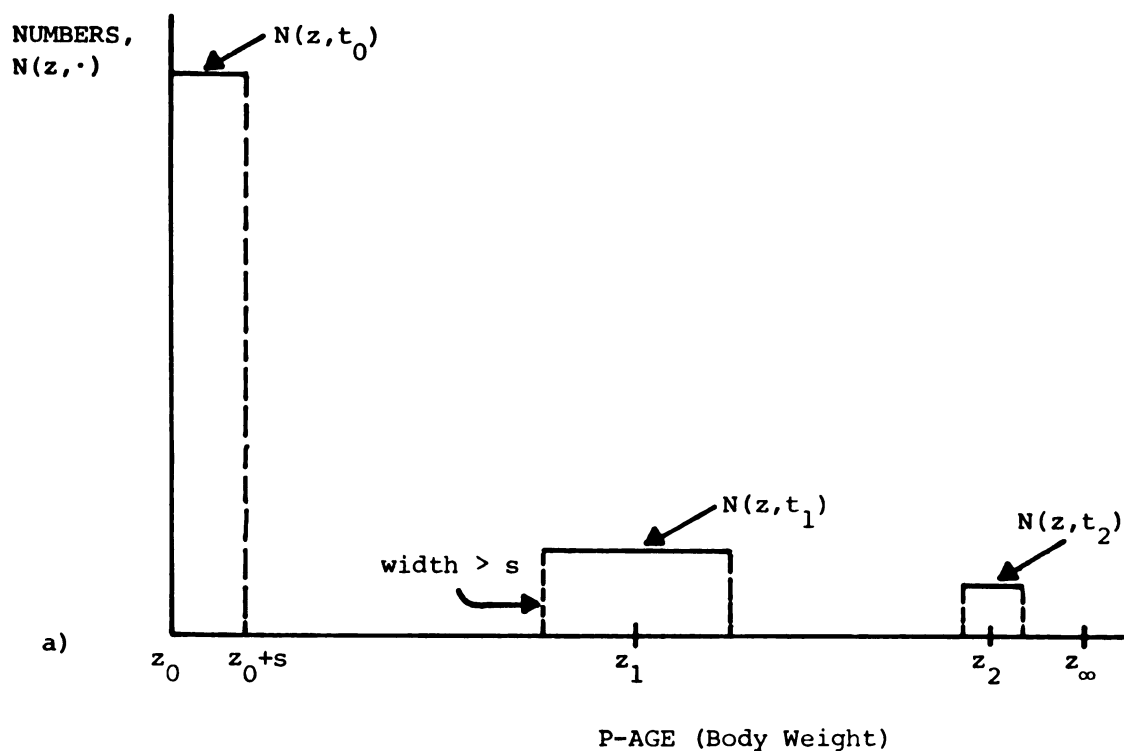
Figure 2.4. Trajectories of idealized cohort: a) Case 1 - Concave growth function and high mortality at all p-ages. b) Case 2 - Concave growth function and high mortality up to z_1, low mortality beyond z_1.

is that the cohort spreads out, or "expands", to encompass a broader weight range. At a later time, t_1, when the cohort has reached a mean weight for individuals of z_1, the weight range of the cohort is broader than s, its initial width. See $N(z,t_1)$ in Figure 2.4a. The total area under the number density curve however, has been reduced by a factor of $e^{-\mu(t_1-t_0)}$ due to mortality losses.

Once individuals reach the mean weight z_1, the expansion process in the cohort changes to a compression process. From z_1 onward, the growth rate is continually decreasing (Figure 2.3b), and larger organisms in the cohort are growing more slowly than smaller ones. The result is that the cohort becomes compressed into a smaller weight range as smaller individuals catch up to larger ones in size. At a time $t_2 > t_1$ and a mean weight of z_2 near z_∞, the cohort looks like $N(z,t_2)$ in Figure 2.4a. Its width has been reduced from the width seen at time t_1, and the area under the number density is again decreased, this time by a factor of $e^{-\mu(t_2-t_1)}$.

From this qualitative picture, it is clear that the magnitude, N, of a number density at any point (z,t) is the result of the force of mortality combined with "compression" or "expansion" of the distribution in individual weight differences.

CASE 2 - High mortality up to weight Z_1, low mortality for larger individuals (Figure 2.4b).

Suppose now that the dynamics of the cohort are identical to those of Case 1, up to mean weight z_1. After this point the growth rate again decreases, as in Figure 2.3b, but this time assume individuals are subject to little or no mortality in the weight range (z_1,z_2).

If this is the case, individuals growing beyond z_1 will begin to "stack up", i.e., the cohort will be compressed into an increasingly narrow weight range, as before, but very few individuals will be lost due to mortality. The result is that, at time t_2 and mean weight z_2, the cohort will look like $N(z,t_2)$ in Figure 2.4b. The area under the number density curve at t_2 has decreased only slightly from the area at t_1, due to the small mortality loss during the interval $[t_1, t_2]$, but the cohort is now squeezed into a much narrower weight range that the range seen at t_1. Thus, the number density at time t_2 has a greater magnitude than it did at t_1.

These cohort growth dynamics provide a mechanistic basis for describing the phenomenon of "stunting" in populations, which will be discussed again in Section 5.2.

The quantitative tradeoff between mortality and growth rates in changing the magnitude of the number density can be determined by examining the solution (2.4-4b). Suppose in some small time increment Δt, an organism of p-age z grows to p-age $(z + \delta z)$.

Then, according to (2.4-4b),

$$N(z+\delta z, t+\Delta t) = N(z,t)\exp\left[\int_z^{z+\delta z} -\left(\frac{g'(\tilde{z})+\mu(\tilde{z})}{g(\tilde{z})}\right)d\tilde{z}\right]. \quad (2.5-2)$$

It is clear that the relative magnitudes of $N(z + \delta z, t + \Delta t)$ and $N(z,t)$ depend on the sign of the integral exponent in (2.5-2) which in turn depends on the sign of the integrand, $-\left(\frac{g'(\tilde{z})+\mu(\tilde{z})}{g(\tilde{z})}\right)$, over the range $[z, z + \delta z]$. Since $g(\tilde{z}) > 0$ over this interval, we can focus on the numerator of this fraction, $- [g'(\tilde{z}) + \mu(\tilde{z})]$.

In both Case 1 and Case 2, up until p-age z_1, the growth rate was increasing, so over each small p-age interval, $[z, z + \delta z]$, the inequality $-[g'(\tilde{z}) + \mu(\tilde{z})] < 0$ was true. Thus, $N(z + \delta z, t + \Delta t) < N(z,t)$. The number density of the cohort decreased in magnitude over each small increment $(\delta z, \Delta t)$.

When p-age/time increments for p-ages greater than z_1 are considered, a difference appears between Cases 1 and 2. In both cases, the growth rate is decreasing so that $g'(\tilde{z}) < 0$. Case 1, with a high mortality rate, evidentally has $|g'(\tilde{z})| < \mu(\tilde{z})$ so that $-[g'(\tilde{z}) + \mu(\tilde{z})] < 0$. Hence, each small p-age/time increment results in a <u>decrease</u> in the number density, according to (2.5-2). The sum of these decreases is seen in the magnitude difference between $N(z,t_1)$ and $N(z,t_2)$ in Figure 2.4a.

On the other hand, Case 2, with very low mortality for $z_1 < \tilde{z} < z_2$, must have $|g'(\tilde{z})| > \mu$ for $z_1 < \tilde{z} < z_2$. In this case, $-[g'(\tilde{z}) + \mu(\tilde{z})] > 0$, and each p-age/time increment <u>increases</u> the number density. The sum of these increases is seen in the difference in height between the number density $N(z,t_1)$ and the number density $N(z,t_2)$ in Figure 2.4b.

In summary, as a cohort grows through the p-age profile, the magnitude of the number density, N, will rise or fall in accordance with the sign of the quantity, $-[g'(\tilde{z}) + \mu(\tilde{z})]$.

From this analysis we begin to see the relationship between an observed p-age distribution of a population and the growth-mortality interaction which gives rise to the p-age profile. Chapter 3 will examine this relationship for a steady-state population in which individuals of every p-age are represented.

CHAPTER 3

ASYMPTOTIC PROPERTIES--THE STABLE P-AGE DISTRIBUTION

3.1 **Asymptotic Properties of C-Age Models**

What is the fate of the p-age distribution and the total population

numbers as t gets "large", that is, on the order of very many generations?

This question can be answered for the system (2.2-1), (2.2-7) restricted

to time-invariant fecundity, mortality and growth rates. To provide

the answers, essentially the same methods employed by A. J. Lotka in

his pioneering work on the dynamics of c-age distributions will be used.

Lotka's well-known results will be compared with the p-age model's

behavior as $t \to \infty$.

Let us briefly review Lotka's three central conclusions about the

limiting (in time) dynamics of populations whose members have time-

invariant, c-age-specific fecundity and mortality rates.

A. As t gets large, the population c-age structure approaches a

fixed c-age distribution. That is, if da is a small c-age increment,

then the ratio $\dfrac{N(z,t)da}{N_t(t)} \to c(a)da$, where $c(a)$ is a function depending

only on individual mortality and fecundity rates [Sharpe and Lotka, 1911].

The quantity $c(a)da$ is the proportion of individuals in the population

aged a to (a+da).

B. For large t, the total population birth rate, B(t), defined

by Equation (2.2-3), grows or decays exponentially in time, as does the

total number, $N_t(t)$. Both B(t) and $N_t(t)$ grow or decay at a constant

rate, r, called the "intrinsic rate of natural increase" [Lotka, 1925].

C. If the stable c-age distribution of an exponentially-growing
population is perturbed, it will eventually return to a stable config-
uration and the population will again grow exponentially. Lotka showed
this using an ingenious graphical method [Lotka, 1922], but the stability
of the c-age distribution is more easily seen by considering a pertur-
bation at some time $\hat{t}$ as defining a new initial condition, $N(a,\hat{t})$, for
the c-age distribution. Then, as the population and its c-age structure
develop from time $\hat{t}$ onward, according to results A and B, a stable c-age
distribution and exponential growth will again emerge for $t \gg \hat{t}$.

Lotka's methods and the results A - C form the basis for modern
population theory in the age-time continuum, and his work has been
refined and expanded by many researchers. The derivation of these
results can be found in a number of books and papers, e.g., Keyfitz
[1968], Langhaar [1972], Lotka [1925], and will not be repeated here.

In this chapter, results A - C will be derived for populations
characterized by physiological age. The methods used are similar to
Lotka's and the usual terminology and notational conventions of con-
tinuous c-age theory will be used wherever possible.

In anticipation of the stability results, consider the relationship
between p-age and c-age which was discussed in Section 2.2. There it
was pointed out that there is a one-to-one correspondence in the
restricted p-age model between an individual's p-age and its c-age.
Thus, as a population acquires a stable c-age distribution, one would
expect it to also show a stable p-age distribution.

The advantage of pursuing a proof of A - C for p-age which is
similar to Lotka's proof for c-age is twofold. First, the method helps

to illustrate how the p-age model generalizes the classical c-age theory. Secondly, the exact shape of the stable p-age distribution will emerge as a function of growth, mortality, and fecundity rates.

3.2 A Renewal Equation for B(t), the Birth Rate

Define $G(t)$ as the instantaneous birth rate due to survivors of the initial population, $N_I(z)$. As t increases, $G(t) \to 0$, since all members of the initial population eventually die or grow past the p-age breeding interval, $[z_b, z_b + \alpha]$. The birth rate now obeys the following integral equation:

$$B(t) = G(t) + \int_{z_b}^{z_b + \alpha} b(\tilde{z}) N(\tilde{z}, t) d\tilde{z}. \qquad (3.2-1)$$

Since $G(t)$ accounts for births from members of the initial population, the density function $N(\tilde{z}, t)$ in the integrand must refer to those individuals in the population born after time $t = 0$. $N(\tilde{z}, t)$ in (3.2-1) is therefore given by the solution (2.4-5b), and substitution into (3.2-1) yields

$$B(t) = G(t) + \int_{z_b}^{z_b + \alpha} b(\tilde{z}) \ N(z_0, t - \tau(\tilde{z}, z_0)) \ \frac{g(z_0)}{g(\tilde{z})} \ \ell(\tilde{z}, z_0) d\tilde{z}. \qquad (3.2-2)$$

Using (2.2-6), this becomes

$$B(t) = G(t) + \int_{z_b}^{z_b + \alpha} b(\tilde{z}) \ B(t - \tau(\tilde{z}, z_0)) \ \frac{\ell(\tilde{z}, z_0)}{g(\tilde{z})} \ d\tilde{z}. \qquad (3.2-3)$$

This renewal equation is the equivalent, for p-age, of the well-known Lotka equation for c-age. In fact, (3.2-3) can be transformed to the Lotka equation by a simple change of variables in the integral term to permit integration over the time domain. Since $\frac{dz}{g(z)} = dt$, it follows that

$$B(t) = G(t) + \int_{\tau(z_b,z_0)}^{\tau(z_b+\alpha,z_0)} b(z(\tilde{t}))\, B(t-\tilde{t})\, \ell(z(\tilde{t}),z_0)\, d\tilde{t}. \qquad (3.2\text{-}4)$$

Also, since $b(z(t)) \equiv 0$ for $t > \tau(z_b + \alpha, z_0)$ and $t < \tau(z_b,z_0)$, equation (3.2-4) can be written as

$$B(t) = G(t) + \int_0^t B(t-\tilde{t}) b(z(\tilde{t}))\, \ell(z(\tilde{t}),z_0)\, d\tilde{t}. \qquad (3.2\text{-}5)$$

Equation (3.2-5) is the familiar Lotka renewal equation. Theorems for the existence and uniqueness of solutions to this equation are given in Keyfitz [1968] and Bellman and Cooke [1963]. The solutions to (3.2-5) are presented by Lotka [1925] and are also found in Keyfitz [1968] and Langhaar [1972].

The unique solution to (3.2-5) can be written as

$$B(t) = Q_1 e^{r_1 t} + \sum_{k=2}^{\infty} Q_k e^{r_k t}. \qquad (3.2\text{-}6)$$

The constants Q_k, $k = 1, 2, \ldots, \infty$, depend upon $G(t)$ [Keyfitz, 1968]. Keyfitz also shows that there is a unique, real root, r_1, for (3.2-6) and that the complex roots, r_k, $k = 2, \ldots, \infty$, all have moduli which are strictly less than $|r_1|$. Hence, $B(t) \simeq Q_1 e^{r_1 t}$ for large t--that is, the birth rate becomes a simple exponential, growing at the rate r_1. The root r_1 is equal to r, the intrinsic rate of increase, and r will replace r_1 in subsequent equations.

Since (3.2-5) and (3.2-3) are equivalent, the solution (3.2-6) with its asymptotic properties must also be the unique solution of (3.2-3). That is, the birth rate associated with the model (2.2-1) using time-invariant parameters becomes exponential as $t \to \infty$.

Let us assume that t is large enough so that $B(t) \simeq Q_1 e^{rt}$,

and G(t) = 0. Then by Equation (3.2-3),

$$B(t) = \int_{z_b}^{z_b+\alpha} b(\tilde{z}) \, B(t-\tau(\tilde{z},z_0)) \, \frac{\ell(\tilde{z},z_0)}{g(\tilde{z})} \, d\tilde{z}. \qquad (3.2-7)$$

That is,

$$1 = \int_{z_b}^{z_b+\alpha} e^{-r\tau(\tilde{z},z_0)} \, \frac{b(\tilde{z})\ell(\tilde{z},z_0)}{g(\tilde{z})} \, d\tilde{z}. \qquad (3.2-8)$$

Equation (3.2-8) is the characteristic equation for the root, r,

and it can be solved numerically by a variety of methods to give r.

[Keyfitz, 1968]. It was claimed above that r is the intrinsic rate of

increase for the total population numbers. To see this, use the solution

(2.4-7b), which is appropriate for large t:

$$N(z,t) = N(z_0,t-\tau(z,z_0)) \, \frac{g(z_0)}{g(z)} \, \ell(z,z_0). \qquad (3.2-9)$$

Stated in terms of a birth rate, this becomes

$$N(z,t) = B(t-\tau(z,z_0)) \, \frac{\ell(z,z_0)}{g(z)} \, . \qquad (3.2-10)$$

If $B(t)$ is exponential, then

$$N(z,t) = Q_1 e^{r[t-\tau(z,z_0)]} \left[\frac{\ell(z,z_0)}{g(z)} \right]. \qquad (3.2-11)$$

Now, the total numbers are given by $N_t(t)$:

$$N_t(t) = \int_{z_0}^{\infty} N(\tilde{z},t)d\tilde{z} = \int_{z_0}^{\infty} Q_1 e^{r[t-\tau(\tilde{z},z_0)]} \left[\frac{\ell(\tilde{z},z_0)}{g(z)} \, d\tilde{z} \right]. \qquad (3.2-12)$$

Differentiation of (3.2-12) confirms that

$$\frac{dN_t}{dt} = rN_t. \qquad (3.2-13)$$

Hence, both the birth rate, B(t), and the total numbers, $N_t(t)$ are

exponential in time for large t.

3.3 Relations in an Exponential Population

The exact shape of the stable p-age distribution can now be easily

derived. As before, let

$$c(z,t)dz = \frac{N(z,t)dz}{N_t(t)} \tag{3.3-1}$$

be the proportion of individuals in the population whose p-age is between

z and (z + dz) at time t.

Using (3.2-10) and the definition of $N_t(t)$, we have

$$c(z,t)dz = \frac{B(t-\tau(z,z_0)) \dfrac{\ell(z,z_0)}{g(z)} dz}{\displaystyle\int_{z_0}^{\infty} B(t-\tau(\tilde{z},z_0)) \dfrac{\ell(\tilde{z},z_0)}{g(\tilde{z})} d\tilde{z}} \, . \tag{3.3-2}$$

Assuming t is large, we use (3.2-11) to write this as

$$c(z)dz = \frac{e^{-r\tau(z,z_0)} \dfrac{\ell(z,z_0)}{g(z)} dz}{\displaystyle\int_{z_0}^{\infty} e^{-r\tau(\tilde{z},z_0)} \dfrac{\ell(\tilde{z},z_0)}{g(\tilde{z})} d\tilde{z}} \, . \tag{3.3-3}$$

In (3.3-3) c(z,t) has been replaced by c(z) since it becomes independent

of time. The function c(z) in (3.3-3) gives the shape of the stable

p-age distribution.

We will return to examine the shape function, c, in more detail,

but first let us generalize the definitions of some vital population

statistics which are widely used in c-age theory. Define the function

Ψ by

$$\Psi(r) \overset{\Delta}{=} \int_{z_b}^{z_b + \alpha} e^{-r\tau(\tilde{z}, z_0)} \left[\frac{b(\tilde{z}) \ell(\tilde{z}, z_0)}{g(\tilde{z})} \right] d\tilde{z}. \qquad (3.3\text{-}4)$$

Then $\Psi(r) = 1$ is the characteristic equation, (3.2-8). The quantity $\Psi(0)$ is known as the net reproductive rate, R_0, of the population [Keyfitz, 1968]. That is, $R_0 \overset{\Delta}{=} \Psi(0)$ is the average number of offspring which will be produced by an arbitrary newborn individual during its lifetime. To see this, note that $b(z)$ is an instantaneous rate, and so $\dfrac{b(z)dz}{g(z)} = b(z(t))dt$ is the number of offspring produced by an individual of p-age $z(t)$ during the time interval $[t, t+dt]$. Since $\ell(z, z_0)$ is the proportion of individuals which survive to p-age z, $\left[\dfrac{b(z) \ell(z, z_0)}{g(z)} dz \right]$ is the number of offspring expected from each newborn as it grows through the p-age interval $[z, z + dz]$. Therefore,

$$\Psi(0) = \int_{z_b}^{z_b + \alpha} \left[\frac{b(\tilde{z}) \ell(\tilde{z}, z_0)}{g(\tilde{z})} \right] d\tilde{z}$$

is the average offspring production during an individual's lifespan.

In a stable population, r, the intrinsic rate of increase, can be written

$$r = \bar{b} - d \qquad (3.3\text{-}5)$$

where $\bar{b}$ and d are the per capita birth and death rates, respectively. The birth rate $\bar{b}$ is also defined as

$$\bar{b}(t) = \frac{B(t)}{N_t(t)} \qquad (3.3\text{-}6)$$

That is,

$$\bar{b}(t) = \frac{B(t)}{\int_{z_0}^{\infty} B(t-\tau(\tilde{z},z_0)) \frac{\ell(\tilde{z},z_0)}{g(\tilde{z})} d\tilde{z}}$$

But $B(t) = Q_1 e^{rt}$, so $b(t)$ becomes a constant:

$$\bar{b} = \frac{1}{\int_{z_0}^{\infty} e^{-r\tau(\tilde{z},z_0)} \left[\frac{\ell(\tilde{z},z_0)}{g(\tilde{z})} \right] d\tilde{z}} \qquad (3.3-7)$$

Once $\bar{b}$ has been determined from r, ℓ, and g via (3.3-7), the death rate, d, can be obtained from (3.3-5).

If $r = 0$, i.e., the population birth and death rates balance, the population is said to be _stationary_, and $\frac{dN_t}{dt} = 0$.

Examination of (3.3-4) and the definition of R_0 confirms the following intuitive relationships between R_0 and r: If $R_0 < 1$, then $r < 0$, and the population numbers are decreasing. If $R_0 > 1$, then $r > 0$ and $N_t(t)$ is increasing. If $R_0 = 1$, then $r = 0$ and the population is stationary.

We have shown that Lotka's asymptotic results A - C and many of the vital statistics useful to demographers can be rigorously obtained in the more general context of physiological age structures.

The asymptotic results A - C were recognized for discrete p-age models by Lefkovitch [1965]. In his discrete matrix model, described briefly in Section 2.3, the shape of the p-age distribution is given by the eigenvector associated with the real eigenvalue of the projection matrix, M, which has the largest absolute value. The Equation (3.3-3) has the advantage of expressing the stable p-age distribution directly in terms of r and individual growth and survivorship functions.

3.4 The Shape of the Stable P-Age Distribution

Let us take a closer look at the shape of the stable p-age distribution, $c(z)$, as specified in (3.3-3). How does it compare with the stable c-age distribution derived by Lotka?

The stable c-age distribution is given [Keyfitz, 1968] by:

$$c(a) = \frac{e^{-ra}\ell(a,0)}{\int_0^\infty e^{-ra}\ell(\tilde{a},0)d\tilde{a}} \cdot \qquad (3.4-1)$$

As would be expected, this is a special case of (3.3-3) with $g(z) = 1$ and $z = a$, chronological age.

For the sake of simplicity, assume that we are discussing the p-age and c-age structures, $c_s(z)$ and $c_s(a)$, of stationary populations. Then $r = 0$, and

$$c_s(z) = \frac{\dfrac{\ell(z,z_0)}{g(z)}}{\int_{z_0}^\infty \dfrac{\ell(\tilde{z},z_0)}{g(\tilde{z})}d\tilde{z}} , \qquad (3.4-2)$$

while

$$c_s(a) = \frac{\ell(a,0)}{\int_0^\infty \ell(\tilde{a},0)d\tilde{a}} \cdot \qquad (3.4-3)$$

Incidentally, the Equations (3.4-2) and (3.4-3) may also be obtained by setting $\frac{\partial N}{\partial t} = 0$ in partial differential Equations (2.2-1) and (2.3-2), respectively, and solving the resulting ordinary differential equations for the stationary number density, N.

In (3.4-3), the shape function $c_s(a)$ must be a nonincreasing function of c-age, since it is a normalized, c-age-specific survivorship curve.

The function $c_s(z)$ is more complex, however. Its most interesting

feature is that it need <u>not</u> be a nonincreasing function of p-age, z.

This is more clearly seen by using the definition of survivorship,

(2.4-6), along with Equation (2.4-5), to rewrite $c_s(z)$ as

$$c_s(z) = \frac{\exp\left[-\int_{z_0}^{z}\left(\frac{g'(\tilde{z})+\mu(\tilde{z})}{g(\tilde{z})}\right)d\tilde{z}\right]}{\int_{z_0}^{\infty}\exp\left[-\int_{z_0}^{\tilde{z}}\left(\frac{g'(\hat{z})+\mu(\hat{z})}{g(\hat{z})}\right)d\hat{z}\right]d\tilde{z}} \qquad (3.4-4)$$

The nature of this shape function becomes apparent if it is

differentiated to get

$$\frac{dc_s(z)}{dz} = -\left[\frac{g'(z)+\mu(z)}{g(z)}\right]c_s(z). \qquad (3.4-5)$$

Since $g(z)$ and $c_s(z)$ are both positive functions, the sign of $\dfrac{dc_s(z)}{dz}$

depends on the quantity $-[g'(z)+\mu(z)]$. If, over some p-age range

$[z_1,z_2]$, the quantity $-[g'(z)+\mu(z)]$ is greater than zero, then the shape

function $c_s(z)$ will actually increase in z over the interval $[z_1,z_2]$.

One would see a stationary p-age distribution which looked like

Figure 3.1.

The curve in Figure 3.1 illustrates the steady-state culmination

of the "compression" and "expansion" phenomena described for a cohort

density function in Section 2.5. The positive slope of the shape curve

$\left(\dfrac{dc_s(z)}{dz} > 0\right)$ seen between z_1 and z_2 is the steady-state version of Case

2 in Section 2.5. A rapidly decreasing growth rate and low mortality,

i.e., $-[g'(z)+\mu(z)] > 0$, force a "compression" in the number density,

N, and result in a "stacking up" of individuals over the interval
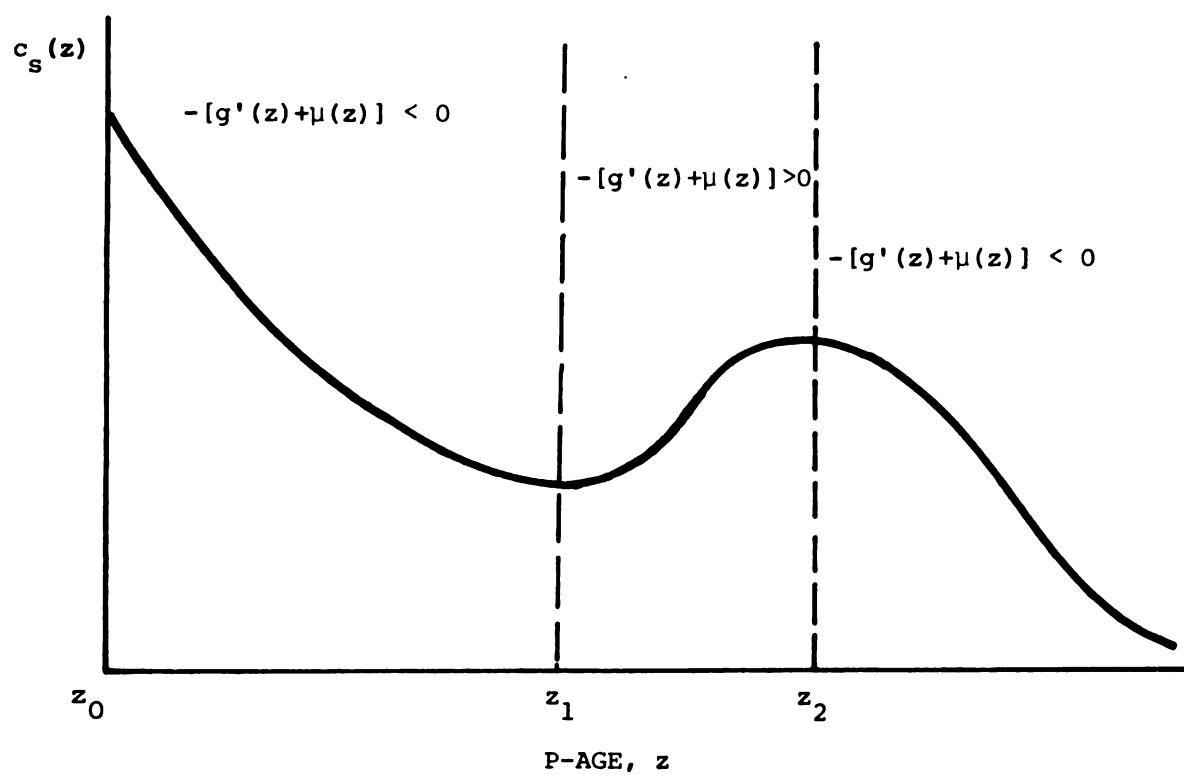
Figure 3.1. Hypothetical p-age frequency distribution for stationary population.

$[z_1, z_2]$. For the population with a full spectrum of p-aged individuals represented, the number density increases in z over the interval $[z_1, z_2]$, as it did for the cohort in Section 2.5.

A third factor, besides individual growth and mortality, shapes the stable p-age distribution if the population is not stationary. In this case, the total population numbers are increasing or decreasing at a rate r, and the shape function c(z) is given by (3.3-3). To put (3.3-3) in forms similar to (3.4-4) and (3.4-5), it is first necessary to recall the definition of $\tau(z, z_0)$, Equation (2.4-3). The exponential term in (3.3-3) can then be written as

$$e^{-r\tau(z, z_0)} = \exp\left[-\int_{z_0}^{z} \frac{r}{g(\tilde{z})} d\tilde{z}\right].$$

As in the derivation of (3.4-4), Equations (2.4-5) and (2.4-6) are used to express the term $\dfrac{\ell(z, z_0)}{g(z)}$ of (3.3-3) in exponential form. When the terms of (3.3-3) are reassembled, they appear as

$$c(z) = \frac{\exp\left[-\int_{z_0}^{z} \left[\frac{r+g'(\tilde{z})+\mu(\tilde{z})}{g(\tilde{z})}\right] d\tilde{z}\right]}{\int_{z_0}^{\infty} \exp\left[-\int_{z_0}^{\tilde{z}} \left[\frac{r+g'(\hat{z})+\mu(\hat{z})}{g(\hat{z})}\right] d\hat{z}\right] d\tilde{z}} \qquad (3.4-6)$$

Upon differentiation, this becomes

$$\frac{dc(z)}{dz} = -\left[\frac{r+g'(z)+\mu(z)}{g(z)}\right] c(z). \qquad (3.4-7)$$

This equation shows that c(z) increases or decreases depending upon the sign of the quantity $[r + g'(z) + \mu(z)]$.

3.5 The Stable P-Age Distribution--Discussion

Equation (3.4-6) describing the stable p-age distribution lays

bare some problems encountered by an experimenter observing a p-age

distribution in a laboratory or wild population which is assumed to have

a stable p-age structure. Individual growth rate, g, mortality, μ,

and the intrinsic rate of increase, r, interact in a complex manner to

determine the shape of the observed p-age frequency distribution.

Determination of any one of these vital statistics from an observed

p-age distribution requires knowledge of the other two.

This problem has been appreciated by many modelers. This section

will briefly discuss three different models from the literature, and

will concentrate on the efforts of each modeler to use experimental

p-age frequencies to determine growth and mortality rates.

Bell and Anderson [1967] seem to be the only other authors to

recognize the relationships between growth and mortality expressed by

(3.4-6). They employ a partial differential equation like (2.3-1) to

describe cell populations whose members are classified by cell volume

and c-age. The c-age variable is eliminated through simplifying assump-

tions, and a model of the form (2.2-1) results, with the p-age variable

of cell volume. Bell and Anderson assume a population is growing

exponentially, so that a stable cell volume distribution is achieved.

Their equation (17) for the stable volume distribution is equivalent,

except for a normalizing factor, to Equation (3.4-6) with $\mu(z) = 0$.

They discuss the problems of determining the intrinsic rate of

increase and the volume-specific rates of cell growth and division when

the only available data are volume frequency distributions of experimental

cultures. Due to the nature of cell life histories, Bell and Anderson's discussion can not be easily generalized to other populations. For example, although one can safely assume that few cells die before division, the very process of cell division removes parent cells from the population. The mortality rate can be assumed = 0 in cell populations, but a loss rate term due to cell division must be added. The shape of the stable volume distribution expressed by their equation (24) is a function of cell division and growth rates and the intrinsic rate of increase. Bell and Anderson conclude that it is impossible to uniquely determine cell division and growth rates from volume frequency data, but they suggest methods of estimating "reasonable" rates in an interative fashion.

This provides an example of the possible value of (3.4-6). Because Bell and Anderson could derive the exact dependence of the cell volume distribution on growth and cell division rates, expressed in equations analogous to (3.4-6), they were able to devise procedures for estimating these vital statistics from experimental volume distributions.

The problem of estimating vital statistics from experimental p-age distributions also arises with discrete p-age models. As we have already mentioned, Lefkovitch [1965] originally could not quantitatively interpret the elements of his projection matrix, M, in terms of growth and mortality. More recently [1971], he has clarified the relationship between growth and mortality by writing the matrix M as:

$$M = W^{-1}AW \qquad\qquad (3.5-1)$$

Here, A is the classical Leslie matrix containing c-age-specific survivorships and fecundities as entries. W is a diagonal matrix with each entry w_{ii} = mean p-age of individuals in the ith c-age class. Lefkovitch shows that the matrix Equation (3.5-1) transforms a c-age matrix, A, into a p-age matrix, M, via the (p-age):(c-age) growth data contained in W.

In the context of Lefkovitch's model, the procedure for estimating vital statistics can be summarized as follows: a sequence of observations from p-age distributions which are assumed to be stable are used to estimate the entries in M for a specific population [Lefkovitch, 1965]. Given M, Equation (3.5-1) indicates that A and W are not uniquely determined. Independent estimates of growth can be used to estimate W. Then A, with its fecundity and mortality statistics, is computed from $A = WMW^{-1}$. This method is a discretized alternative to the use of (3.4-6). The most obvious disadvantage of the discrete method is that it is not suitable for analytical purposes. The quantitative tradeoffs among mortality, growth, and fecundity in determining the stable p-age distribution cannot be traced through the discretization process and its attendant matrix manipulations.

It remains to be seen whether Lefkovitch's methods for parameter estimation in the discrete model will satisfy Equation (3.4-6) as time intervals and p-age classes become infinitesimal in length.

A discrete approach similar to that of Lefkovitch is taken by Usher [1966] in his model of selection forests. A vector of size classes for trees based on trunk girth is determined and a transition matrix like that of Lefkovitch is used to update the size-distribution

vector. In Usher's model, the projection matrix takes a particularly simple form. The update interval is chosen so that trees can advance at most one size class during the interval. This assumption results in a projection matrix, M, with non-zero entries only on the diagonal. In addition, mortality is assumed to be zero during the update interval, so the entries in M are a function solely of growth rate. These assumptions make it possible to compute size-specific growth rates directly from two sequential size-frequency observations.

The three examples given above have a common theme. In each case, the authors attempt to compute a vital population rate--growth, fecundity, or mortality rate, or the intrinsic rate of increase. The values of other vital statistics are assumed to be known. The authors then use empirical p-age distributions, which are assumed stable, to determine the unknown vital rate, which may be a function of p-age.

The methods of Bell and Anderson and of Usher seem to produce valuable results. However, each method is applicable only to that small class of populations with life-history phenomena similar to that of the population for which it was designed.

Although the method of Lefkovitch has a greater generality than those of Bell and Anderson or Usher, it must be applied to data on a case-by-case basis and cannot make predictions based on vital rates which are functions, not data values.

We claim that the equations in Sections 3.3 and 3.4 for stable or stationary p-age distributions are a basis for procedures to compute vital rates of any stable population classified by p-age which has observable p-age distributions.

R_0 and r can be determined from (3.3-4) with a knowledge of $b(z)$, $\ell(z,z_0)$, and $g(z)$. The crude birth rate, b, is given by (3.3-7), which uses growth and survivorship functions, and r. Equations (3.4-4) and (3.4-6) give the relationships among $c_s(z)$, $c(z)$, $g(z)$, $\mu(z)$, and r. Chapter 4 will give an example of the use of (3.4-4) in determining mortality rates for fish populations.

As a caution, it should be noted that application of these formulae will not be as simple as it may appear. The equations of Chapter 3 are expressed in terms of continuously varying functions of physiological age. There will be a considerable amount of work involved in developing a sampling theory for estimation of these vital parameters. Even the use of discrete-age and discrete-time data in the parameterization of the continuous (c-age)x(time) models of Lotka and von Foerster still presents statistical problems, as a reading of Keyfitz [1968] shows. Nonetheless, it is clear that the equations for vital statistics in this chapter can increase the utility of observing and modeling populations on a demographic basis other than chronological age. Future work on this model will proceed toward this goal.

CHAPTER 4

APPLICATIONS TO FISH POPULATION DYNAMICS

4.1 Introduction

Partly because of their importance as a food source for man, fish populations have received a great deal of attention by ecologists. Fishery biologists have long understood that a detailed knowledge of fish population dynamics is essential for the efficient management of exploited fisheries.

For these reasons, there is a large amount of data available on the vital statistics of fish populations, and a good deal of work has been done in the development and practical use of age-structured models of fisheries. A good summary of such models can be found in Gulland [1962].

A key factor in fish population models is individual growth in body size. Fish are exploited by man on a size-specific basis, and the total biomass yield of a fishery cannot be determined without knowledge of population size distributions.

However, the dynamics of size distributions in fish populations have been difficult to examine, for several reasons. In the first place, individual fish typically show very labile growth. Individuals which survive the very high probability of death in the egg and larval stages will respond to changes in ambient temperature or available resources by changing their growth rates [Weatherley, 1972]. This indeterminate growth is generally felt to be the principal form of self-regulation

for many populations of marine and freshwater fish [Weatherly, 1972; Backiel and LeCren, 1967].

The ages of fish in wild populations are often difficult to determine. A great deal of effort has been expended in trying to age individuals from various species, and the methods which are currently used, such as finding aging marks on scales or various bones, are quite tedious [deBont, 1967]. These aging methods decrease in accuracy for older fish due to generally decreased growth rates and individual differences in growth for fish of the same age [deBont, 1967; Weatherley, 1972].

A further complication in the attempt to study dynamics of fish populations is that individual growth, fecundity, and mortality are largely size-dependent rather than age-dependent. It has already been mentioned that losses due to human harvesting are size-specific, and there is evidence that natural predators also select fish on the basis of size [Earl Werner, pers. comm.]. Several authors discuss the correspondence of fecundity [Weatherley, 1972; Bagenal, 1967] and growth rate [Weatherley, 1972; Parker and Larkin, 1959] with size as opposed to age.

For these reasons it seems clear that dynamic population models based on size distributions might be easier to work with than those based on population age structure. However, most of the theoretical models which have been developed to estimate yields, recruitment, and survivorship in commercial fisheries are age-structured. This is due in part, no doubt, to the marked seasonal reproduction of most temperate zone fish. Females in most populations spawn during only a

few weeks in the spring. Thus, a fish population is most often visualized as comprised of a set of "year classes", which form the peaks of traveling waves moving through an age distribution. Figure 4.1 shows a snapshot in time of an idealized age distribution arising from seasonal reproduction. The peaks in the waves are separated by approximately one year.

The most widely-used models [Beverton and Holt, 1957; Ricker, 1958] employ this concept of year classes with simplified assumptions concerning mortality and recruitment to describe population age structure. The age distributions are then combined with size-at-age data or fitted growth curves to give a picture of the population size distribution.

The remainder of this chapter will try to show how the p-age model developed in Chapters 2 and 3 can be a useful alternative to models based on year classes in studying fish populations. Rather than discuss a single application of the p-age model in detail, the chapter aim is to illustrate how a variety of new perspectives on population dynamics emerge when individuals are classified by p-age instead of c-age.

Section 4.2 shows the relationships of the p-age model to commonly-used methods of computing production in fish populations. Sections 4.3 and 4.4 examine fish growth and recruitment, respectively, in the context of the p-age model of Chapters 2 and 3. The two sections discuss growth and recruitment models used by Beverton and Holt [1957] in their comprehensive commercial fishery model, and Section 4.5 examines the p-age distributions associated with their model populations. In Section 4.6 we turn to a different topic. This section examines the problem
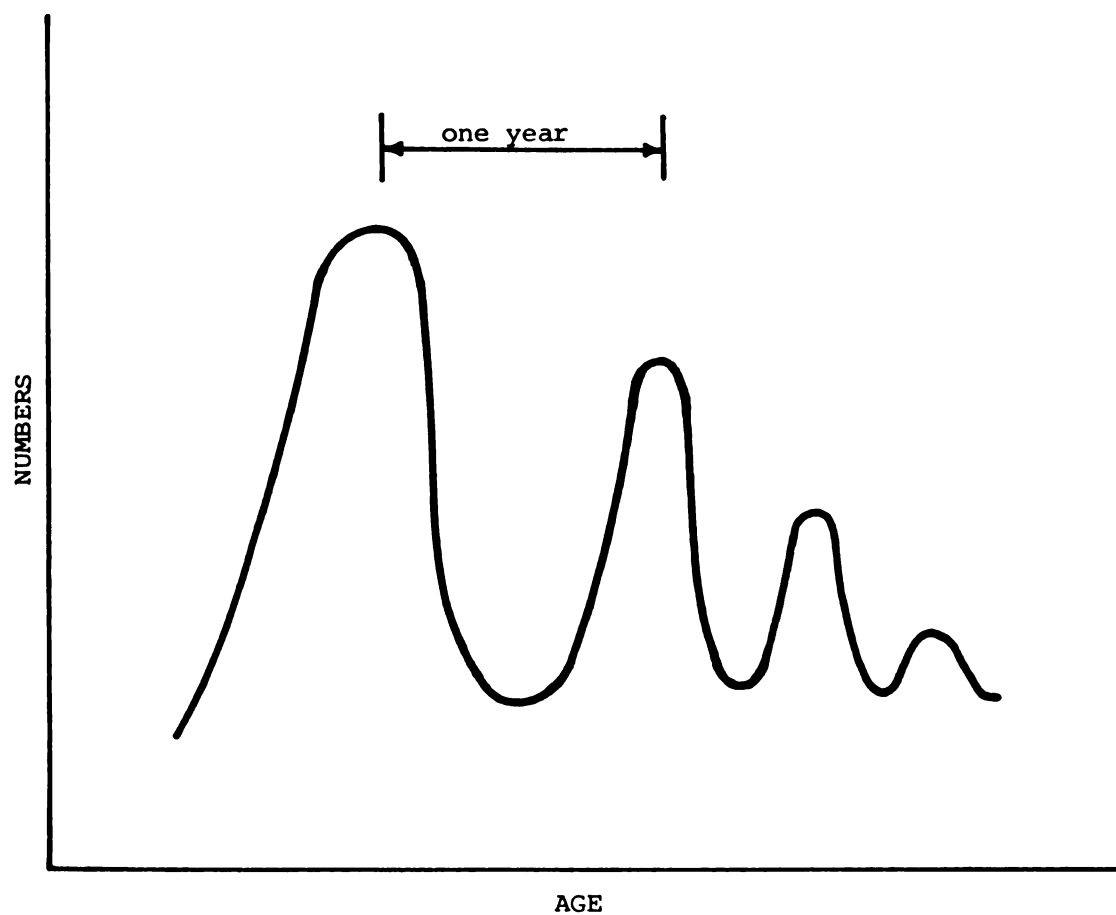
Figure 4.1. Idealized age distribution at fixed point in time, showing year classes arising from seasonal reproduction.

of estimating fish mortality rates from sampled size or age frequency distributions.

In this chapter, the p-age variable, z, will represent body size, either length or weight, of individual fish. The state variable of the model is the function N(z,t), the number density of fish of size z at time t. The dynamics of a fish population are then described by the system (2.2-1), (2.2-7). From a modeling standpoint, one advantage of using this system to describe a fish population is immediately apparent. All three vital rates of fish population dynamics--growth, fecundity, and mortality--are explicitly contained in the dynamical equations.

4.2 Biomass and Production

Computation of biomass production for a population is of primary interest to fishery biologists as well as other resource managers. Production over a time interval $[t_1, t_2]$ is defined as the total biomass elaborated by the fish population, regardless of the ultimate fate of the biomass [Chapman, 1967]. Thus, production during $[t_1, t_2]$ is equivalent to the total net change in population biomass, plus the sum of the weights of all fish that die during $[t_1, t_2]$ with weight being assessed at the moment of death [Chapman, 1967].

From the model (2.2-1), (2.2-7), a differential equation can be derived for population biomass. If the size variable, z, is chosen to be body weight, then

$$\int_{z_0}^{\infty} \tilde{z}\, N(\tilde{z},t)\, d\tilde{z} \triangleq N_B(t) \triangleq \text{population biomass.} \qquad (4.2-1)$$

When differentiated, this takes the form

$$\frac{dN_B(t)}{dt} = \int_{z_0}^{\infty} \tilde{z} \, \frac{\partial N(\tilde{z},t)}{\partial t} \, d\tilde{z},$$

by Liebniz rule. Using (2.2-1), this can be written

$$\frac{dN_B(t)}{dt} = \int_{z_0}^{\infty} \tilde{z} \left[-\frac{\partial g(\tilde{z},t)N(\tilde{z},t)}{\partial z} \right] d\tilde{z} - \int_{z_0}^{\infty} \tilde{z}\mu(\tilde{z},t)N(\tilde{z},t)d\tilde{z}.$$

If the first term is integrated by parts, the equation becomes

$$\frac{dN_B(t)}{dt} = z_0 g(z_0,t)N(z_0,t) + \int_{z_0}^{\infty} g(\tilde{z},t)N(\tilde{z},t)d\tilde{z} - \int_{z_0}^{\infty} \tilde{z}\mu(\tilde{z},t)N(\tilde{z},t)d\tilde{z},$$

assuming no individuals survive to size $z = \infty$, so that $N(\infty,t) = 0$. This can be also written

$$\frac{dN_B(t)}{dt} = z_0 B(t) + \int_{z_0}^{\infty} g(\tilde{z},t)N(\tilde{z},t)d\tilde{z} - \int_{z_0}^{\infty} \tilde{z}\mu(\tilde{z},t)N(\tilde{z},t)d\tilde{z}.$$

$$(4.2-2)$$

Equation (4.2-2) is an equation for net population biomass change which shows clearly the three possible sources of biomass gain or loss in any closed population. The first term in (4.2-2) is the rate of addition of new biomass due to reproduction, the second term is the addition of new biomass due to growth, and the third term is the total loss rate due to mortality.

Assume now that we wish to compute production in a fish population between time t_1 and t_2. Also assume that no reproduction occurs between t_1 and t_2 so that $B(t) = 0$ in that time interval. This is a common

assumption, since production is often computed over the summer months beginning just after the spring spawning.

Then, production = [net biomass change] + [total biomass lost to mortality]. That is,

$$P = \int_{t_1}^{t_2} \left[\frac{dN_B(\tilde{t})}{d\tilde{t}} \right] d\tilde{t} + \int_{t_1}^{t_2} \left[\int_{z_0}^{\infty} \tilde{z}\mu(\tilde{z},\tilde{t})N(\tilde{z},\tilde{t})d\tilde{z} \right] d\tilde{t}.$$

Using (4.2-2) this equation can be rewritten as:

$$P = \int_{t_1}^{t_2} \left[\int_{z_0}^{\infty} g(\tilde{z},\tilde{t})N(\tilde{z},\tilde{t})d\tilde{z} \right] d\tilde{t}. \qquad (4.2-3)$$

The use of (4.2-3) requires full knowledge of the number density $N(z,t)$ over the interval $[t_1,t_2]$. This is rarely practical for wild populations, so (4.2-3) must be simplified. Suppose the production of a year class, or cohort, is being computed as it grows during $[t_1,t_2]$.

Then $N(z,t)$ at any fixed time t, is non-zero over only a restricted size range centered roughly on the mean size of the similarly-aged individuals in the cohort. Thus, the inner integral in (4.2-3) can be approximated by

$$\int_{z_0}^{\infty} g(\tilde{z},t)N(\tilde{z},t)d\tilde{z} \simeq g(\tilde{z}(t))N_c(t). \qquad (4.2-4)$$

In this approximation, $\tilde{z}(t)$ is the mean size of individuals in the cohort at time t, and $N_c(t)$ is defined as the number of indiviudals in the cohort. Since $g(z(t)) = \frac{dz}{dt}$, production can be written as

$$P = \int_{t_1}^{t_2} \frac{dz(\tilde{t})}{dt} N_c(\tilde{t})d\tilde{t}. \qquad (4.2-5)$$

Equation (4.2-5), with slightly different notation, is equivalent to the basic production formula (9.19) of Beverton and Holt [1957]. Equation (4.2-5) is also the mathematical equivalent of the well-known "Allen curve" graphical method for computing production [Chapman, 1967; Weatherley, 1972], as is noted by Beverton and Holt [1957].

A slightly different approach is taken by Ricker [1958]. He uses a simple equation for net biomass gain in a population, and then integrates over $[t_1, t_2]$, as above, to get a formula for production.

Ricker's formula can also be derived as follows: once again, assume that biomass input from reproduction is negligible during $[t_1, t_2]$. Then Equation (4.2-2) becomes

$$\frac{dN_B}{dt} = \int_{z_0}^{\infty} g(\tilde{z}, t) N(\tilde{z}, t) d\tilde{z} - \int_{z_0}^{\infty} \tilde{z} \mu(\tilde{z}, t) N(\tilde{z}, t) d\tilde{z}$$

If $G(z, t) = \frac{g(z, t)}{z}$ is defined as the instantaneous growth rate per unit of body weight, then

$$\frac{dN_B}{dt} = \int_{z_0}^{\infty} [G(\tilde{z}, t) - \mu(\tilde{z}, t)] \tilde{z} \, N(\tilde{z}, t) d\tilde{z}. \qquad (4.2-6)$$

Now, suppose that growth per unit body weight and mortality are constants across all sizes during $[t_1, t_2]$. Let $G(z, t) = \hat{G}$ and $(z, t) = \hat{\mu}$. Then (4.2-6) becomes

$$\frac{dN_B}{dt} = (\hat{G} - \hat{\mu}) N_B.$$

From this simplified equation for biomass, Ricker derives the production formula $P = \hat{G}\bar{B}$ where $\bar{B}$ is the mean biomass during $[t_1, t_2]$. Details can be found in Ricker [1958] or Chapman [1967].

The preceding derivations are valuable for several reasons. They show that both of these commonly used production estimates are derived via approximations to the same detailed expression for population biomass (4.2-2). It appears that, this demonstration has not been made before. The derivations also clearly show the mathematical nature of the approximations inherent in the simplified production estimates. The formulae of Beverton and Holt and Allen are accurate to the extent that Equation (4.2-4) is a good approximation. This approximation, in turn, depends on the extent to which individuals in the same age cohort have similar body weights.

The accuracy of Ricker's formula, on the other hand, depends on the constancy, in time and size, of growth and mortality rates for the population size distribution being considered. Thus, Ricker's model is most suitable for single year classes with the added restriction that production be computed over short time intervals, as has been noted by Chapman [1967]. Both production estimates assume that reproduction does not contribute immediately to biomass change rates. The approximations outlined above should be kept in mind by those workers who attempt to modify fishery production equations for use in other populations whose size distributions may show quite different dynamics from those of fish.

In turn, the derivations provide a partial validation of the biomass Equation (4.2-2) and thus of the physiological age model (2.2-1), (2.2-7).

4.3 The Growth Function, g(z), for Fish Populations

Chapters 2 and 3 showed how both individual growth, g(z), and its derivative, $g'(z) = \frac{dg}{dz}$, influence the dynamics of size distributions. Growth in fish has been extensively studied, and there is a wide variety of models, both theoretical and empirical, from which to choose.

Most of the models of fish growth have one thing in common. They present growth in body weight as being a sigmoidal function of c-age. That is, fish grow at an accelerating rate up to some weight, after which growth rate decreases monotonically so that older fish appear to approach an asymptotic upper weight limit. This "logistic" type growth is shown by Figure 2.3. A single curve from the family shown in Figure 2.3a is a sigmoidal growth curve, and Figure 2.3b shows a typical growth rate curve which generates sigmoidal growth.

Curves of the type shown in Figure 2.3 are felt by D'Arcy Thompson to describe the general course of growth in many organisms, and in his treatise, "On Growth and Form" [1942], he gives numerous empirical examples of this curve, e.g., growth in humans, bamboo, whales, tortoises, and beanstalks.

There are a number of functions which describe sigmoidal growth curves, and the particular function used depends on whether one wishes to assume an upper size asymptote, and on the steepness and symmetry properties of the sigmoid curve. Comparisons of various growth functions are found in Weatherley [1972], Holt [1962], and in Beverton and Holt [1957].

In passing, it should be noted that this chapter assumes that

g(z) does not depend on time. Growth of fish in temperate-zone waters certainly has a seasonal component, with growth rates being reduced in the winter months due to lower water temperatures [Weatherley, 1972], but we will follow the lead of other fish population modelers and temporarily ignore this environmental component of growth.

The most striking effect of the sigmoidal growth pattern on the dynamics of a size distribution is due to the decrease in growth rate for larger sized fish. This effect is illustrated by Figure 4.2, which is a size-frequency distribution shown at one point in time. As a result of decreasing growth rates, the modes in the size-frequency distribution due to year classes begin to overlap for older and larger fish [deBont, 1967; Holt, 1962].

The overlapping is further aggravated in many cases by differences in growth among individuals of the same age [Weatherley, 1972]. For many populations it becomes impossible to identify age-size relationships for more than the first or second year-class by examining size-frequency distributions.

This blurred modality in size-frequency curves is the major obstacle encountered in using such curves to determine ages of larger-sized fish, and the difficult aging methods mentioned in Section 4.1 must be attempted. In the language of Section 2.5, the overlapping is caused by the "stacking up" or "compression", along the body size dimension, of different-aged individuals due to decreasing growth rates.

A well known, sigmoidal growth formula, the von Bertalanffy growth equation, will be used to illustrate how a specific function,
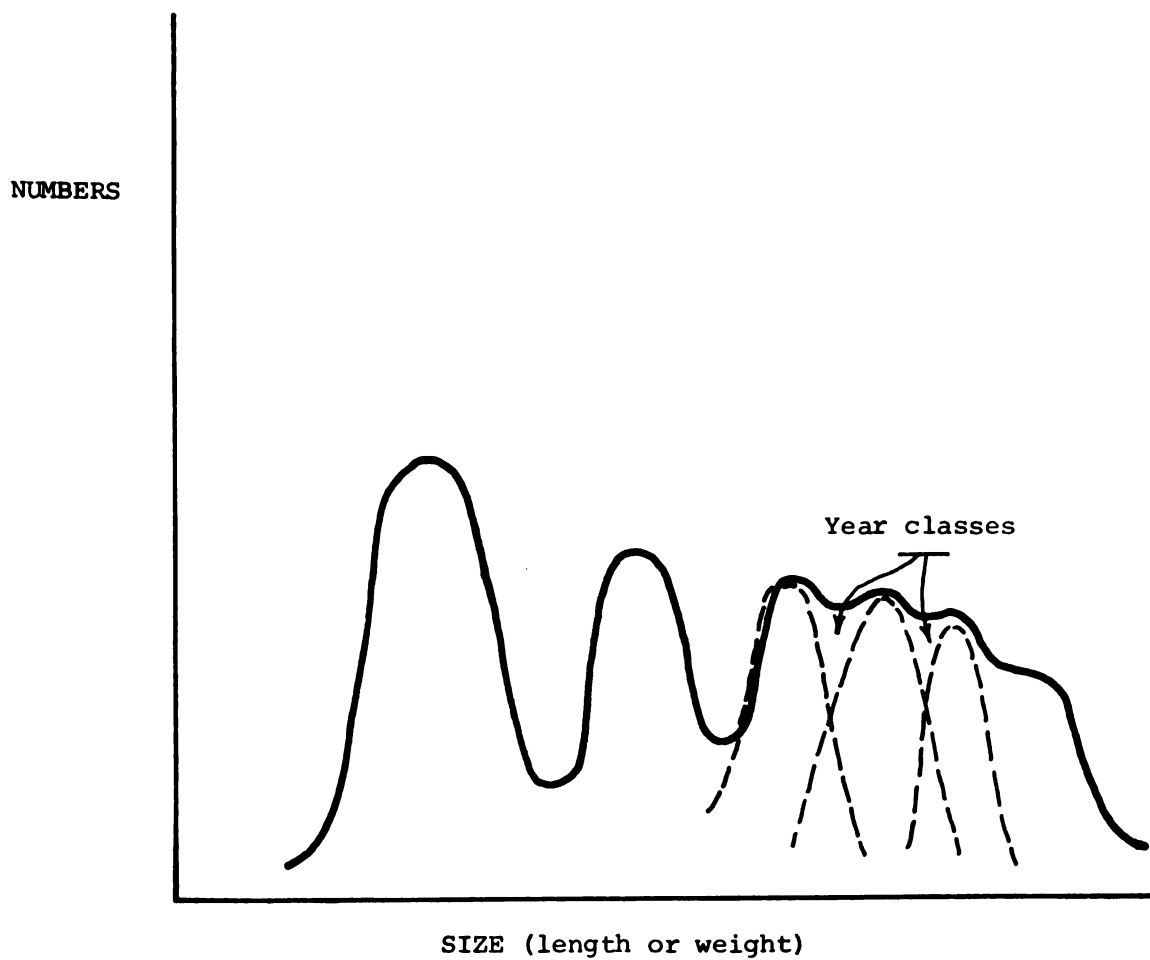
Figure 4.2. Idealized size distribution of fish showing overlapped year classes due to decreasing growth rate.

g(z), can be used in the population model. The von Bertalanffy growth

equation describes growth rate in body weight as a concave function

of weight [von Bertalanffy, 1957, 1968].

The equation for growth can be written

$$g(z) = \frac{dz}{dt} = 3K \, [z_\infty^{1/3} \, z^{2/3} - z]. \tag{4.3-1}$$

Integration of this curve gives:

$$z(t) = z_0 + (z_\infty - z_0) [1 - e^{-k(t-t_0)}]^3. \tag{4.3-2}$$

In these equations, z_∞ is the asymptotic weight limit for the

particular species and ecosystem being considered, and K is a parameter

expressing the rate of approach to the upper size limit. The functions

are plotted in Figure 4.3. Notice that the growth curve is slightly

asymmetrical, with the maximum growth rate seen about 1/3 of z_∞. The

family of characteristic curves in Figure 2.3 is composed of von

Bertalanffy growth curves. Since the equation has only been applied

in fish to post-larval growth, we let z_0 be the size of young fry at

time t_0.

This growth function has been widely used and discussed in fish

population models. It is advocated by Beverton and Holt who use it

as a basis for their theoretical age-structured models [Beverton,

1954; Beverton and Holt, 1957; Holt, 1960]. Beverton and Holt [1957]

show methods of estimating z_∞ and K from growth data, and they also

discuss the dependence of the parameters on population density.

Criticism of the von Bertalanffy equation as applied to fish growth

can be found in Parker and Larkin [1959].
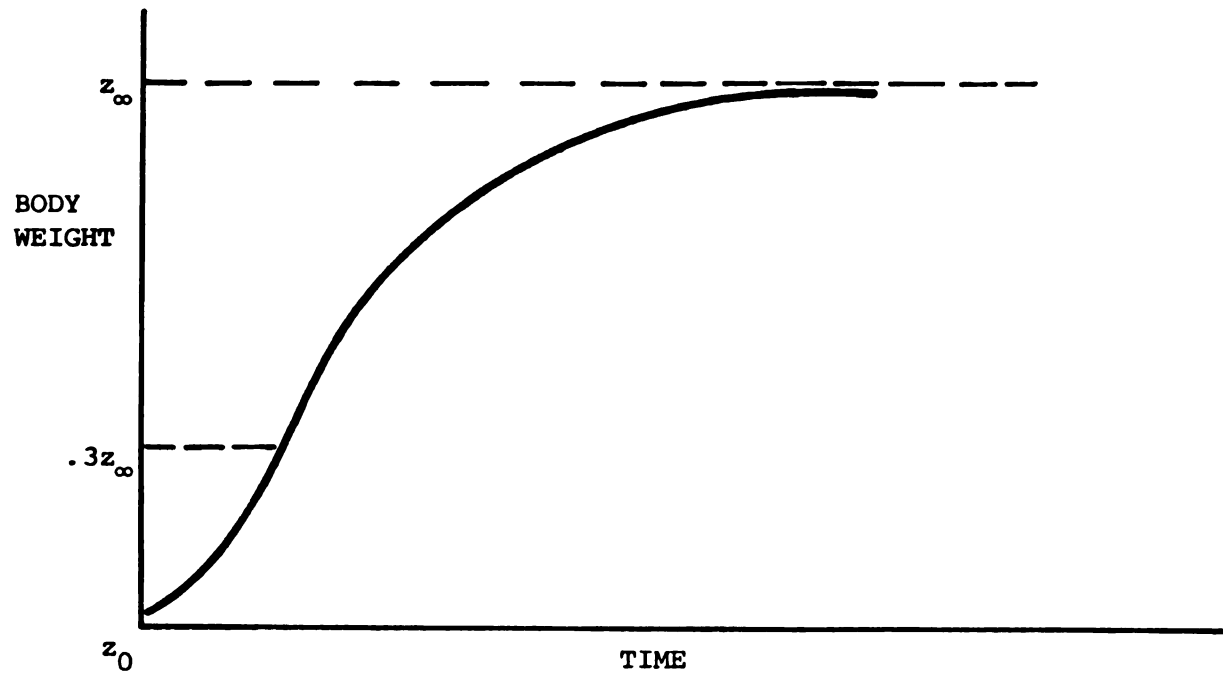
a)   von Bertalanffy growth curve.
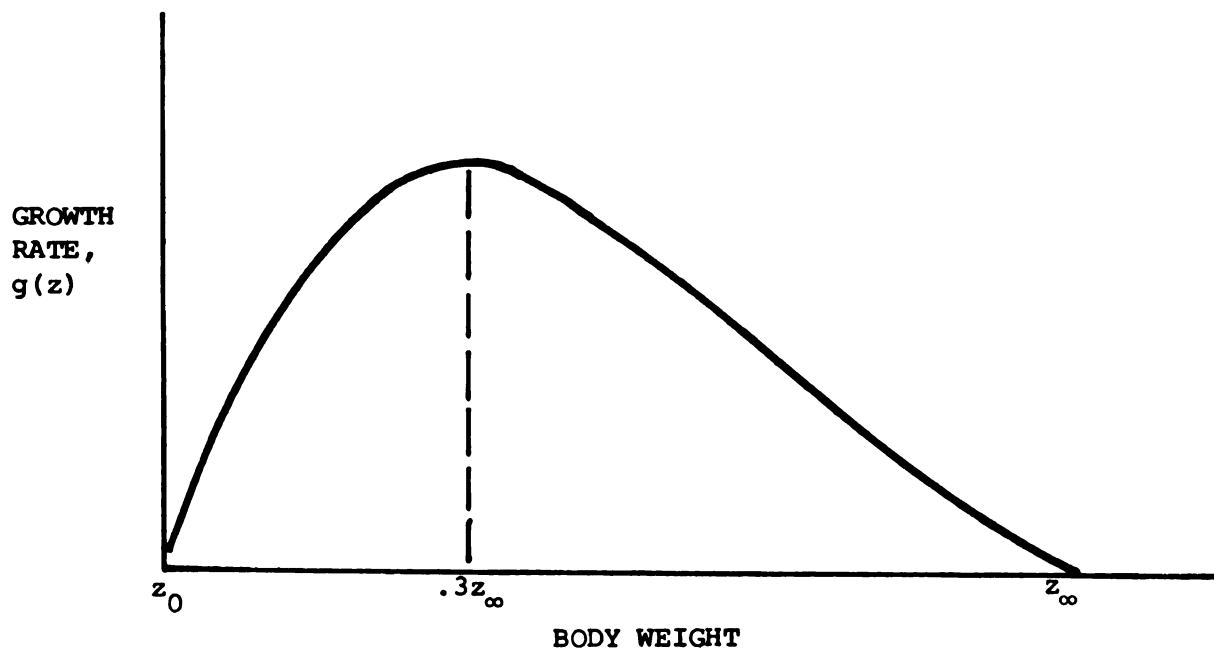
b)   Growth rate for von Bertalanffy growth curve.

Figure 4.3.

4.4 Recruitment and Seasonal Periodicity

The reproductive feedback in fish populations is poorly understood. Since mortality in the egg and larval stages is very high (>99.9+ % loss), it has been very difficult to relate the number of surviving fry of a given size to the size of the breeding population which produced them [Bagenal, 1967]. Thus, the basic models of Ricker [1958] and Beverton and Holt [1957] simply assume that a constant number, R, of recruits of a certain size, are added to the population each year. For the moment, let z_R = the size of recruits.

We will also assume, along with the modelers mentioned above, that recruitment, R, is constant from year to year and that yearly deaths balance yearly births. The hypothetical fish population is in a steady-state condition, since the net increase in population numbers is zero from year to year, but such a population is still dynamic in that a train of wavelike modes due to year classes moves through the age and size distributions.

This produces a population which is in a periodic type of steady-state condition. Weatherley [1972, p. 155] defines the broadened steady-state concept as follows:

> To say that a population is in 'steady state' means that such changes in growth, biomass, recruitment and mortality as occur, do so with a regular pattern and magnitude so that at a particular point in each phase of a population 'cycle', age and size structures will tend to be similar.

Total numbers, $N_t(t)$, in this steady-state population would show a "limit cycle" behavior, as described by May [1973]. Population numbers oscillate up and down in a stable, periodic fashion between fixed maxima and minima.

In the p-age model, Equation (3.4-2) gives the shape of the size distribution for a stationary population, but it can be adapted to this fish population which has yearly, pulsed reproduction.

Define a time-averaged size distribution,

$$\bar{N}(z,t) = \frac{1}{T} \int_t^{t+T} N(z,\tilde{t}) d\tilde{t}. \qquad (4.4-1)$$

Here, t is arbitrary and T is the period of the reproduction cycle, about 1 year in this case.

Assume that the population has been in steady-state for several generations so that $N(z,t)$ can be replaced by the expression for the solution (2.4-7b):

$$\bar{N}(z,t) = \frac{1}{T} \int_t^{t+T} N(z_R, \tilde{t} - \tau(z,z_R)) \frac{g(z_R)}{g(z)} \ell(z,z_R) d\tilde{t}. \qquad (4.4-2)$$

Notice that z_R replaces z_0 in this usage of Equation (2.4-7b), because z_R is the size at which recruits are assumed to enter the population.

Now assume that a total of R recruits enter the population at a constant rate over a time interval T_R. The interval T_R would be on the order of a few weeks to a few months for most temperate zone fish.

Then,

$$N(z_R, \tilde{t} - \tau(z,z_R)) g(z_R) = \begin{cases} \dfrac{R}{T_R}, & \text{for } \tilde{t} \text{ in } [t,t+T] \text{ and in } T_R \\ 0, & \text{for } \tilde{t} \text{ in } [t,t+T] \text{ but not in } T_R \end{cases}$$

Here, the interval T_R occurs sometime within the yearly period $[t,t+T]$. Equation (4.4-2) can now be written

$$\bar{N}(z) = \frac{R}{T} \frac{\ell(z,z_R)}{g(z)}. \qquad (4.4-3)$$

In (4.4-3) we have replaced $\bar{N}(z,t)$ by $\bar{N}(z)$ since $\bar{N}$ becomes time independent. Notice that $\bar{N}(z)$ is equivalent, except for a normalization factor, to the shape function $c_s(z)$ defined in Equation (3.4-2) for stationary populations.

This time-averaging procedure smooths out any periodic "square wave" reproductive input. Beverton and Holt [1957] use an instantaneous yearly pulse for recruitment in their models, and this is handled in (4.4-2) simply by making $N(z_R, t-\tau(z,z_R))$ a Kronecker-delta impulse function in the variable t. Ricker [1958], on the other hand, assumes that recruitment occurs at a constant rate over the whole year in his model. In this case $T_R = T$ and the time average in (4.4-3) still applies. The remainder of this chapter will discuss the time-averaged size distribution, $\bar{N}(z)$, of steady-state fish populations with pulsed reproduction.

We ask the reader to look again at the idealized size-frequency distribution for fish in Figure 4.2 and imagine its fluctuations during the course of a year. The train of year-class modes will move to the right in the distribution, and sometime during the year, a new wave of recruits will appear at the left side of the distribution. However, because of the small growth rate in larger-sized fish the position of the blurred year-class modes comprising the right-hand section of the curve should change little, and the relative fluctuations of numbers of larger-sized fish should be comparatively small during the year.

This observation is illustrated by Figure 4.4, a year-long series of monthly length-frequency curves drawn by Fairbridge for the tiger flathead, an important species of the east Australian trawl fishery.
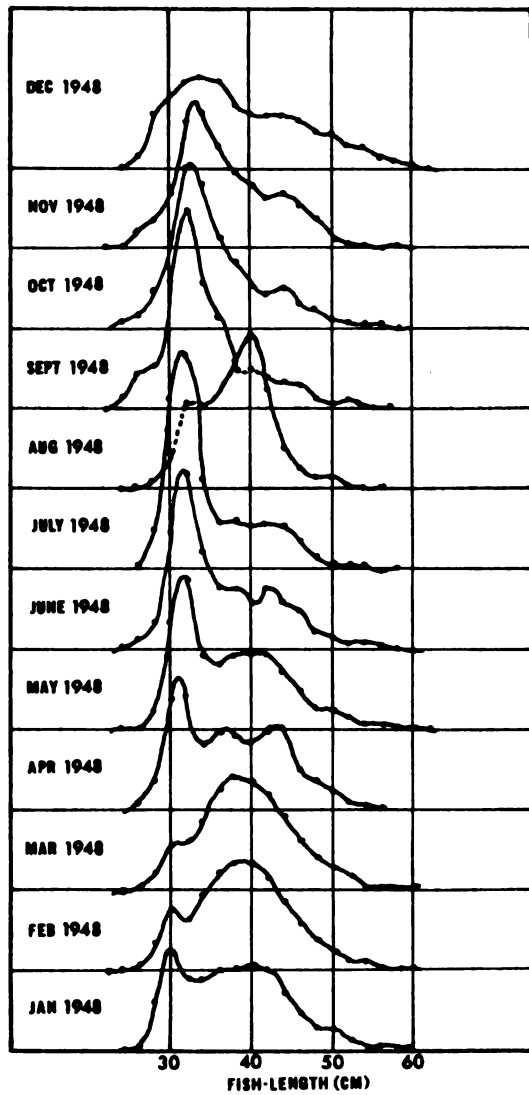
Figure 4.4. Monthly series of length frequencies for the New South
Wales Tiger Flathead population in the east Australian
trawl fishery. Taken from Fairbridge [1951].

Since spawning in this species is protracted over a six-month period, the year-class modes are not distinctly seen. However, one can see that the changes in the length-frequency curve for sizes larger than about 45 cm. are small compared with fluctuations in the curve for smaller fish.

Therefore, the number of different time points chosen for sampling such a population should not be crucial in building an estimate of the seasonal average, $\bar{N}(z)$. This observation may give some comfort to experimenters who would like to sample trawl catches as infrequently as possible.

To recapitulate: it has been shown that, for a steady-state population (annual births = annual deaths) with constant annual recruitment, the periodically-varying size distribution can be replaced by a time-averaged size distribution which is stationary in time. Furthermore, for larger fish, the average distribution, $\bar{N}(z)$, gives a good approximation to the distribution $N(z,t)$ at any instant t.

The average distribution $\bar{N}(z)$, or its normalized equivalent, $c_s(z)$, can now be used to examine the relationships between the growth and survivorship functions, g and ℓ, since these parameters are unaffected by the time-averaging procedure.

The problems associated with time-varying parameters such as reproduction in age-structured models merit a short digression. Recently, several authors have begun to examine population models with time-varying parameters and have obtained satisfying results. Lopez [1967] defines ergodic properties of a population. The tendency for a closed population to approach a stable c-age distribution when subjected to time-invariant, age-specific vital rates is called strong ergodicity. The tendency for

a closed population to have a c-age distribution which is independent of its shape in the distant past and is determined exclusively by the history of fertility and mortality is referred to as <u>weak ergodicity</u>. Lotka proved the theorem for strong ergodicity of c-age distributions, and Chapters 2 and 3 of this thesis prove the strong ergodicity property of p-age distributions.

Lopez [1967] proves a weak ergodic theorem for the continuous c-age model with some restrictions. Special cases of weak ergodicity dealing with periodically varying vital rates have recently received deserved attention. As a result, a good deal of biological realism can be added to analytically tractable population models. Oster and Takahashi [1973], using the von Foerster model (2.3-2), discuss the results of synchrony or asynchrony between an environmentally-cued reproductive pulse and the individual maturation delay.

In an excellent paper on seasonal periodicity, Skellam [1967] shows the weak ergodic property for a Leslie matrix model whose parameters vary periodically. The normalized age-frequency vector is shown to depend asymptotically only on the season of the yearly cycle at which it is computed.

Cull and Vogt [1974] also prove a weak ergodic theorem for the Leslie model--namely, that a projection matrix with periodic entries results in a stable c-age distribution for the average value of the asymptotic population vectors over one period.

We conjecture that weak ergodic properties shown for the discrete age-time models will also be seen in continuous age-time models with periodic vital rates. Further, it is felt that these same ergodic

properties will be seen in p-age distributions. The time-averaging

results of this chapter for the steady-state size distribution with

periodic reproduction are a special case of the weak ergodic property

for the continuous p-age model.

4.5 Application to a Specific Model

A well-known theoretical fishery model can now be examined from

the p-age point of view. In their basic model for a fishery, Beverton

and Holt [1957] assume a steady-state population of the type described

in Section 4.4.

The model of Beverton and Holt was specifically derived for

computing yields of exploited populations, and its many results will

not be reviewed here. However, the fundamental components of the model

will be outlined.

In addition to the steady-state assumption, Beverton and Holt

assume that the number of fish surviving to age a, out of R fish

recruited into the population at age a_R, is given by $Re^{-(F+\mu)(a-a_R)}$.

The total instantaneous mortality rate is the sum of a natural mortality

rate, μ, and a fishing mortality rate, F, both constants. The expression

is assumed to be valid for fish up to an age a_λ, the maximum age of

individuals sampled for the fishery.

The weight of an individual fish aged a is given by the von

Bertalanffy formula (4.3-2). Using these expressions for survivorship

and growth, Beverton and Holt derive equations for biomass yield,

production, and other statistics of interest for a given population.

Let us discuss the size distribution, $\bar{N}(z)$, associated with the

growth and survivorship functions described above. Combining (4.4-3)

and (2.4-5) yields

$$\bar{N}(z) = \frac{R}{Tg(z_R)} \exp\left[- \int_{z_R}^{z} \frac{g'(\tilde{z}) + \mu(\tilde{z})}{g(\tilde{z})} \right] d\tilde{z}. \qquad (4.5-1)$$

For von Bertalanffy growth, $g'(z)$ is obtained by differentiating (4.3-1)

to get

$$g'(z) = K\left[2\left(\frac{z_\infty}{z}\right)^{1/3} - 3 \right]. \qquad (4.5-2)$$

Thus, letting $T = 1$ year, $\bar{N}(z)$ becomes

$$\bar{N}(z) = \frac{R}{g(z_R)} \exp\left[- \int_{z_R}^{z} \frac{K\left[2\left(\frac{z_\infty}{\tilde{z}}\right)^{1/3} - 3\right] + (\mu+F)}{3K\left[z_\infty^{1/3}\tilde{z}^{2/3} - \tilde{z}\right]} d\tilde{z} \right]. \qquad (4.5-3)$$

This equation gives the size distribution for the fish populations

described by Beverton and Holt's basic model. Figure 4.5 shows $\bar{N}(z)$ for

two different unexploited populations of North Sea plaice, using parameter

values estimated by Beverton and Holt [1957]. For these populations,

Beverton and Holt are interested in fish between the ages of $a_R = 3.7$

years and $a_\lambda = 18$ years. For $K = 0.1$, z_{R1} and $z_{\lambda 1}$ are the corresponding

upper and lower size limits, while z_{R2} and $z_{\lambda 2}$ are upper and lower size

limits, respectively, for $K = 0.2$. The curve for $K = 0.1$ describes a

"canonical" population whose statistics are felt to accurately reflect

pre-WWII conditions in the population. Beverton and Holt use this set

of parameters as a baseline for parameter perturbation analyses. This

particular size distribution could be compared with the original weight

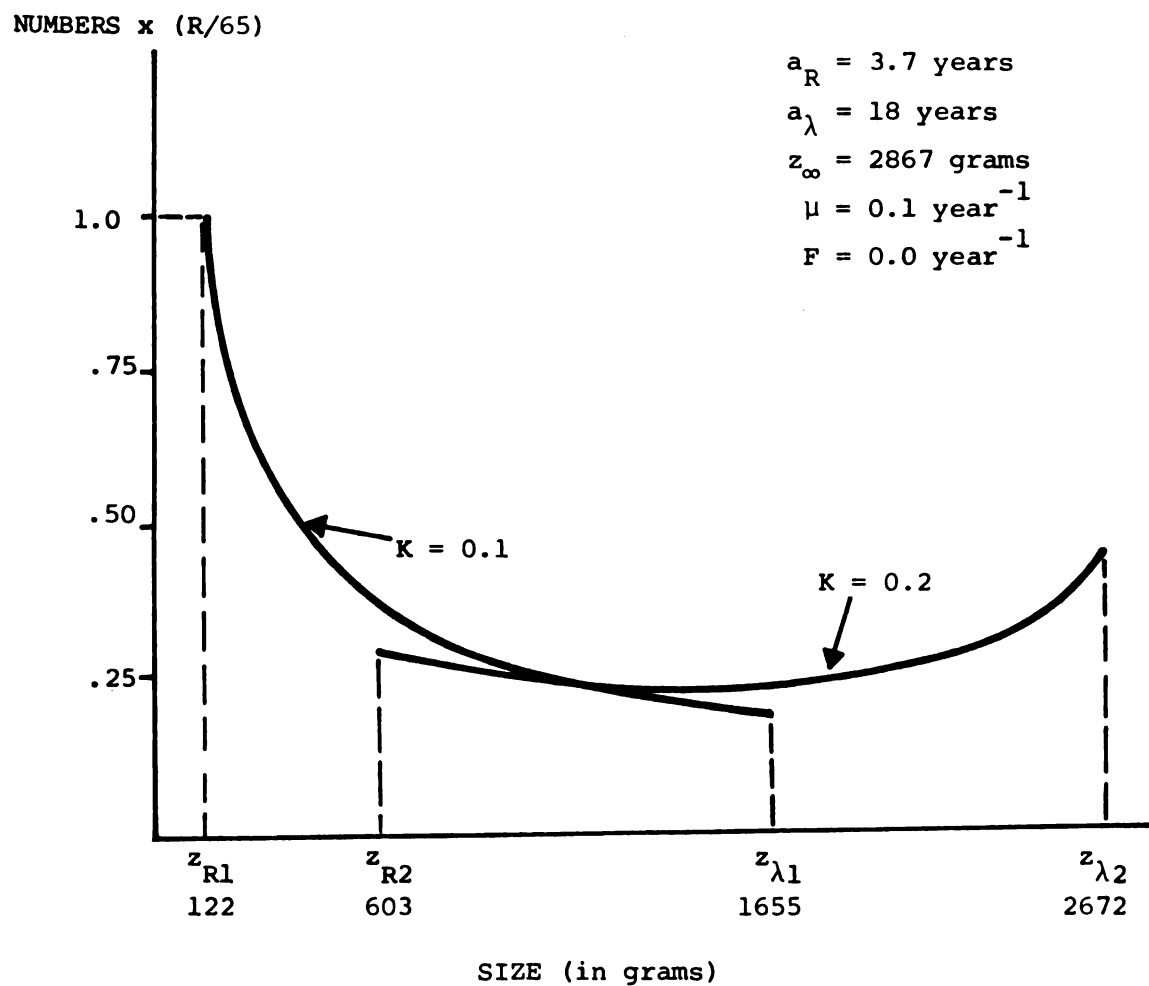frequency data to validate the derived values of μ, K, and z_∞.

Figure 4.5. Steady-state size distributions associated with a theoretical fishery model. Parameters from Beverton and Holt [1957]. (See text for explanation.)

As was pointed out in Section 3.4, a size-frequency curve can actually increase in z, if the condition $[g'(z)+\mu(z)] < 0$ holds over some interval $[z_1, z_2]$. For the size distribution $\bar{N}(z)$ described by (4.5-3), $g'(z)$ decreases monotonically to $(-K)$ as z increases to z_∞. (See Figure 4.3b and Equation 4.5-2.)

Thus, if $(-K + (\mu + F)) < 0$, the size distribution $N(z) \rightarrow \infty$, as $z \rightarrow z_\infty$. Since this can not be the case in an actual size distribution, caution must be exercised in choosing values for K, μ, and F for the Beverton-Holt model.

Even if the maximum size of fish seen in the population is a value $z_\lambda < z_\infty$, one may still get size distributions increasing in z. This is illustrated by the graph in Figure 4.5 with K = 0.2.

The fish in this hypothetical population grow much faster than those in the plaice population with K = 0.1. Notice that, with K = 0.2, the number density of fish increases for fish larger than about 1300 grams.

The effects of altering K on the yield of a population are discussed in Section 17.5 of Beverton and Holt's monograph. They conclude that a population of faster-growing fish (K = 0.2) would produce a greater biomass yield to fishing at low values of F than one with slower growing fish.

This is corroborated by comparing the two curves in Figure 4.5. The steady-state population with K = 0.2 shows a greater number of larger-sized fish available in the exploitable size range, and one would expect greater yield from such a population if it were not fished intensively enough to radically change its size structure. Figure 4.5

is also a striking illustration of the problems encountered in assuming,
along with Beverton and Holt, that fish growing at widely different
rates (K = 0.1 or K = 0.2) become liable to capture at a fixed age,
rather than a fixed size. Fishing gear must select fish largely on the
basis of size, and fish at the assumed age of recruitment, a_R, in the
two populations in Figure 4.5 differ in weight by a factor of 5.

4.6 Catch Curves and Mortality Estimation

In the preceding section, $\bar{N}(z)$ was used to predict the size
distribution associated with a given set of growth and mortality para-
meters for a steady-state population. This procedure can be reversed.
Empirical size distributions can be used to estimate growth and
mortality statistics for populations.

A catch curve is the size-frequency distribution of a sample of
the population at one point in time. The population is usually presumed
to be in steady-state for analysis of such distributions. The sample
is often taken from the catch of a commercial fishing vessel. Catch
curves are used primarily for assessing survivorship in fish populations,
whether mortality is due to natural causes or fishing. Chapter 2 of
Ricker's Handbook [1958] is a detailed account of the analysis of catch
curves for mortality estimation, and most of this section is based on
his work.

A typical catch curve is shown in Figure 4.6. Such curves are
characteristically dome-shaped. The ascending left-hand limb reflects
increasing selectivity of fishing gear for larger fish and is not
representative of the actual size distribution. The descending right-
hand limb, however, is taken to be a true sample of the actual population
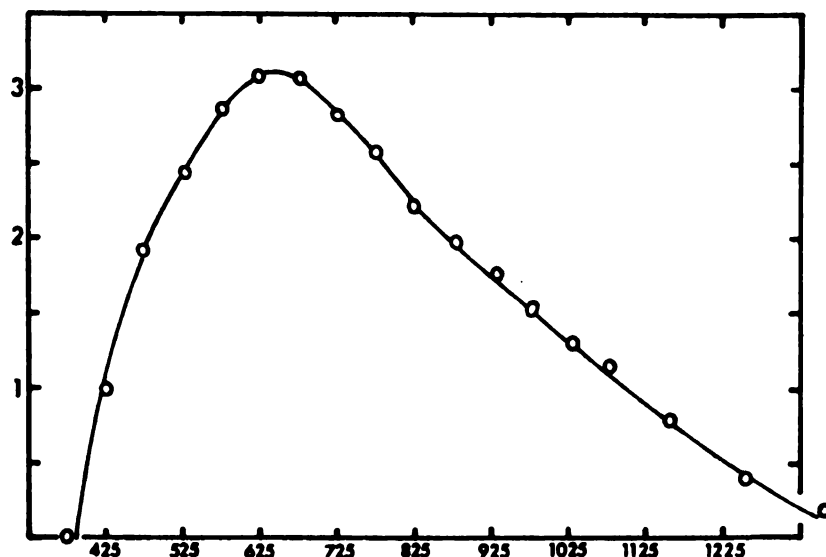
Figure 4.6.   Catch curve for the Pacific Halibut population, 1925-1926.
Abscissa--mean length of successive 5 centimeter length
groups, in millimeters. Ordinate--logarithm of the number
of fish taken at each length interval. From Ricker [1958].

size distribution. In our notation, the right-hand limb of a catch

curve is an estimate of a segment of $\bar{N}(z)$, the steady-state size

distribution.

Fishery biologists, like the modelers mentioned in Section 3.5,

recognize that a size-frequency distribution of a steady-state population

shows the compounded effects of growth and mortality. To estimate

mortality in a population, growth must be "factored out" of the size-

frequency curve. For this reason, most recent authors use catch curves

showing chronological age, rather than body length or weight, along the

abscissa [Ricker, 1958]. Mortality is easily determined from catch

curves which are c-age-frequency distributions. A catch curve based on

c-age is equivalent to an empirical estimate of c-age-specific

survivorship, $\ell(a,0)$, except for a scale factor (see Equation (3.4-1) with $r = 0$). The definition of survivorship, Equation (2.4-6), can then be used to estimate mortality.

However, catch curves based on age are often difficult to obtain. First, a large sample must be obtained and a size-frequency catch curve drawn. Then, subsamples of the original sample are taken for age determination. From the resulting size-at-age data, the size-frequency curve is transformed to an age-frequency distribution [Seber, 1973; Ricker, 1958].

There are situations when the direct use of a size-frequency curve is more attractive. To quote Ricker [1958 , p. 72],

> For example, when it is a question of assembling a repre-
> sentative sample of the catch from a widely scattered fishery,
> it may be necessary to sample so many fish that determina-
> tion of the age of all of them becomes very tedious. . . .
> In such a situation there would be two curves available:
> (a) a curve of mean length against age, based on a relatively
> limited body of data, and (b) a representative curve of the
> logarithm of frequency against length, based on all the
> samples available.

To correspond to Ricker's treatment, the logarithm of $\bar{N}(z)$ will be used as a description of catch curves. From Equation (4.5-1) we get

$$\ln N(z) = - \int_{z_R}^{z} \frac{g'(\tilde{z})+\mu(\tilde{z})}{g(\tilde{z})} \, d\tilde{z} + \ln \frac{R}{Tg(z_R)} \, . \qquad (4.6-1)$$

The slope of the logarithmic size distribution takes the simple form

$$\frac{d[\ln\bar{N}(z)]}{dz} = - \left[\frac{g'(z)+\mu(z)}{g(z)} \right]. \qquad (4.6-2)$$

An estimate of mortality is then given by

$$\mu(z) = -g(z) \frac{d[ln\bar{N}(z)]}{dz} -g'(z). \qquad (4.6-3)$$

Ricker is able to handle a size-frequency curve directly only if

he assumes growth, mortality, and the slope of the curve are all constant.

Letting growth and mortality rates be g and μ, respectively, and

$\frac{d[ln\bar{N}(z)]}{dz}$ =-i', Ricker derives the estimate for mortality,

$$\mu = i'g. \qquad (4.6-4)$$

See Sections 2B and 2G of Ricker's Handbook. This is clearly a special

case of Equation (4.6-3) with g and μ constant, and g'(z) = 0.

Ricker then states that Formula (4.6-4) is useful only for

segments of size frequency curves where growth is constant. To illustrate

this problem, he uses a model to generate size distributions (Ricker's

Figure 2.11) showing the "stacking up" phenomenon described in Chapters

2 and 3.

Ricker states that ". . .when the mortality rate is small and

fairly steady, and rate of increase in size is decreasing at a moderate

rate. . .," the size distribution increases in z[Ibid., p. 72]. This

is a qualitative description of our criterion that [g'(z)+μ(z)] < 0

for the distribution to be increasing in z (see Figure 3.1).

An example of "stacking up" in an actual fish population is

shown in Figure 4.7 which is a smoothed catch curve drawn by Hart [1932]

for whitefish in an Ontario lake. Since a fine-meshed net was used,

the curve is representative of the actual length distribution for fish

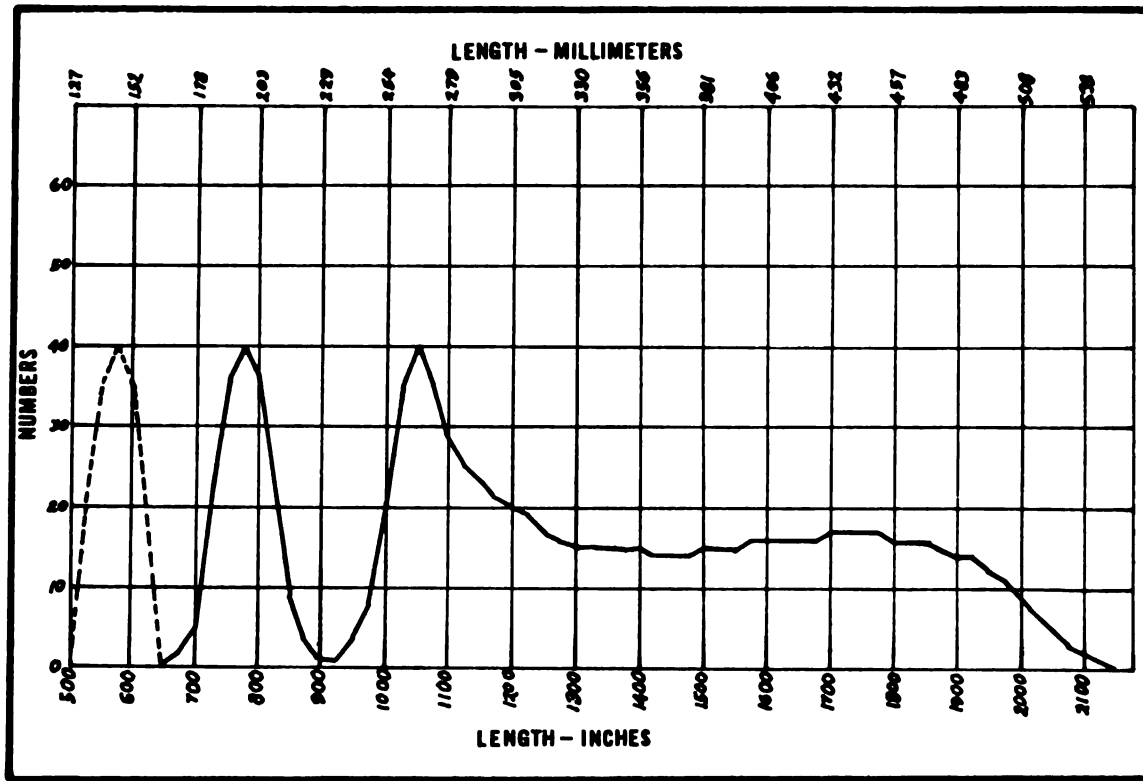longer than about 6 inches. The slight mode in the distribution at

Figure 4.7. Length-frequency curve for whitefish in Shakespeare Island Lake, Ontario, showing mode at 17-18 inches due to decreasing growth rate and low mortality in that length range. From Hart [1932].

17-18 inches is the result of fish "piling up", in Hart's words, due to low mortality and decreasing growth rates in this length range. This length-frequency curve is also a nice example of the coalescence of year classes in a size distribution due to decreased growth, as described in Section 4.3 and Figure 4.2.

Ricker points out that Formula (4.6-4) clearly will not work for a catch curve such as the one in Figure 4.7. For such a distribution, $\frac{d\ln\bar{N}(z)}{dz}$ is positive over a size interval. Using (4.6-4) one would conclude that there is a negative mortality rate over that size interval.

Equation (4.6-3) enables one to deal with changing rates of growth and sinuous catch curves to estimate size-specific mortalities.

Ricker also notes that mortality rates estimated using (4.6-4) ". . .always tend to be too small, if absolute rate of increase in length is decreasing with age." [Ibid., p. 73] Equation (4.6-3) explains why this is the case. If growth rate is decreasing with age/size, then $g'(z)$ is negative, and the term $[-g'(z)]$ is a positive quantity not accounted for in the approximation (4.6-4). Thus, mortality rates are underestimated by an amount roughly equal to $[-g'(z)]$, the absolute rate of decrease in the individual growth rate. This observation can refine mortality estimates based on size-frequency curves.

The various forms of $\bar{N}(z)$ given in this chapter provide a general mathematical description of size distributions in terms of growth and mortality. They should help in increasing the utility of size distributions, which are more easily obtained than age distributions, in estimating vital statistics of fish populations.

CHAPTER 5

THE REGULATION OF POPULATION GROWTH BY INDIVIDUAL GROWTH

5.1 Density-Dependence in Fish Populations

In earlier chapters, populations were modeled for which individual growth was time-independent, fixed presumably by the physiology of each organism as expressed by its physiological age. Many organisms, however, have a highly plastic growth response to changes in their environment. In particular, individual growth rates for some species are strongly affected by intraspecific competition for resources such as food or space. This phenomenon is perhaps most striking in plant populations. Harper [1971] points out that individual plant growth responds to competition so flexibly that biomass production per unit area remains virtually constant for many species over a wide range of number densities.

In this chapter, the p-age model (2.2-1), (2.2-7) will be used to study the dynamics of populations whose members respond to their limited environment by altering their growth rates. Both analysis and simulation will be used to uncover the behavior of the model when growth rate, g, is assumed to depend on some measure of population density.

As in Chapter 4, the discussion will use fish populations for a paradigm. A central theme of Weatherley's [1972] book is that individual growth response to competitive pressure plays a key role in regulating fish populations, and we will draw heavily on his observations.

A number of empirically based relationships between individual

growth and population density in fish have been suggested, all of which state a negative correlation between growth rate and total numbers or biomass. For example, Backiel and LeCren [1967] propose that $\frac{dg}{dD} \propto \frac{1}{D}$ describes the effect of density, D, on growth rate, g. This implies that variations in density at a low population level result in much greater changes in growth than do variations at a high density level. In contrast, from experiments in warm-water fish ponds, Hepher [1967] found a log-log relationship between g and D. Hepher's model states that a relative decrease in individual growth rate is a constant multiple of the relative increase in density.

Another more detailed connection between growth and density is proposed by Beverton and Holt [1957], who also give an excellent general discussion with numerous examples of density-dependent growth. Using the von Bertalanffy growth equation (see Section 4.3), they conclude, on both theoretical and empirical grounds, that the asymptotic weight limit, z_∞, decreases with increasing D, while the parameter K remains unaffected by density.

All of these models implicitly evaluate density relative to the carrying capacity of the environment, which will be denoted by C. Another advantage of discussing density-dependence in the context of fish populations is that managers of hatcheries and farm ponds have, for some time worked with a practical, quantitative definition of carrying capacity. Hepher [1967], for example, defines the carrying capacity as the maximum weight of fish which can be sustained by a pond. A population would be considered to be at the carrying capacity if it were composed of individuals having zero growth, and if it showed no net biomass production over some time interval.

To construct a more realistic model of density-dependent growth, a detailed picture of the dietary habits of a particular species would be necessary. One might divide the observed size range, $[z_0, z_\infty]$, of fish into subintervals to account for the switching of preferred diets shown by individuals of many species as they increase in size. Associated with each subinterval, j, would be a carrying capacity, C_j, based on the availability of prey items for fish in that size range. Growth of individuals in the subinterval, j, is then a function of the total numbers, D_j, relative to C_j. For example, the size range of bluegills in the experimental ponds of Hall, et al. [1970] could be divided into two intervals. Individuals less than about 35mm. in length fed largely on zooplankton, while individuals greater than 35mm. in length preyed predominantly on benthic insect larvae. Unfortunately, we can not, as yet, deal analytically with growth models of this complexity.

The relationships between density and the other vital rates, $\mu(z)$ and $b(z)$, must also be considered. Mortality in adult fish is generally felt to be density-independent for most species [Beverton and Holt, 1957; Weatherley, 1972]. However, some researchers see density-dependent mortality during larval stages as a prime component of natural population regulation [Backiel and LeCren, 1967]. The model used here deals with post-larval fish, so it will be assumed that density does not influence mortality.

The influence of density upon reproduction is still an open problem for fish. Most fish modelers do not attempt to follow the dynamics of hatching and larval mortality. Instead, stock-recruitment

models are used to relate the current density of fish to future expected recruitment of juveniles into the population at a given size or age [Ricker, 1958; Beverton and Holt, 1957]. These models show recruitment increasing with increasing population density, up to a certain point. At higher densities, recruitment begins to decrease, due to increasingly density-dependent egg and larval mortality.

In this chapter, it is assumed that recruitment of fish sized z_0 does not depend on density. That is, $b(z)$ will be the instantaneous rate of production of fish sized z_0, per breeding individual of size z. The size z_0 will be the size of the smallest post-larval fish in the population, since it is assumed that mortality in post-larval fish is density-independent.

With this definition of z_0, the rate $b(z)$ is no longer a true representation of fecundity, since fecundity is usually defined as the number of eggs laid by an individual [Pielou, 1969]. Since $b(z)$ stands for the rate of production of live offspring sized z_0, per individual of size z, it is more accurately a fertility rate, and it will be referred to as such for the remainder of the chapter. Keep in mind that, with these definitions of z_0 and $b(z)$, the time lag between the egg stage and the attainment of size z_0 is being ignored.

The seasonal component of reproduction will also be temporarily ignored in order to apply the model (2.2-1), (2.2-7) with time-invariant mortality and fertility rates.

In summary, then, we are studying a hypothetical fish population with the following characteristics:

i. The population is growing in a limited, but continuously

renewable, environment with carrying capacity C.

ii. Individual mortality rates, μ, and fertility rates, b, are time invariant, i.e., density-independent, but they may be functions of body size, z.

iii. Individual growth, g, is negatively correlated with population density.

5.2 Stability Theory

To understand growth as a regulatory mechanism, we will outline Weatherley's [1972] description of the growth of a fish population in a virgin environment, beginning with only a few colonizers. Figure 5.1, reproduced from Weatherley [1972], illustrates this process.

Initially, with abundant food and space, growth rates will be high. Fish mature early, and large numbers reproduce before being lost to mortality. As numbers and biomass increase, individual growth rate decreases in response to increasing competition. With decreasing growth rates, fewer individuals survive to reach a size at which they can reproduce, and the total population growth slows down. Eventually, total numbers and biomass approach an upper limit. At this population level, growth has become adjusted to permit a balance between replacement and loss of individuals.

The fate of the population size structure is not quite so clear. It would be expected that due to the integrative nature of the breeding interval, any initial size distribution in this population would evolve to a stable size distribution in the same fashion as the populations of Chapter 3 with fixed growth rates. Weatherley [1972] claims that a more complex size-structure will be seen as the population grows.
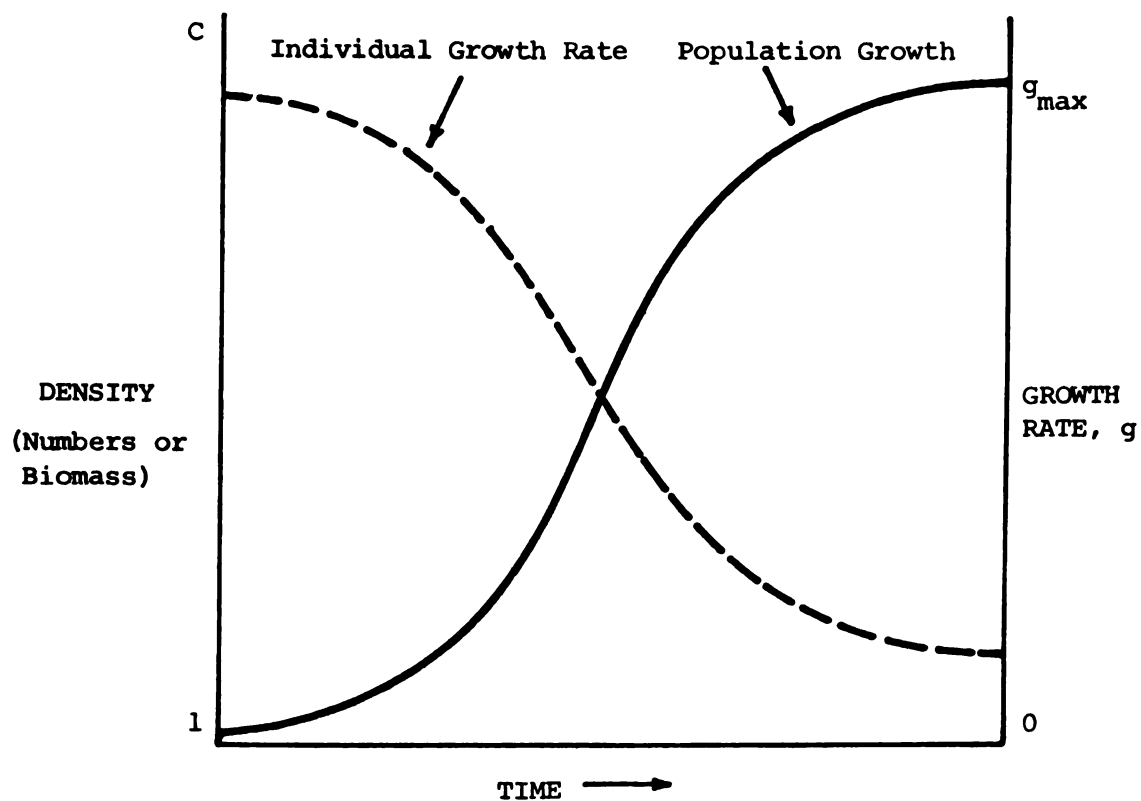
Figure 5.1. Growth of a fish population in a limited environment.
The population initially has only a few individuals.
From Weatherley [1972].

However, it is not immediately clear that the density-dependent growth rate could not resonate with an unstable size structure in such a manner as to produce persistent oscillations in population numbers about the upper asymptote of Figure 5.1.

If the size distribution did stabilize after several generations, the population would become stationary, with balanced birth and death rates, fixed total numbers and biomass, and unchanging individual life histories.

To model the population described above, a simple and realistic relationship between growth and density is assumed--namely that, at any size $\hat{z}$, the growth rate $g(\hat{z},D,C)$ is monotonically decreasing function of density, D. That is, growth rates are greatest when the individual has no competition for space or food, and they decrease with increasing D, approaching zero as $D \rightarrow C$. All three of the growth-density relationships proposed in Section 5.1 follow this general pattern.

To model the dynamics of a population using (2.2-1), (2.2-7) with a growth function such as the one described above, it is necessary to consider density as a state variable of the model. If, for example, D is equated to total population biomass, then Equation (4.2-2) for biomass must be solved along with (2.2-1). If D is measured by total population numbers, $N_t(t)$, then equations (2.2-5) and (2.2-7) can be combined to give the differential equation:

$$\frac{dN_t(t)}{dt} = \int_{z_b}^{z_b+\alpha} b(\tilde{z})N(\tilde{z},t)d\tilde{z} - \int_{z_0}^{\infty} \mu(\tilde{z})N(\tilde{z},t)d\tilde{z}. \qquad (5.2-1)$$

A useful tool for analyzing the effect of density-dependent growth on population dynamics is the net reproductive rate, R_0, defined by

Equation (3.3-4) with r = 0. Since the mortality and fertility parameters are assumed to be time-invariant, the net reproductive rate can be expressed as a function of D, which varies with time.

Using the definition of survivorship, (2.4-6), in Equation (3.4-3) the net reproductive rate can be written as

$$R_0(D,C) = \int_{z_b}^{z_b+\alpha} \frac{b(\tilde{z})}{g(\tilde{z},D,C)} \exp\left[-\int_{z_0}^{\tilde{z}} \frac{\mu(\hat{z})}{g(\hat{z},D,C)} d\hat{z}\right] d\tilde{z}. \qquad (5.2-2)$$

Recall that $R_0 > 1$ implies an increasing (in total numbers) population, while $R_0 < 1$ implies a decreasing population.

An important feature of the net reproductive rate is that its definition and implications for population growth over the long term are completely independent of the status of the c-age or size distribution. Equation (3.3-4) merely expresses the relationship between R_0 and the characteristic equation of a stable population. Thus, Equation (5.2-2) can be used to predict population growth or decay as a function of growth, density, mortality and fertility, without regard to the form of the number density $N(z,t)$.

Suppose for the moment that a population is stationary--individual growth and density have mutually adjusted so that $R_0(D,C) = 1$. What will be the result of a perturbation in D, either through the removal or addition of individuals? Will the population return to an equilibrium state? One would intuitively expect it to do so, since increased density results in decreased growth, which in turn should depress the birth rate, thus lowering the density. Likewise, a decrease in D should speed up growth--the resulting increase in reproduction should force density to increase once again.

Upon differentiating (5.2-2) with respect to D, however, it can be seen that it is possible for R_0 to actually increase with increasing density. A closer examination of (5.2-2) and the definition of the fertility rate, b(z), reveals the problem. In addition to the negative feedback loop between R_0 and density described in the previous paragraph, there is a positive feedback loop operating through the instantaneous birth rate.

To see this, consider the definition of R_0 on page 32 , and set $\ell(z,z_0) = 1$. The quantity

$$R_0^* = \int_{z_b}^{z_b+\alpha} \frac{b(\tilde{z})}{g(\tilde{z},D,C)} \, d\tilde{z} \qquad (5.2-3)$$

must be the total number of offspring produced by each individual which survives to grow all the way through the breeding interval. This quantity is clearly a function of growth, g. Although the rate of fertility, b(z), remains constant, a low growth rate means that individuals remain in the breeding interval for a longer time, and the total fertility, R_0^* , is increased. An increase in growth rate similarly implies a decrease in total fertility, since individuals now reproduce at a constant rate for a shorter period of time.

Apparently, reproduction, in the sense of total fertility, has not really been uncoupled from density. This result also suggests that the integral Equation (2.2-3) for total birth rate B(t) is, in most cases, a poor model for reproduction when it is used for a population with time-varying growth rates but time-invariant fertility rates. Such a model implies that slower-growing individuals produce more offspring

than faster-growing ones. Species of fish are not likely to be seen with this type of reproductive feedback.

This reproductive model provides a positive feedback on population density. As density increases, growth decreases and total fertility increases, thus increasing density still further. The opposite occurs if density decreases. The population will have a stable equilibrium only if the negative feedback effects of altered survivorship up to the breeding interval can more than compensate for the positive feedback effect of density on total fertility.

A much more realistic model is obtained if we allow the fertility rate, $b(z)$, to vary with density, but assume that total fertility, R_0^*, is constant. This can be done by using a fertility function of the form

$$b(z,D,C) = \hat{b}(z)\ g(z,D,C). \qquad\qquad (5.2-4)$$

This model says that slower-growing individuals produce offspring at a reduced rate compared to faster-growing ones. The net result is that the total number of offspring expected from any individual which survives through the breeding interval is constant, regardless of growth rates. Now the total fertility is given by

$$R_0^* = \int_{z_b}^{z_b+\alpha} \hat{b}(\tilde{z})\,d\tilde{z},$$

which does not depend on growth. This reproductive model, with constant total fertility, will be called model **B**. The original model, with time-invariant fertility rates, will be referred to as model **A**.

The net reproductive rate for model **B** takes the form

$$R_0(D,C) = \int_{z_b}^{z_b+\alpha} \hat{b}(\tilde{z}) \exp\left[-\int_{z_0}^{\tilde{z}} \frac{\mu(\hat{z})}{g(\hat{z},D,C)} d\hat{z}\right] d\tilde{z}. \quad (5.2-5)$$

The derivative of (5.2-5) is

$$\frac{dR_0}{dD} = \int_{z_b}^{z_b+\alpha} \hat{b}(\tilde{z}) \left[\int_{z_0}^{\tilde{z}} \frac{\mu(\hat{z})}{g^2(\hat{z},D,C)} \left(\frac{\partial g(\hat{z},D,C)}{\partial D}\right) d\hat{z}\right] \cdot$$

$$\exp\left(-\int_{z_0}^{\tilde{z}} \frac{\mu(\hat{z})}{g(\hat{z},D,C)} d\hat{z}\right) d\tilde{z}. \quad (5.2-6)$$

Since growth is assumed to be a decreasing function of density, the quantity $\frac{\partial g}{\partial D}$ is negative for all $\hat{z}$ and D. All other terms in (5.2-6) are always positive, so the derivative $\frac{dR_0}{dD}$ is strictly less than zero for all D.

Thus, the function R_0 has the properties shown in Figure 5.2. The value D* satisfying $R_0(D^*,C) = 1$ is a stable equilibrium point for population density. If D is perturbed away from D*, the net reproductive rate will automatically adjust via the growth rate to oppose the perturbation. Since R_0 is a monotonic function of D, the equilibrium density is unique.

The above analysis is independent of the population size structure, so there is still no mathematical guarantee that some combinations of the vital rate functions $\hat{b}$, μ, and g can not produce persistent oscillations in the size distribution. However, the negative feedback loop described above will act to oppose any changes in D which occur as the size structure fluctuates. It is difficult to imagine a set of vital rates in (2.2-1), (2.2-7) which would allow a continuously changing size
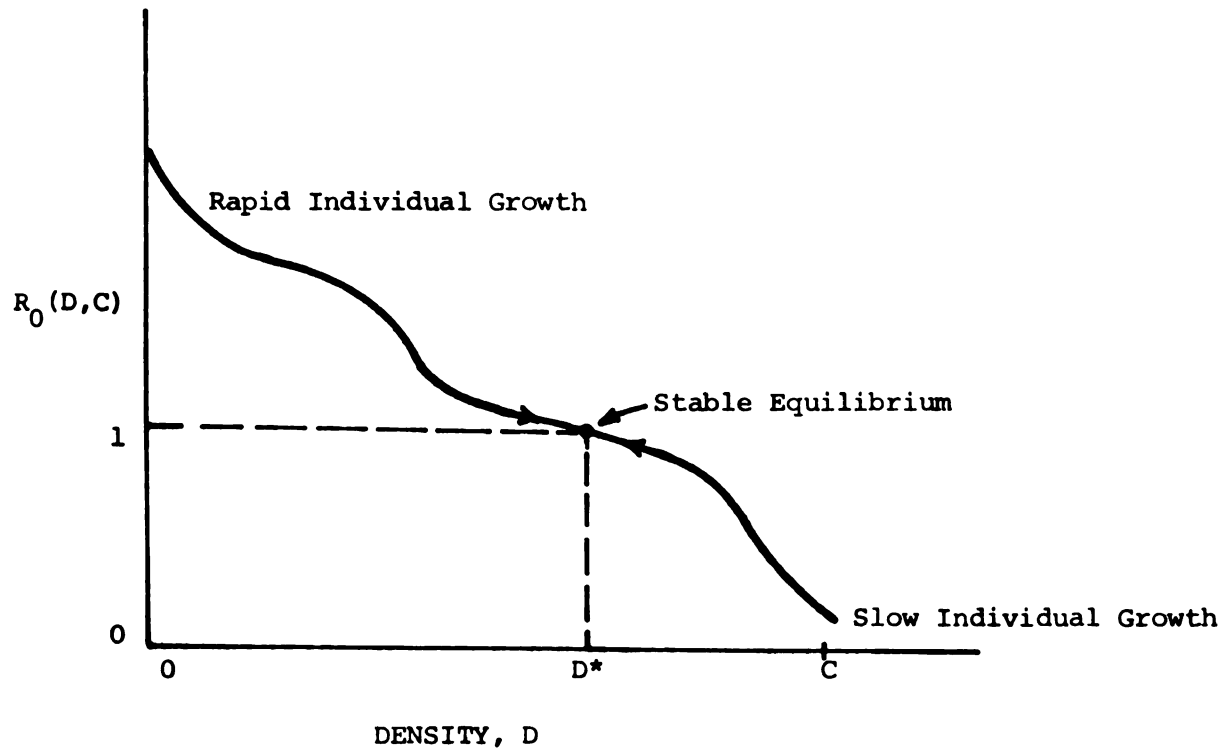
Figure 5.2. Net reproductive rate versus density for reproductive model **B**.

distribution while keeping total population numbers or biomass constant at D*. This is a further indication that the size structure in this feedback model will ultimately stabilize.

Two other features of this density-limited form of the model are worth noting. Figure 5.2 and Equation (5.2-5) suggest that the model population density may stabilize well below the carrying capacity, C. The actual value of D* at which the population stabilizes is determined by the vital parameters of the particular species being considered, as expressed by the equation $R_0(D^*,C) = 1$. In contrast, the familiar logistic equation,

$$\frac{dN}{dt} = rN(1 - \frac{N}{K}),$$

predicts that a population will saturate its environment and will only stabilize when density, N, is equal to carrying capacity, K.

A second feature of the density-dependent p-age model is its high degree of structural stability. This means that the system is stable with respect to perturbations of its parameters [Rosen, 1970]. The requirement for any initial population to eventually stabilize at a unique, non-zero density $D^* \leq C$ is simply that the population can grow successfully in an unlimited environment. In other words, a necessary and sufficient condition for the population density to be stable is that the following inequality be satisfied:

$$R_0(0,C) = \int_{z_b}^{z_b+\alpha} \hat{b}(\tilde{z}) \exp\left[- \int_{z_0}^{\tilde{z}} \frac{\mu(\hat{z})}{g(\hat{z},0,C)} d\hat{z}\right] d\tilde{z} \geq 1 \qquad (5.2-7)$$

All sets of vital functions $\hat{b}$, g, and μ which satisfy (5.2-7)

as a strict equality comprise the region of parameter space for which the model is structurally unstable. This is a relatively small region. For most vital rate functions, a small perturbation will not result in a change of the inequality sign in (5.2-7). Unlike a density perturbation, however, a perturbation of a vital rate in a stable population will result in a new equilibrium value, D*, toward which population density will tend.

According to this theory, a stable population with very labile individual growth can respond successfully to enormous increases in total fertility and/or decreases in mortality rate. An example of this is seen in Weatherley's [1972] discussion of H. S. Swingle's experiments with bluegill sunfish populations in farm ponds. Weatherley describes these populations, which are subject to very low mortality, as being comprised of individuals which have virtually stopped growing. There are a large number of juveniles and very few breeding adults. These "stunted" populations remained stationary for several years.

The theoretical arguments of this section show how a labile individual growth rate is a powerful regulator of population growth in a model in which total individual fertility remains constant (model B). The remainder of this chapter will use simulations to examine the manner in which a growth-regulated population approaches its equilibrium density.

5.3 Simulation--Goals and Methods

The stability analysis of the last section reveals the asymptotic behavior of the system (2.2-1), (2.2-7) when growth is density-dependent.

There are a number of questions, however, which can be asked about the model dynamics for smaller values of t.

The first question which comes to mind is whether the model population has similar trajectories for density and growth to those described at the beginning of Section 5.2 for fish populations. We also would like to settle the question of the fate of the size structure. Does it stabilize, and, if so, how rapidly? How does its stabilization depend on initial conditions and on vital parameters?

Another interesting problem has to do with the measure of density to which growth responds in controlling the population. Will the population respond differently if D is measured in units of biomass rather than numbers? These are questions which can best be answered by simulation based on the model.

Simulation experiments for the system (2.2-1), (2.2-7) are difficult to design. The system has several parameters, some of which are functions of body size. As a first step in reducing the parameter space to a manageable size, this functional dependence was removed. Growth, fertility, and mortality were made constant with respect to z.

The growth-density relationship was assumed to be as simple as possible. Growth was defined by

$$g(D,C) = g_{max} \left(1 - \frac{D}{C}\right). \qquad (5.3-1)$$

With this growth function, individuals grow at the rate g_{max} when no competitors are present, and growth decreases linearly to zero as the carrying capacity is approached. This is also the relationship between density and growth implied by Figure 5.1.

These simplifying assumptions result in a seven-dimensional parameter space, not including the initial condition, $N_I(z)$. The parameters are g_{max}, μ, $\hat{b}$, C, z_b, α, and z_0. Once the simulation model was operating correctly, the parameters could be checked one at a time for their influence on system dynamics.

To further restrict the parameter set, an effort was made to select values in the same range as those generally used for fish populations. The variable z represented body weight, and z_0 was set equal to 1 in arbitrary weight units, while the upper limit for weight was taken to be 21 units. The simulation time scale and g_{max} were mutually set so that an individual growing in an unlimited environment would reach the maximum size in about 4 years. The size of first breeding, z_b, was taken to be 1/2 to 3/4 of the maximum size reached, and breeding was, for most runs, allowed to continue up to the maximum size.

The mortality rate, μ, and fertility rate, $\hat{b}$, were the parameters most frequently altered to produce different trajectories. Mortality ranged between 50 percent and 90 percent loss per year, while total fertilities spanned a range of 10 to 2,000 offspring per individual. These values are all similar to the ones employed by Weatherley in his models of fish population growth and maintenance [Weatherly, 1972, Ch. 6].

Some runs of the simulation were made with the reproduction model A of Section 5.2, but this model proved to be stable only within a narrow range of parameter values. This was due to the positive feedback effect of density on total fertility which was described in Section 5.2. All of the runs reported in Section 5.4 employ the reproduction

model $\underline{B}$ with its more realistic assumption of constant total fertility. This model showed stable behavior for virtually any combination of fertility and mortality rates satisfying the inequality (5.2-7).

There was one set of conditions, however, under which the simulation could "blow up." As developed here, the system cannot deal with the growth function (5.3-1) if the population density overshoots the carrying capacity. If this happens, then D > C, and growth, according to (5.3-1) is negative. To handle this problem, it was necessary to artificially set both growth and reproduction equal to zero whenever D > C. The simulation continued to run until mortality reduced the density below the carrying capacity, at which time reproduction and growth began again.

The numerical techniques used for solution of the system (2.2-1), (2.2-7) are discussed in Appendix A.

5.4 Simulation Results

Figure 5.3 shows the results of a typical simulation run. Both numbers and biomass follow a generally sigmoidal path to steady-state values. This model population initially had 10 individuals, all weighing between one and two units.

Oscillations about the sigmoidal curves are due to the size structure of the model population. The peaks in the oscillations are separated by one "generation", where a generation is defined as the time required to reach the size, z_b, of first reproduction. Thus, as the initial cohort moves through the breeding interval, it generates a pulse of young, producing a peak in total numbers. A corresponding
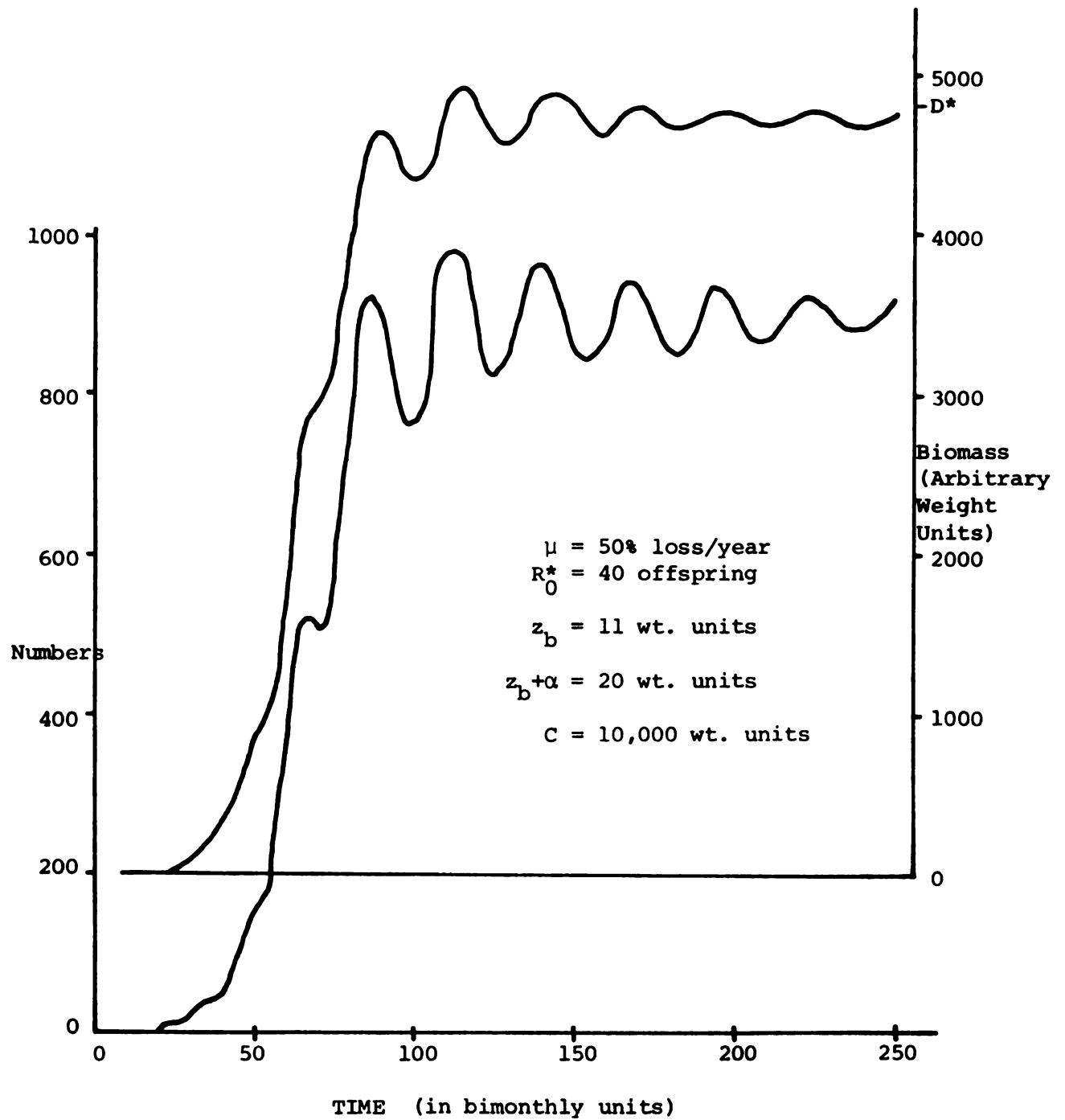
Figure 5.3.   Sample trajectories of model population growth.

peak in biomass occurs in few time units later, as individual size increases while numbers decrease due to mortality. Numbers and biomass begin to decrease rapidly, as mortality depletes both the new cohort and the remnant of the reproducing initial population, and they reach a minimum just before the new cohort reaches size z_b. When the new cohort grows to size z_b, a new reproductive pulse is produced, a generation later than the previous pulse, and the cycle begins again.

Two other dynamics of the population can be observed. As time increases, the integrative nature of the breeding interval broadens successive cohorts, so that the size structure eventually stabilizes. This action accounts for the damping of the oscillations in Figure 5.3. At the same time, the total density increases and growth rate simultaneously decreases. By the time density, which is equivalent to total biomass for the population in Figure 5.3, has stabilized at D*, the individual growth rate is only half as great as in the small initial population.

This general pattern of population growth was followed by nearly all the simulation runs made. Two variations on this theme are sketched in Figure 5.4. Figure 5.4a shows total population numbers for a run with a low value for $\hat{b}$ relative to μ and a long breeding interval. The population size structure loses the traveling wave of the reproduced initial condition within two generations. Since the difference between fertility and mortality rates is small, the population takes longer to reach its equilibrium density.

Figure 5.4b is a very different case. In this model population, individuals have very high total fertility and high mortality rates
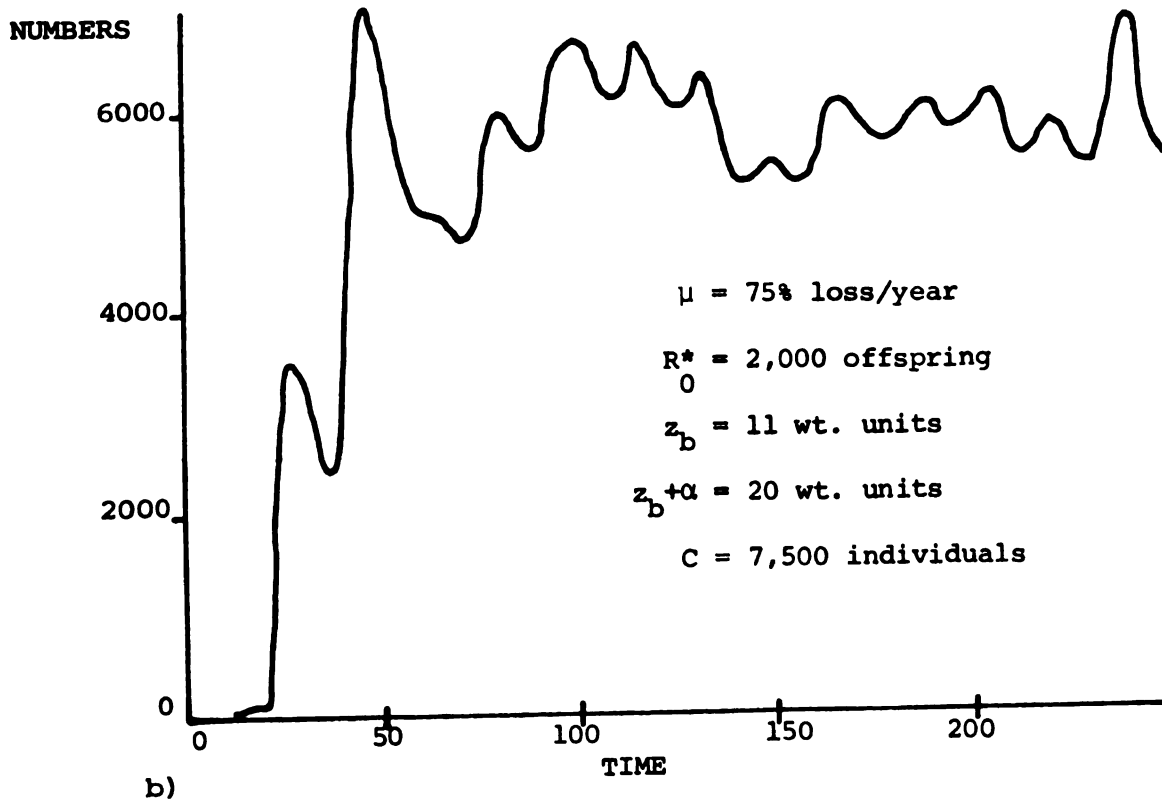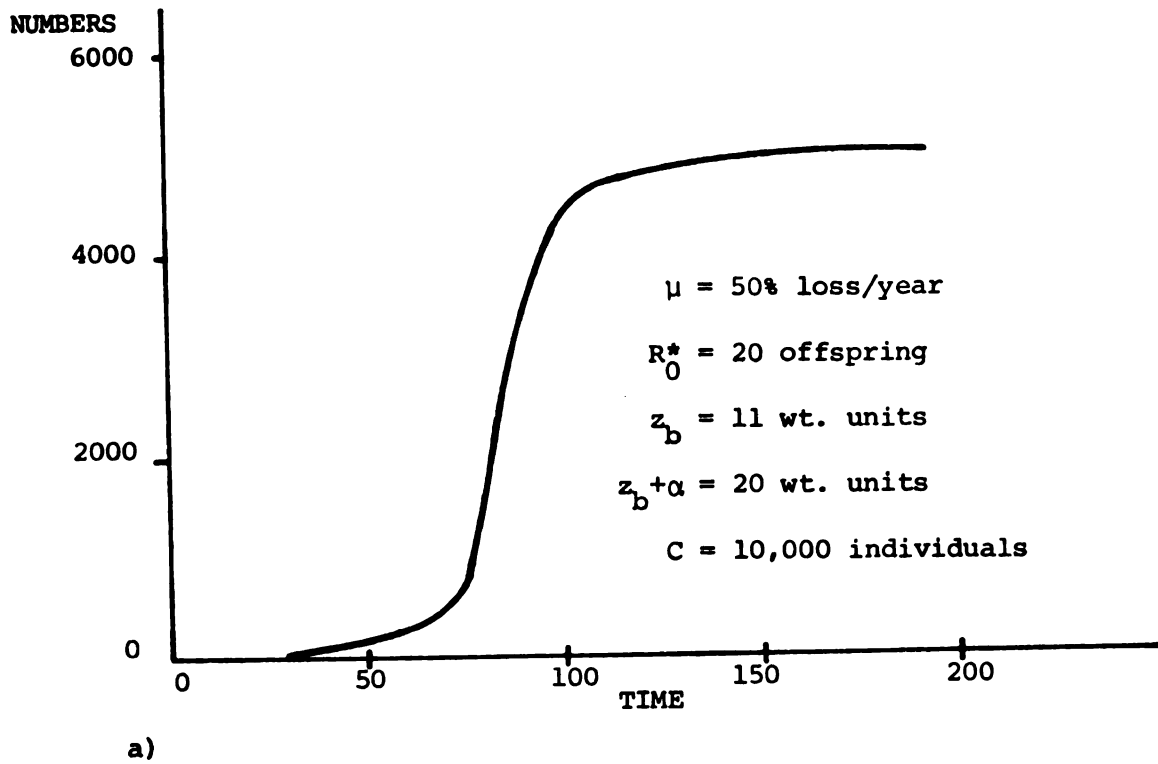
Figure 5.4. Sample trajectories for model populations.

compared with the populations of Figures 5.3 and 5.4a. Oscillations in total numbers and biomass showed no sign of abatement when the simulation was run for 14 generations. Note, however, that population density is not out of control, in the sense that D does not approach either zero or C.

As Figure 5.4a illustrates most clearly, the pattern of population growth shown in the simulations is identical to the dynamics described at the beginning of Section 5.2 and in Figure 5.1. The model also corroborates other qualitative descriptions by Weatherley of fish population dynamics.

For example, Weatherley [1972] states that, as the population density increases towards its asymptotic value, the size structure of the population should contain an increasingly larger proportion of smaller individuals due to declining growth rates. This feature is clearly seen in the model populations such as the one in Figure 5.4a where the population comes to contain a full spectrum of individual sizes early in its growth. As the population numbers and biomass climb the sigmoid curve in Figure 5.4a, the mean size of individuals in the population decreases.

The theory of Section 5.2, along with supporting evidence from simulation runs, also provides an answer to one of Weatherley's questions about carrying capacity. In describing the expected effects of a sudden improvement in food or living space for a steady-state population, Weatherley [1972, p. 160] wonders whether the population, after increasing to a new higher density, would regain the same c-age structure it possessed at the old, lower density.

According to Section 5.2, the answer to this question is "yes", provided that density does not affect mortality or total fertility, and that growth depends on relative density, i.e.,

$$g(z,D,C) = g(z,\frac{D}{C})$$
(5.4-1)

To see this, recall that the density of a stationary population satisfies the equation $R_0(D^*,C) = 1$, for the net reproductive rate. When relative density is considered, this equation reads $R_0[(D/C)^*] = 1$. For reproduction model B, this solution is unique. By the stability analysis of Section 5.2, any increase in C for a stationary population would force D to adjust to maintain the ratio $(D/C)^*$. The population would again become stationary at the higher absolute density, and growth rates would return to their previous steady-state values, $g(z,(D/C)^*)$.

According to Equation (3.4-4), the shape of the stationary size distribution depends solely on the vital rates, so it too would return to its original configuration, as would the c-age distribution.

In Equation (5.3-1), growth depends on relative density. Using this growth function, the simulation runs showed the behavior described above, regardless of whether reproduction model A or B was used. The carrying capacity, C, could be set at any value, and, if the other parameters remained unchanged, the population would always stabilize with the same size structure and at the same relative density.

Weatherley observes these dynamics in fish. He writes:

> It is difficult to avoid the impression that growth flexibility
> is an adaptive mechanism by means of which fish populations
> tend always towards the same population age/size structure
> and the same individual growth rate, no matter what the nutri-
> tional status of their environment. [Weatherley, 1972, p. 172]

The theory of Section 5.2 and the simulations are evidence that any perturbations in the stationary density and/or size structure of these model populations will be damped out. The return of the population to its original stationary state will be characterized by damped oscillations in total density. If a perturbation of a vital parameter occurs, the same stabilizing behavior will be seen, although the new stationary state of the population may be quite different from the old one.

These damped oscillations in density suggest a measure of the "resilience" [May, 1973] of the population in response to perturbations. Borrowing from the vocabulary of control systems theory, we will define the <u>settling time</u> of a population as the number of generations required for D to settle within 10 percent of its steady-state value, D^*, following a perturbation. Since the oscillations in D are due primarily to a fluctuating size structure, the settling time is really a measure of how long it takes the size structure to stabilize.

In the simulations, D was measured either by total numbers or biomass. Using Equation (5.2-5) for net reproductive rate and the steady-state versions of Equations (5.2-1) and (4.2-2) for numbers and biomass, one can easily predict what the steady-state values of numbers, biomass, birth rate, and growth rate must be for any given set of parameters, z_0, z_b, α, μ, δ, g_{max}, and C. Each simulation run computed settling times by continuously comparing current system states with their predicted steady-state values.

Table 5.1 summarizes three features of the model populations with respect to changes in life history parameters. The table shows

how a perturbation of a single parameter in a stationary population alters

the ultimate relative density, (D/C)*, mean size of individuals, $\bar{z}$, and

settling time of the new stationary population.

TABLE 5.1

Model Population Features as a
Function of Life History Parameters

	b	α	z_b	μ
(D/C)*	+	+	−	−
$\bar{z}$	−	−	+	+
Settling Time	+	−	+	+

Plus signs indicate that the parameter and the population feature

both increase or both decrease, while a minus sign means that the para-

meter and the feature have an inverse relationship.

Many of the entries in Table 5.1 can be predicted from the theory,

and all were corroborated by simulation runs. The relationships shown

in the table generally conform to our intuitions about the life-histories

of individuals in limited environments. It is to be expected, for example,

that an increase in the width of the breeding interval, α, would result

in an increased birth rate and thus an increased stationary density, (D/C)*.

From the point of view of growth, the entries for $\bar{z}$ may seem somewhat

redundant in that they each have an opposite sign with respect to cor-

responding entries for (D/C)*. Any increase in (D/C)* means a reduced

growth rate, and thus, a smaller mean individual size.

Of greater interest, perhaps, are the relationships between

various parameters and settling time. The table implies, for example, that a stationary population with very high fertility and mortality rates would be rarely seen in the real world. Any slight perturbation in the size structure of such a population, due perhaps to its environment, would result in traveling waves in the size structure and oscillations in total density which would take a long time to damp out. This is the situation seen in the model population of Figure 5.4b, which has a settling time greater than 14 generations.

A representative sample of settling times for various model populations is shown in Table 5.2. The runs are listed in the table in pairs. The model populations of each pair are identical in their parameter sets and initial conditions. They differ only in the measure of density which was used in (5.3-1) to control growth rate. One population of each pair has a growth response to biomass while growth in the other population responds to total numbers.

Two characteristics of this table are of special interest. The first is that, for the runs reported, the settling time for total population biomass was less than that for total numbers. This was, in fact, a feature of all the runs, and, as the table shows, settling times for biomass were shorter regardless of whether individual growth was controlled by numbers or biomass.

Secondly, a comparison of settling times within each pair reveals that, with the exception of pair B, the settling times for both numbers and biomass were shorter when D was measured by numbers as opposed to biomass. This relationship was true in six out of the seven paired runs which were made.

TABLE 5.2

Settling Times for Representative Model Populations.
Mortality Rate of .116 Results in 50% Loss/Yr., While μ = .231
Results in 75% Loss/Yr. Settling Times are Measured in Generations.

Model Population Parameters	Measure of D	Settling Time for Numbers	Settling Time for Biomass
A| b = 45, z_b = 11,	Numbers	3.8	2.2
z_b+α = 20, μ = .231	Biomass	4.2	3.0
B| b = 10, z_b = 11,	Numbers	1.3	1.2
z_b+α = 20, μ = .116	Biomass	1.8	1.1
C| b = 10, z_b = 14	Numbers	7.3	3.4
z_b+α = 20, μ = .116	Biomass	10.0	4.9
D| b = 5, z_b = 14	Numbers	7.0	4.0
z_b+α = 20, μ = .116	Biomass	10.4	4.2

Taken together, these results suggest some differences between model populations in which individual growth, and thus population growth, are controlled by biomass rather than numbers. Total biomass responds more sluggishly than numbers to fluctuations in the size structure which are induced by reproductive pulses. A reproductive pulse causes an immediate sharp change in numbers, but its effect on biomass is delayed, and thus reduced by mortality. Consequently, biomass fluctuations damp out more quickly than fluctuations in numbers as the size structure stabilizes.

This relative insensitivity of biomass to fluctuating size distributions also explains why it is a "sloppier" controlling variable than numbers, in the sense that populations controlled by biomass usually have longer settling times.

These conclusions suggest a broader generalization; populations regulated by biomass, as opposed to numbers, will allow greater fluctuations in numbers, especially of young individuals, before responding with altered individual growth rates.

5.5 Discussion

The model populations of the last section follow the same trajectories for growth, total numbers, and size structure as are seen in real fish populations. They may also be compared with studies of other populations having density-dependent vital parameters.

Previous simulation studies of age-structured populations have considered mortality and/or fertility to be functions of population density. The p-age model discussed here allows for the third vital parameter, growth rate, to be influenced by population size. In addition, it compares density with a carrying capacity to regulate population growth, a feature not found in other distributed parameter models.

A brief review of these models and comparisons with the trajectories shown here reveal a common pattern of growth in total numbers, regardless of which vital rates are made density-dependent. Leslie [1948] and Pennycuick, et al. [1968] show how total numbers in matrix model populations grow sigmoidally, settling down to a steady-state through damped oscillations.

Motivated by laboratory populations such as Nicholson's blowflies [Nicholson, 1957] and Frank's Daphnia cultures [Frank, 1960], several modelers have been especially interested in the oscillatory nature of the numbers of biomass trajectory. We will briefly outline their efforts and discuss the origins of oscillations seen in p-age and c-age

simulations. The most important feature of density-dependent simulations, however, is that oscillations have a bounded amplitude.

Several investigators have induced persistent oscillations in age structured models by explicitly employing time lags in the negative feedback loops between density and the regulated vital parameters [Leslie, 1959; Frank, 1960; Pennycuick, et al., 1968].

Fluctuations can also be produced by assuming that density affects mortality or fertility on a c-age-specific basis. If this assumption is coupled with c-age-specific rates which are very sensitive to small changes over a short range of densities, stable oscillations in total density will result [Usher, 1972]. Williamson [1974] carries this sensitivity to an extreme by using one of two different sets of vital parameters depending on the relation of density to some threshold, and he too obtains limit cycle behavior.

In these systems as well, time lags give rise to the oscillations. These lags are not explicit, however. Because of their age structures, the models have inherent time delays. For example, an increase in mortality rates for juveniles in the population will affect the total birth rate only after a maturation delay.

A third method of producing persistent oscillations is to use extreme values for mortality and fertility in model populations. This effect is seen in Figure 5.4b and is also found in a simulation from Pennycuick, et al. [1968] in which a very high fecundity rate was used. There is probably a time lag operating here, as well, although its exact nature is unclear. The density-regulating factors apparently cannot respond quickly enough to large fluctuations in population

numbers to allow c-age or size structures to stabilize. More work on this is needed.

As a first step, we intend in future work to define settling times for populations with time-invariant values of g, b, and μ. In such populations, the numbers or biomass trajectory would "settle" down to trace an exponential curve of increase or decrease, as predicted by the theory of Chapters 2 and 3. These model populations will be compared with density-regulated populations. If there is a time delay hidden in the density feedback mechanism, then settling times for the regulated populations should be longer than those for unregulated populations.

Fluctuations in size structure, density, and vital rates in real populations are the result of environmental perturbations, as well as time lags. Whatever their nature, these fluctuations are kept under control in our model populations, as in many fish populations, by a highly flexible individual growth response. Those fluctuations which are not damped completely, as in Figure 5.4b, tend to be centered about a density D^* which is considerably less than C. Hence, minor perturbations of the population are not likely to result in an overshooting of the carrying capacity. This is an attractive alternative to the steady-state "saturation" of the environment implied by the density-carrying capacity relationship of the logistic equation.

A good deal of territory remains to be explored with the simulation model. For example, it would be useful to quantify the relationships in Table 5.1. Lefkovitch [1971] began a study of this sort when he ranked various forms of age-specific fecundity and mortality with respect

to settling times for c-age distributions in unregulated populations. His simulations indicate the large number of runs which would be necessary to quantify Table 5.1. Perhaps a formal sensitivity analysis using the equation for net reproductive rate would be more fruitful. The results would be well worth the effort. As May [1973] points out, parameters such as mortality rates and carrying capacities are much more likely to be perturbed in natural populations than are size distributions or number densities.

The most interesting and realistic simulation dynamics, however, are likely to be seen when growth is made a function of p-age as well as population density. The logical starting point at this next level of model complexity is Beverton and Holt's density-dependent form of the von Bertalanffy growth equation for fish populations (cf. Section 5.1). This avenue of research would provide a natural culmination to the work of Chapters 3, 4, and 5.

In the future, most of our work with the distributed parameter system will continue to focus on the phenomenon of individual growth and its influence on populations. This is where the strength and novelty of the physiological age model lies. For many populations, notably fish, growth is a life-history parameter of equal importance to fertility and mortality in determining population trajectories. We feel that this model will be a useful tool for exploring the dynamics of these populations.

BIBLIOGRAPHY

BIBLIOGRAPHY

Ames, W. F. 1969. <u>Numerical Methods for Partial Differential Equations</u>. New York: Barnes and Noble.

Auslander, D. M., Oster, G., and Huffaker, C. B. 1974. "Dynamics of Interacting Populations," <u>J. Franklin Institute</u>, 297, No. 5: 345-376.

Backiel, T. and LeCren, E. D. 1967. "Some Density Relationships for Fish Population Parameters." In <u>The Biological Basis of Freshwater Fish Production</u>. Edited by S. D. Gerking. New York: Wiley.

Bagenal, T. B. 1967. "A Short Review of Fish Fecundity." In <u>The Biological Basis of Freshwater Fish Production</u>. Edited by S. D. Gerking. New York: Wiley.

Bailey, N. T. J. 1964. <u>The Elements of Stochastic Processes with Applications to the Natural Sciences</u>. New York: Wiley.

Baranov, T. I. 1918. "On the Question of the Biological Basis of Fisheries." <u>Rep. Div. Fish Management and Scientific Study of the Fishing Industry</u>, I:81-128. Moscow.

Bell, G. I. and Anderson, E. C. 1967. "A Mathematical Model with Applications to Cell Volume Distributions in Mammalian Suspension Cultures," <u>Biophysical Journal</u>, 7:329-351.

Bellman, R. E. and Cooke, K. L. 1963. <u>Differential-Difference Equations</u>. New York: Academic Press.

von Bertalanffy, L. 1957. "Quantitative Laws in Metabolism and Growth," <u>Quarterly Rev. Biology</u>, 32, No. 3:217-231.

_____. 1968. <u>General System Theory</u>. New York: George Braziller.

Beverton, R. J. H. 1954. "Notes on the Use of Theoretical Models in the Study of the Dynamics of Fish Populations." Misc. Cont. #2, U.S. Fishery Lab., Beaufort, North Carolina.

_____ and Holt, S. J. 1957. <u>On the Dynamics of Exploited Fish Populations</u>. London: Her Majesty's Stationery Office.

Billups, C., Wilkins, B. and Pike, R. W. 1971. "Application of Population Balance Models in the Prediction of Shrimp Size Distributions." Coastal Studies Bulletin #6, Special Sea Grant Issue, Center for Wetland Resources, Louisiana State University, Baton Rouge, Louisiana.

Boling, R. H., Jr. 1973. "Toward State-Space Models for Biological Populations," Journal Theoretical Biology, 40:485-506.

Chapman, D. W. 1967. "Production in Fish Populations." In The Biological Basis of Freshwater Fish Production. Edited by S. D. Gerking. New York: Wiley.

Coulman, G. A., Reice, S. R. and Tummala, R. 1972. "A Systems Analytic Approach to Single Species Population Dynamics," Science, 175:518-521.

Courant, R. and Hilbert, D. 1966. Methods of Mathematical Physics, Vol. II. New York: Wiley.

Cull, P. and Vogt, A. 1974. "The Periodic Limit for the Leslie Model," Math. Biosciences, 21:39-54.

deBont, A. F. 1967. "Some Aspects of Age and Growth of Fish in Temperate and Tropical Waters." In The Biological Basis of Freshwater Fish Production. Edited by S. D. Gerking. New York: Wiley.

Frank, P. W. 1960. "Prediction of Population Growth Form in Daphnia Pulex Cultures," American Naturalist, 44:357-372.

Gulland, J. A. 1962. "The Application of Mathematical Models to Fish Populations." In The Exploitation of Natural Animal Populations. Edited by LeCren, E. D. and Holdgate, M. W.

Hall, D. J., Cooper, W. E. and Werner, E. E. 1970. "An Experimental Approach to the Production Dynamics and Structure of Freshwater Animal Communities," Limnology and Oceanography, 15, No. 6:839-928.

Harper, J. L. 1971. "The Regulation of Numbers and Mass in Plant Populations." In Population Biology and Evolution. Edited by R. C. Lewontin. Syracuse, New York: Syracuse University Press.

Hart, J. L. 1932. "Statistics of the Whitefish (Coregonus clupeaformis) Population of Shakespeare Island Lake, Ontario." University of Toronto Studies, Biol. Ser. #36.

Hepher, B. 1967. "Some Biological Aspects of Warm-Water Fish Pond Management." In The Biological Basis of Freshwater Fish Production. Edited by S. D. Gerking. New York: Wiley.

Holt, S. J. 1962. "The Application of Comparative Population Studies to Fisheries Biology--An Exploration." In The Exploitation of Natural Animal Populations. Edited by E. D. LeCren and M. W. Holdgate.

Hughes, R. D. 1962. "A Method of Estimating the Effects of Mortality on Aphid Populations," J. Anim. Ecol., 31:389-396.

Keyfitz, N. 1968. Introduction to the Mathematics of Populations. Reading, Mass.: Addison-Wesley.

Kharkar, A. N. 1973. "A Stochastic Approach to Population Balance Models." Ph.D. dissertation, Department of Chemical Engineering, Michigan State University, East Lansing, Michigan.

_____, Coulman, G. A. and Barr, R. O. 1974. "Population Models in Ecological Systems: A Systems Perspective." (To be published).

Langhaar, H. L. 1972. "General Population Theory in the Age-Time Continuum," J. Franklin Institute, 293:199-214.

Lee, K. Y., Barr, R. O., Gage, S. and Kharkar, A. N. 1975. "Formulation of a Mathematical Model for Insect Pest Ecosystems--The Cereal Leaf Beetle Problem," J. Theor. Biol. (in press).

Lefkovitch, L. P. 1965. "The Study of Population Growth in Organisms Grouped by States," Biometrics, 21:1-18.

_____. 1971. "Some Comments on the Invariants of Population Growth." In Sampling and Modeling Biological Populations and Population Dynamics. Edited by G. P. Patil, E. C. Pielou and W. E. Waters. University Park, Penn.: Penn. State University Press.

Leslie, P. H. 1945. "The Use of Matrices in Certain Population Mathematics," Biometrika, 33:183-212.

_____. 1948. "Some Further Notes on the Use of Matrices in Population Mathematics," Biometrika, 35:213-245.

_____. 1959. "The Properties of a Certain Lag Type of Population Growth and the Influence of an External Random Factor on a Number of Such Populations," Physiol. Zool., 32:151-159.

Levin, S. A. and Paine, R. T. 1974. "Disturbance, Patch Formation and Community Structure," Proc. Nat. Acad. Sci. U.S.A., Vol. 71, No. 7:2744-2747.

_____. 1975. "The Role of Disturbance in Models of Community Structure." In Eco-System Analysis and Prediction. Edited by S. A. Levin. SIAM Conference Proceedings, Alta, Utah.

Lewis, E. G. 1942. "On the Generation and Growth of a Population," Sankhyā, 6:93-96.

Lopez, A. 1967. "Asymptotic Properties of a Human Age Distribution Under a Continuous Net Maternity Function," Demography, 4:68-687.

Lotka, A. J. 1922. "The Stability of the Normal Age Distribution," Proc. Nat. Acad. Sci.,VIII:339-345.

_____. 1925. Elements of Physical Biology. Baltimore: Williams and Wilkins. Republished by Dover Publications, 1956.

May, R. M. 1973. Stability and Complexity in Model Ecosystems. Princeton, New Jersey: Princeton University Press.

Nicholson, A. J. 1957. "The Self-Adjustment of Populations to Change," Cold Spring Harbor Symposia Quantitative Biology, 22:153-173.

Nooney, G. C. 1967. "Age Distributions in Dividing Populations," Biophysical Journal, 7:69-75.

Oster, G. and Takahashi, Y. 1974. "Models for Age-Specific Interactions in a Periodic Environment," Ecol. Mon., 44:483-501.

Parker, R. R. and Larkin, P. A. 1959. "A Concept of Growth in Fishes," J. Fish. Res. Bd. Can., 16:721-745.

Pennycuick, C. J., Compton, R. M., and Beckingham, Linda. 1968. "A Computer Model for Simulating the Growth of a Population, or of Two Interacting Populations," J. Theor. Biol., 18:316-329.

Pielou, E. C. 1969. An Introduction to Mathematical Ecology. New York: Wiley.

Ricker, W. E. 1958. "Handbook of Computations for Biological Statistics of Fish Populations," Fish. Res. Bd. Can. Bull. 119:300.

Rosen, R. 1970. Dynamical System Theory in Biology, Vol. I. New York: Wiley-Interscience.

Sharpe, F. R. and Lotka, A. J. 1911. "A Problem in Age Distribution," Philosophical Mag., Ser. 6, XXI:435-438.

Sinko, J. W. 1969. "A New Mathematical Model for Describing the Age-Size Structure of a Population of Simple Animals." Ph.D. dissertation, Department of Electrical Engineering, University of Rochester, Rochester, New York.

_____ and Streifer, W. 1967. "A New Model for the Age-Size Structure of a Population," Ecology, 48:910-918.

_____. 1969. "Applying Models Incorporating the Age-Size Structure of a Population to Daphnia," Ecology, 50:608-615.

_____. 1971. "A Model for Populations Reproducing by Fission," Ecology, 52:330-335.

Skellam, J. G. 1967. "Seasonal Periodicity in Theoretical Population Ecology." In Fifth Berkeley Symp. on Math. Statistics and Probability, Vol. IV. Edited by L. M. Lecam and J. Neymann.

Streifer, W. 1974. "Realistic Models in Population Ecology," Advances in Ecological Research, 8:199-266.

_____, and Istock, C. A. 1973. "A Critical Variable Formulation of Population Dynamics," Ecology, 54, No. 2:392-396.

Thompson, D'Arcy W. 1942. On Growth and Form, Vol. I. Cambridge, England: Cambridge University Press.

Trucco, E. 1965. "Mathematical Models for Cellular Systems: The von Foerster Equation, Parts I and II," Bull. of Math. Biophysics, 27:285-303 and 449-471.

Usher, M. B. 1966. "A Matrix Approach to the Management of Renewable Resources with Special Reference to Selection Forests," Journal Applied Ecol., 3:355-367.

_____. 1972. "Developments in the Leslie Matrix Model," Symp. Brit. Ecol. Soc.,12:29-60.

Von Foerster, H. 1959. "Some Remarks on Changing Populations," In The Kinetics of Cellular Proliferation. Edited by F. Stohlman, Jr. New York: Grune and Stratton.

Von Rosenberg, D. U. 1969. Methods for the Numerical Solution of Partial Differential Equations. New York: American Elsevier.

Weatherley, A. J. 1972. Growth and Ecology of Fish Populations. London: Academic Press.

Weiss, G. H. 1968. "Equations for the Age Structure of Growing Populations," Bull. Math. Biophysics, 30:427-435.

Williamson, M. 1974. "The Analysis of Discrete Time Cycles." In Ecological Stability. Edited by M. B. Usher and M. H. Williamson. London: Chapman and Hall.

APPENDIX

APPENDIX

NUMERICAL SOLUTION TECHNIQUES

A variety of numerical techniques are available for simulation of the dynamics of system (2.2-1), (2.2-7). For the simulations reported in Chapter 5, an explicit finite differencing scheme was employed. Because of its desirable stability and convergence properties, a centered difference equation was chosen to approximate the continuous system.

Let Δz and Δt be discretization increments of p-age and time, respectively. As in Figure A.1, a grid is constructed on the (p-age) x (time) plane, with $\underline{n}$ p-age values along any time line. Let the subscript (i,j) refer to the grid point (z_i, t_j). Then the centered difference scheme is derived by approximating the partial derivatives of Equation (2.2-1) at the center of a grid rectangle by first-order, centered differences. That is,

$$\frac{\partial N_{i+1/2,j+1/2}}{\partial t} \cong \frac{1}{2}\left[\frac{N_{i+1,j+1} - N_{i+1,j}}{\Delta t} + \frac{N_{i,j+1} - N_{ij}}{\Delta t}\right] \qquad (A.1)$$

likewise,

$$\frac{\partial (gN)_{i+1/2,j+1/2}}{\partial z} \cong \frac{1}{2}\left[\frac{g_{i+1,j+1}N_{i+1,j+1} - g_{i,j+1}N_{i,j+1}}{\Delta z}\right.$$

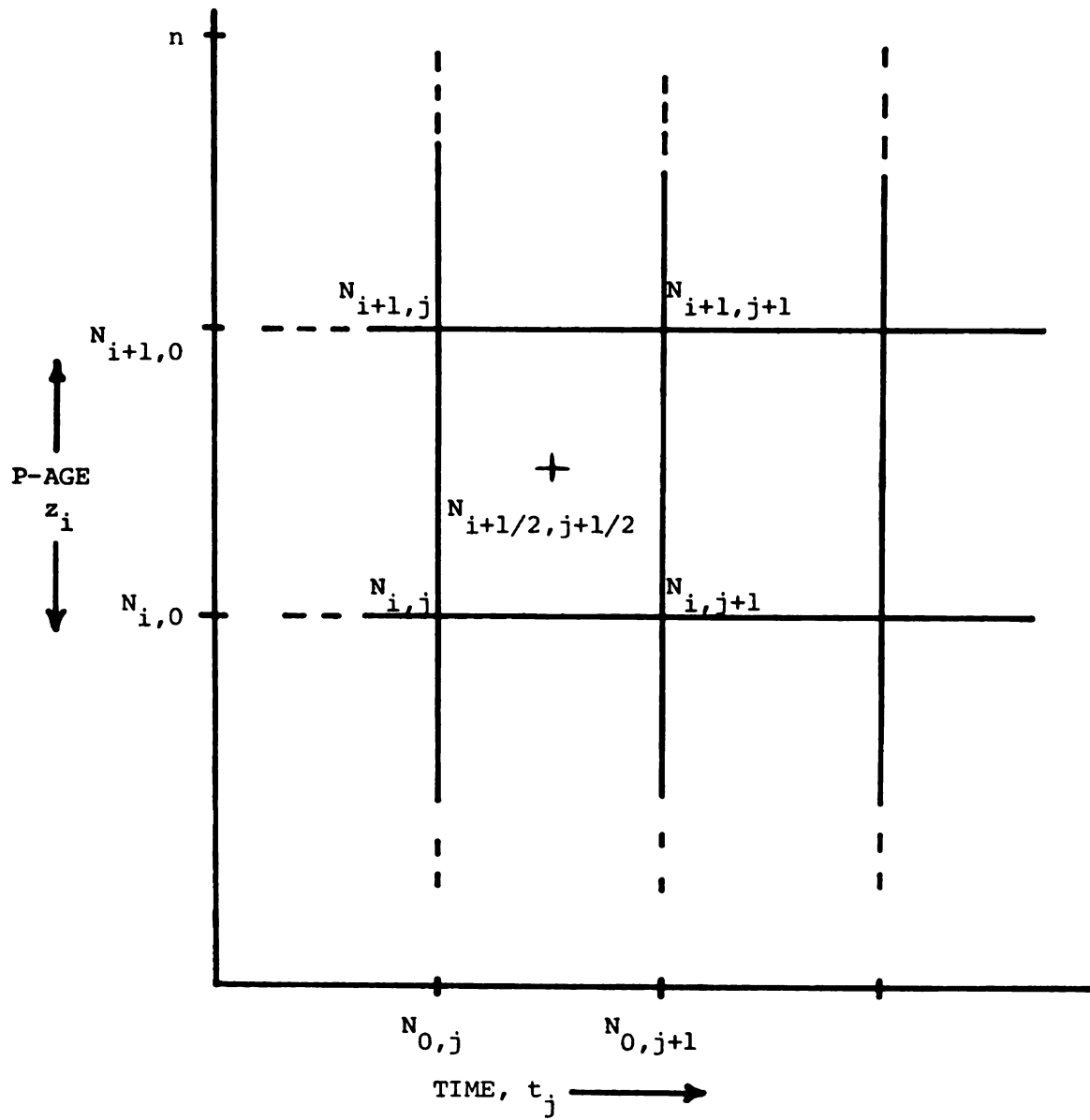$$\left. + \frac{g_{i+1,j}N_{i+1,j} - g_{ij}N_{ij}}{\Delta z}\right] \qquad (A.2)$$

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Figure A.1. Rectangular grid for centered difference scheme.

A four-point average is also taken for the mortality term.

$$(\mu N)_{i+1/2,j+1/2} \cong \frac{1}{4} [\mu_{i+1,j+1}N_{i+1,j+1} + \mu_{i+1,j}N_{i+1,j} + \mu_{i,j+1}N_{i,j+1}$$

$$+ \mu_{ij}N_{ij}] \qquad\qquad (A.3)$$

The terms in (2.2-1) are replaced by their approximation A.1-A.3, and the resulting difference equation is solved for $N_{i+1,j+1}$. This provides an explicit, or recursive, method of computing $N_{i+1,j+1}$ from values $N_{i+1,j}$, N_{ij}, and $N_{i,j+1}$. The boundary condition provides the values $N_{0,j}$, at any time t_j, and the initial condition $N_I(z)$ is discretized to give initial values $\{N_{i,0}\}$, $i = 1,\ldots n$.

The centered difference method requires values of g at time $(j+1)$ to compute the discretized p-age distribution $\{N_{i,j+1}\}$, $i = 1,\ldots,n$. Since g_{j+1} depends on the total population numbers, or biomass (if z = body weight), at t_{j+1}, a predictor-corrector algorithm is used. At the beginning of a time step, g_{j+1} is set equal to g_j. Then a distribution $\{N_{i,j+1}\}$, $i = 1,\ldots n$, is predicted, and the value of g_{j+1} is revised. Using the new value of g_{j+1}, the distribution $\{N_{i,j+1}\}$, $i = 1,\ldots n$, is corrected. This process only required one iteration to give successive estimates of g_{j+1} which were identical to three decimal places.

The integral expressions for reproduction, total numbers, and total biomass were approximated using the trapezoid rule. More elaborate methods, such as Simpson's rule, were tried, but there was no significant effect on the solution curves.

A finite differencing technique is said to be __stable__ if truncation and roundoff errors do not grow as the numerical solution trajectory is

traversed, and it is said to be <u>convergent</u> if the difference equation

converges to the differential equation as Δt, $\Delta z \rightarrow 0$ [Ames, 1969].

Ames states that a generally applicable condition for stability of a

finite difference scheme is that $g \cdot \Delta t \leq \Delta z$, where g is the slope of

the characteristic curves in the (p-age) x (time) plane. In this simula-

tion of population growth, this restriction means that an individual

does not grow through more than one increment in p-age, Δz, in a single

time step of length Δt. If the coefficients of the hyperbolic equation

(2.2-1) are constants then the centered difference method is stable and

convergent for almost any ratio of Δz and Δt [von Rosenberg, 1969].

With a time-varying growth rate, g, it was found that unstable

behavior resulted whenever the ratio $\Delta t_{/\Delta z}$ was considerably smaller

than $1/g$. Incorporating Ames' stability condition, we obtained the best

results using a time increment Δt which could be adjusted at each

iteration, j, to satisfy the condition:

$$\frac{\Delta z}{2} \leq g_i (\Delta t)_j < \Delta z.$$

It was also found that the p-age increment, Δz, could independently

affect the numerical solution. If Δz is too large relative to the width

of the breeding interval, α, then spurious oscillations of total numbers

of biomass are seen. These oscillations disappear, for a given value of

α, as Δz is decreased.

As an independent check on the accuracy of the numerical solution,

the net reproductive rate, R_0, was computed at the conclusion of every

simulation run. The net reproductive rate is a function of growth,

fecundity, and mortality rates, as specified by Equation (5.2-5), and

if a population is stationary, it must satisfy the equation $R_0 = 1$. In all simulations which were allowed to run long enough so that the numerical solution became stationary, the equation $R_0 = 1$ was satisfied to within ±1%.

A centered difference technique was applied by Billups, et al. [1971] to a simulation of an equation of the form (2.3-1) describing a shrimp population. These authors also discuss an implicit differencing scheme which involves solution of a large set of simultaneous linear equations.

Another investigation of numerical schemes for first order, hyperbolic models was carried out by Sinko [1969]. He tried a variety of methods, including a combined centered difference-forward difference equation. He also examined a hybrid method in which the p-age derivative was approximated by finite differences, while the resulting set of ordinary differential equations were solved in the time domain using Runge-Kutta methods. Sinko finally settled on the technique of integrating along the characteristic curves of the partial differential equation [Ames, 1969]. In this method, the characteristic curves themselves are used to construct the discretization mesh. The solution is found by integrating between nodes along characteristics in the mesh. First-order differences are used to approximate the differential equations describing the characteristics.

Sinko decided against using differencing techniques, largely because of their tendency to generate occasional negative values for the discretized density function $N_{i,j}$. This phenomenon is also seen in our simulations, and it appears to be due to the manner in which the

numerical techniques distort the wave-front of a pulse-wave type of initial condition as it moves through the p-age domain. As a square wave moves along the p-age axis in the numerical solutions, waves of small amplitude and high frequency are propagated ahead of the original numerical population wave. When the population numbers are near zero, the troughs of these waves have negative number densities of small magnitudes. These negative number densities become insignificant as Δz is decreased.

As long as they are recognized as numerical artifacts, the negative number densities do not imply that we are modeling a population with "negative" individuals. Unlike Sinko's computer runs, the simulations reported in Chapter 5 are not intended to accurately reproduce the trajectory of a particular population. Since we have been more interested in the general behavior of the system with a density-dependent feedback on the growth rate, the centered-differencing method was more than adequate.

Sinko's comparison tests between differencing techniques and the method of characteristics were conducted using a time-invariant growth function. In future work, we intend to compare the performance of the method of characteristics and the centered-difference method when both use time-varying growth and mortality functions.