

THE DYNAMICS OF STRUCTURED POPULATIONS: SOME EXAMPLES

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1. INTRODUCTION

In realistic models in population ecology individuals are distinguished from one another according to relevant quantities such as age, weight, amount of toxic substances accumulated in the body etc. (Streifer, 1974). The *state of the individual* (i-state) is given by the values of these quantities, whereas the *state of the population* (p-state) is given by the distribution (or density) function describing the number of individuals within each i-state.

In the course of time each individual passes through a trajectory in the i-state space. The beginning and the end of this trajectory are simply its i-state at birth and death, respectively. In between the trajectory is determined by a differential equation describing the instantaneous rate of change of the i-state (aging, growth or accumulation of toxic substances).

Just as in statistical mechanics the evolution of the p-state is governed by a partial differential equation which describes the consequences of these processes at the individual level for the distribution function. The birth and death processes are described by source and sink terms, respectively (one assumes that the number of individuals in the relevant i-states is so large that one can use a deterministic approximation). The continuous change in the i-state is described by a differential operator (such that the characteristics of the first order p.d.e. are precisely the trajectories in the i-state space).

Thus any model for a population with physiological structure consists of at least the three submodels for birth, "growth" and death. The model specification is a description of:

- (i) the chances that an individual with some specific i-state is born or dies;
- (ii) the rate of i-state change;

both as a function of the p-state, the environmental state and the p-states of all other populations which interact with the one under consideration.

The aim of structured population dynamics is to derive information about the dynamics of the population from information about the dynamics of the individuals or vice versa. These models provide links between life history studies on the one hand and measurements of population distributions, as a function of time, on the other. They relate knowledge of physiological processes and behavioural patterns to the

development of the population as a whole.

A characteristic feature of biological (as opposed to physical models) is the occurrence of *non-local terms* (transformed arguments) in the birth term. Consequently, the analysis of such models poses nontrivial and challenging mathematical problems.

One of the reasons to take the population structure into account is to provide a framework for the detailed modelling of the interaction of a population and its environment (or some other population) on the basis of biological knowledge. So, as a rule, one obtains *nonlinear* equations. Although the long term objective is the study of nonlinear problems, I shall here mainly review some recent work on *linear* equations and only in passing will I comment on the incorporation of density dependence. In particular I shall concentrate on the concept of a *stable distribution*. I hope that the examples presented below will give some feeling for the general ideas underlying models of structured populations and that their mathematical analysis serves, apart from its intrinsic interest, as a finger-exercise for the solution of nonlinear problems.

The present paper is based on work by T. Aldenberg, F.H.D. van Batenburg, H.J.A.M. Heijmans, H.A. Lauwerier, J.A.J. Metz, H. Thieme and the author.

2. GROWTH AND DIVISION

Consider a population of unicellular organisms and assume that the physiological state of an arbitrary individual is determined completely by the value of one quantity, denoted by x and called "size", which obeys a physical conservation law (for example, total mass or the amount of nitrogen atoms in the cell). The cells are subject to the following processes: growth, death (= outflow in a chemostat) and fission. Growth is a deterministic process: the rate of size increase of a cell of size x is described by some function $g(x)$, which we assume to be known and to be strictly positive. Death is a stochastic process: the chance (per capita, per unit of time) that a cell of size x dies is described by a nonnegative function $\mu(x)$, which we assume to be known. Fission is a stochastic process. There are (at least) two ways to describe fission into two identical daughters. Let

- (i) $b(x)$ be the rate at which individuals of size x divide;
- (ii) $\gamma(x)$ be the chance (per unit of size) that an individual will have size x at the moment of division;

then one can assume that either b or γ is known. Under constant environmental conditions this just amounts to two different ways of representing the statistics (or, bookkeeping). By following a cohort one finds the relations

$$b(x) = g(x) \frac{\gamma(x)}{1 - \int_a^x \gamma(\xi) d\xi}; \quad \gamma(x) = \frac{b(x)}{g(x)} e^{-\int_a^x \frac{b(\xi)}{g(\xi)} d\xi}, \quad (2.1)$$

where by definition a is the smallest size at which fission can occur (i.e., the smallest point in the support of b and γ). So let us assume for the moment that conditions are constant indeed and return to this point later.

Let $n(t,x)$ be the size distribution function. The balance law for n is (Bell & Anderson (1967), Fredrickson, Ramkrishna & Tsuchiya (1967), Sinko & Streifer (1967, 1971))

$$\frac{\partial n}{\partial t}(t,x) = -\frac{\partial}{\partial x} (g(x)n(t,x)) - \mu(x)n(t,x) - b(x)n(t,x) + 4b(2x)n(t,2x). \quad (2.2)$$

In order to let it fit into our general description one should interpret fission as the "death" of the mother cell and the "birth" of two daughter cells, each of them having half the size of the mother. This equation is supplemented by the boundary condition

$$n(t, \frac{1}{2}a) = 0, \quad (2.3)$$

which expresses that no cells are created with a size less than $\frac{1}{2}a$, and the initial condition

$$n(0,x) = n_0(x). \quad (2.4)$$

Let us assume that cells have to divide before they reach a maximal size, which we normalize to be one. This amounts to the assumption that $\int_a^1 \gamma(\xi) d\xi = 1$, or (see (2.1))

$$\lim_{\varepsilon \rightarrow 0} \int_a^{1-\varepsilon} b(\xi) d\xi = +\infty. \quad (2.5)$$

As a consequence we have to interpret the term $4b(2x)n(t,2x)$ in (2.2) as zero for $x \geq \frac{1}{2}$.

Now suppose that $a \geq \frac{1}{2}$ (i.e., cells cannot undergo two divisions immediately after each other) and put

$$B(t) = n(t, \frac{1}{2}). \quad (2.6)$$

Using elementary integration techniques we find the relation

$$B(t) = \int_{\frac{1}{2}a}^{\frac{1}{2}} K(\xi) B(t+G(\xi)-G(2\xi)) d\xi, \quad (2.7)$$

for $t \geq \bar{t} = \max\{G(2\xi)-G(\xi) \mid \frac{1}{2}a \leq \xi \leq \frac{1}{2}\}$, where by definition

$$K(x) = 4 \frac{b(2x)}{g(2x)} \exp\left(-\int_x^{2x} \frac{\mu(\xi)+b(\xi)}{g(\xi)} d\xi\right) \quad (2.8)$$

and

$$G(x) = \int_{\frac{1}{2}a}^x \frac{d\xi}{g(\xi)}$$

(note that $G(x)$ = the time it takes a cell to grow from size $\frac{1}{2}a$ to size x that K is integrable and that $\int_{\frac{1}{2}a}^{\frac{1}{2}} K(\xi) d\xi = 2$ when $\mu \equiv 0$). B depends in a certain manner on the initial condition for $0 \leq t \leq \bar{t}$. This reduction to equation (2.7) reflects the fact that any cell which takes part in the reproduction necessarily has to pass the size $x = \frac{1}{2}$, so that we can base our bookkeeping of cells at this size. We shall analyse three different cases.

Case (i): $g(2x) < 2g(x)$ for $\frac{1}{2}a \leq x \leq \frac{1}{2}$.

Under this condition the transformation

$$\eta = G(2\xi) - G(\xi)$$

is invertible (note that $\frac{d\eta}{d\xi} = \frac{2}{g(2\xi)} - \frac{1}{g(\xi)}$) and we can write (2.7) as a convolution integral equation

$$B(t) = \int_{G(a)}^{G(1)-G(\frac{1}{2})} K(\xi(\eta)) \frac{d\xi}{d\eta}(\eta) B(t-\eta) d\eta,$$

which has a *positive* kernel. Hence (see, for instance, Hoppensteadt (1980))

$$B(t) \sim C_1 e^{\lambda_d t}, \quad t \rightarrow +\infty,$$

where λ_d , the *dominant eigenvalue*, is the unique real root of the *characteristic equation*

$$\int_{\frac{1}{2}a}^{\frac{1}{2}} K(\xi) e^{\lambda(G(\xi)-G(2\xi))} d\xi = 1,$$

which is obtained by substituting $B(t) = \exp \lambda t$ into (2.7). Note that the left-hand side of (2.13) can be interpreted as the expected offspring ($x = \frac{1}{2}$) of an arbitrary expectant mother cell passing size $x = \frac{1}{2}$. So λ_d exceeds one. The asymptotic behaviour (2.12) is a consequence of that, due to the positivity of the kernel in (2.11), all other roots λ have $\operatorname{Re} \lambda < \lambda_d$. For the size distribution function one finds, after some mo-

$$n(t, x) = e^{\lambda_d t} \{C_2 n_d(x) + o(1)\}, \quad t \rightarrow +\infty,$$

where

$$n_d(x) = \frac{1}{g(x)} \exp\left(-\int_{\frac{1}{2}a}^x \frac{\lambda_d + \mu(\xi) + b(\xi)}{g(\xi)} d\xi\right) \rho(x)$$

with

$$\rho(x) = \begin{cases} 1 & , \quad \frac{1}{2} \leq x \leq 1 \\ \int_{\frac{1}{2}a}^x K(\xi) e^{\lambda_d(G(\xi)-G(2\xi))} d\xi, & \frac{1}{2}a \leq x \leq \frac{1}{2}, \end{cases} \quad (2.16)$$

and

$$C_2 = \frac{\int_{\frac{1}{2}a}^{\frac{1}{2}} e^{\lambda_d(G(\xi)-G(\frac{1}{2}))} \frac{n_o(\xi)}{\{ \frac{n_o(\xi)}{g(\xi)} + K(\xi) \} \int_{\frac{1}{2}}^{2\xi} e^{\lambda_d(G(\eta)-G(2\xi))} \frac{n_o(\eta)}{g(\eta)} d\eta} d\xi}{\int_{\frac{1}{2}a}^{\frac{1}{2}} e^{\lambda_d(G(\xi)-G(2\xi))} (G(\xi)-G(2\xi)) K(\xi) d\xi} . \quad (2.17)$$

In words this says that the population grows exponentially with exponent λ_d (or decays when $\lambda_d < 0$), while the size distribution converges towards the *stable distribution* $n_d(x)$. The initial distribution n_o manifests itself only in the constant C_2 . The infinite dimensional dynamics are asymptotically only one-dimensional!

Case (ii): $g(2x) = 2g(x)$ for $\frac{1}{2}a \leq x \leq \frac{1}{2}$.

Now $G(2\xi) - G(\xi) = G(a)$, a constant, and (2.7) degenerates into the difference equation

$$B(t) = \int_{\frac{1}{2}a}^{\frac{1}{2}} K(\xi) d\xi B(t-G(a)). \quad (2.18)$$

The corresponding characteristic equation is

$$e^{-\lambda G(a)} \int_{\frac{1}{2}a}^{\frac{1}{2}} K(\xi) d\xi = 1, \quad (2.19)$$

and all roots

$$\lambda_k = \frac{1}{G(a)} \{ \ln \int_{\frac{1}{2}a}^{\frac{1}{2}} K(\xi) d\xi + 2k\pi i \}, \quad k \in \mathbb{Z}, \quad (2.20)$$

lie on a vertical line. This vertical periodicity of the spectrum corresponds to the fact that the evolution according to (2.18) is given by multiplication and periodic continuation. As another manifestation of the big difference between this case and the former we mention that, although the cone of nonnegative functions is left invariant, the solution does attain the value zero for arbitrary large time, if the initial function attains zero.

The biological reason for this remarkable behaviour should be clear from the following observation (Bell & Anderson, 1967): if two cells with equal size divide some time after each other their respective daughters will again have the same size since in the time interval between the two divisions the second mother grows exactly

twice as fast as each of the daughters of the first mother! The relation "equal size" is hereditary. Of course this behaviour hinges upon the assumption that each daughter has exactly half the size of the mother. Heijmans (in preparation 1) shows that, also for the case $g(2x) = 2g(x)$, one obtains a stable distribution if the size of a daughter is related to the size of the mother by a smooth probability distribution.

Case (iii): for some $\beta \in (a, 1)$ $g(2x) = 2g(x)$ for $\frac{1}{2}a \leq x \leq \frac{1}{2}\beta$ and $g(2x) < 2g(x)$ for $\frac{1}{2}\beta < x \leq \frac{1}{2}$.

Equation (2.7) can be rewritten as the difference-integral equation

$$B(t) = \int_{\frac{1}{2}a}^{\frac{1}{2}\beta} K(\xi) d\xi B(t-G(a)) + \frac{G(1)-G(\frac{1}{2})}{G(\beta)-G(\frac{1}{2}\beta)} \int_{\frac{1}{2}\beta}^1 K(\xi(n)) \frac{d\xi}{dn} B(t-n) dn. \quad (2.21)$$

The characteristic equation takes the form

$$e^{-\lambda G(a)} \int_{\frac{1}{2}a}^{\frac{1}{2}\beta} K(\xi) d\xi + \int_{\frac{1}{2}\beta}^1 K(\xi) e^{\lambda(G(\xi)-G(2\xi))} d\xi = 1 \quad (2.22)$$

and the unique real root λ_d is dominant: $\operatorname{Re} \lambda < \lambda_d$ for all $\lambda \neq \lambda_d$ which satisfy (2.22). The Laplace transform method may be used to show that the asymptotic behaviour is again given by (2.12) and (2.14)-(2.17). We conjecture that the same results hold in every case in which the complement of $\{x \mid g(2x) = 2g(x)\}$ has positive measure. Finally, we mention that cases with the opposite inequality are mathematically similar but biologically irrelevant.

In Diekmann, Heijmans & Thieme (in preparation) the results reported above are proved in a somewhat different manner (without the restriction $a \geq \frac{1}{2}$). Key ingredients are the theory of semigroups of operators, positivity theory (Krein-Rutman theorem) and compactness arguments. The expansion of the solution into generations (in fact finitely many at each fixed time) turns out to be a very useful tool. In case (i) the semigroup is compact after finite time and this guarantees that the spectrum of the semigroup operators consists of isolated eigenvalues which are related to the spectrum of the infinitesimal generator by the mapping $\lambda \rightarrow \exp \lambda t$. In case (iii) this relation remains valid in the region $\{\mu \mid |\mu| \geq e^{\sigma t}\}$ for some $\sigma < \lambda_d$ (the essential spectrum is bounded inside the circle $|\mu| = e^{\sigma t}$). The determination of the eigenvalues of the generator (including the explicit derivation of the characteristic equation in the general case) is presented in Heijmans (preprint, 1982). Extensions of these results to periodic environments (periodic g , μ and b) are in preparation.

When trying to apply these calculations to "real" microbial populations it might be difficult to determine g and b experimentally whereas the measurement of the stable distribution might be relatively easy. Thus one is led to consider the inverse problem: given the left-hand side of (2.15), derive information about μ , b and g . This is discussed in some detail in the pioneering paper of Bell & Anderson (1967).

The present model allows for the incorporation of density dependence (or, more precisely, nutrient limitation) in a natural and biologically justified manner.

Indeed, one can describe the available substrate by a dynamical variable S and specify how the growth rate g depends on S and how, in turn, the consumption influences S . However, since now the growth rate becomes a function of time, it matters how the fission process is described: should one take b or γ independent of time? Or, possibly, still some other function? What is the intrinsic quantity?

If one assumes that (i) γ is time-independent; (ii) the substrate concentration influences the growth rate as a factor; (iii) the death rate μ is independent of x ; then one can show that the dynamics is described by the linear problem and a non-linear, implicit, time-scaling (these assumptions imply that growth and fission scale with the same factor). One finds convergence towards the stable distribution (which does *not* depend on the dilution rate or the inflowing substrate concentration) and an asymptotic dynamical behaviour described by an unstructured total population system of ordinary differential equations. So, under these conditions, the time evolution of the size structure decouples from the nonlinear interaction. In other words: because of the stable distribution it is safe to ignore the size structure (note, however, that these results might still be relevant in view of the inverse problem). We refer to Diekmann, Lauwerier, Aldenberg & Metz (preprint, 1983) and Heijmans (in preparation 1) for the details. We intend to study the case where (ii) is not satisfied (i.e., the basal metabolism is taken into account) in the near future.

This example shows that a population can be stabilized through a density-dependent effect on the growth-rate of individuals only. Another recent example of the same phenomenon is presented in Nisbet & Gurney (1983). Gyllenberg (1983) uses a somewhat different approach.

3. THE FUNCTIONAL RESPONSE DERIVED FROM THE BALANCE OF DIGESTION AND PREY CONSUMPTION

The functional response of a predator is the number of prey eaten (per predator, per unit of time) as a function of the prey density. Holling (1959, 1966) has analysed prey-predator interactions in some detail in order to derive the functional response from the underlying processes. An important realistic assumption is that these processes take place on a much shorter time-scale than the population reproduction so that, effectively, prey and predator densities may be considered to be constant when calculating the functional response. As a consequence, the functional response may be derived from a linear equation and subsequently used as an input into the nonlinear equations for the prey-predator dynamics.

Neglecting handling times and concentrating on prey consumption and digestion, Metz & van Batenburg (preprint, 1983) arrive at the equation

$$\frac{\partial p}{\partial t}(s,t) = -\frac{\partial}{\partial s}(f(s)p(s,t)) - xg(s)p(s,t) + xg(s-w)p(s-w,t) \quad (3.1)$$

where t = time, s = satiation (i.e., some measure for stomach and gut filling or, in

other words, the inverse of hunger), and

- $p(s,t)$ = the distribution of predators with respect to satiation at time t ,
 $f(s)$ = digestion rate (experimentally found to be described by - as for some constant $a > 0$),
 w = prey weight (assumed to be a constant; Holling (1966) kept it constant in his experiments),
 x = prey density,
 $g(s)$ = catching tendency ($xg(s)$ = catching rate).

In the terminology of the introduction, a predator which eats a prey "dies" and at the same time a new predator with satiation $s+w$ is "born". One can, alternatively, interpret $p(s,t)$ as the probability that one given predator will be in the i -state s at time t . The term $xg(s-w)p(s-w,t)$ should be interpreted as zero for $0 \leq s \leq w$. Experimentally one finds a satiation threshold and this is reflected in the assumption

$$g(s) = 0 \quad \text{for } s \geq c \text{ (the predator is full up).}$$

As a consequence one has to supplement (3.1) with the boundary condition

$$p(c+w,t) = 0. \tag{3.2}$$

Heijmans (in preparation 2) shows that one can associate with (3.1)-(3.2) a semigroup of bounded linear operators on a space of functions on the interval $[0, c+w]$. In fact he first treats the backward equation

$$\frac{\partial q}{\partial t}(s,t) = f(s) \frac{\partial q}{\partial s}(s,t) - xg(s)q(s,t) + xg(s)q(s+w,t) \tag{3.3}$$

on the space $C[0, c+w]$ and then interprets the forward equation (3.1)-(3.2) as the adjoint problem of (3.3) in the space of (normalized) bounded variation functions provided with the weak* topology. Again there exists a dominant real eigenvalue λ_d (of course $\lambda_d = 0$; note that the number of predators $\int_0^{s+w} p(\sigma,t) d\sigma$ is a constant which we take to be 1 with a corresponding nonnegative eigenfunction $p_d(s)$ (also normalized to have integral 1). Moreover, the essential spectrum of the semigroup operators consists of a full circle whose radius is given by $\exp(-xg(0)t)$. Thus one finds the asymptotic behaviour

$$p(s,t) = p_d(s) + o(1), \quad t \rightarrow +\infty. \tag{3.4}$$

Or, in other words: under the influence of prey consumption and digestion the satiation structure stabilizes in the course of time. The functional response is now defined to be the function

$$x \rightarrow x \int_0^{c+w} g(s) p_d(s) ds$$

(note that this is a nonlinear function since $p_d(s)$ depends on x).

Taking formally the limit $w \rightarrow 0$, $x \rightarrow \infty$, $\xi = xw$ constant, we find

$$\frac{\partial p}{\partial t}(s,t) = -\frac{\partial}{\partial s} ((f(s)+\xi g(s))p(s,t)). \quad (3.5)$$

Prey capture is now conceived as a deterministic process: the predator is constantly slurping prey soup. It appears that (3.5) has a stable Dirac delta distribution $\delta(s-\hat{s})$, where \hat{s} is the unique value for which $f(s) + \xi g(s) = 0$. The functional response $\xi \rightarrow \xi g(\hat{s})$ describes the amount of prey soup eaten. In the special case that $g(s) = b(1-\frac{s}{c})$ and $f(s) = -as$ one finds $\hat{s} = bc\xi(ac+b\xi)^{-1}$ and the functional response

$$\xi \rightarrow \frac{b\xi}{1+\frac{b}{ac}\xi}. \quad (3.6)$$

Numerical experiments of Metz & van Batenburg (in preparation) indicate that the deterministic limit yields a very good approximation in most cases of practical interest. Finally, we mention that Heijmans (in preparation 2) uses a Trotter-Kato type theorem to justify the limiting procedure.

4. REMARKS

In recent years the study of age-structured population dynamics has flourished (Busenberg & Iannelli (to appear), Cushing (1980) Gurney & Nisbet (1980), Gurtin & MacCamy (1979), Prüss (1981, to appear), Webb (1982, to appear)) and in the mathematical analysis semigroup methods have proved to be useful. The present note calls attention to two points:

- (i) other-than-age-structures are biologically relevant and mathematically interesting;
- (ii) semigroup methods are appropriate in this context as well.

Until now the area of nonlinear structured models is largely unexplored. Some special examples of age-structured interactions have been studied (Auslander, Oster & Huffaker (1974), Cushing & Saleem (1982), Frauenthal (1983)), Gurtin & Levine (1979), Hastings & Wollkind (1982); sometimes, but not always, the analysis is based on a reduction to a system of ordinary differential equations which is possible under certain restrictive assumptions) and first attempts to investigate the effect of a density-dependent individual growth rate (due to competition for food) have been made (Nisbet & Gurney (1983) Diekmann et al. (preprint 1983)). In a very interesting paper Botsford (1981) argues that the combined effects of a density dependent individual growth rate and cannibalism can lead to multiple stable equilibria and catastrophic effects of parameter (fishing pressure, for example) variation. (See May (1977)

for a discussion of similar phenomena in unstructured population models.) Botsford indeed finds this behaviour in numerical simulations.

Let me mention some more recent work on structured population models (without any claim of completeness) Lasota (1981) and Brunovský (1983) find stable but also chaotic behaviour in a model for the proliferation of differentiating red blood cells (see Lasota, Mackey & Wazenska-Czyżewska, 1981).

Kooijman & Metz (preprint, 1983) study the effects of toxic chemicals on the population growth rate, given the effects on individuals, in the context of a general model for the age- and food-dependent growth and reproduction of individuals (the model is shown to fit the available data on the development of *Daphnia magna* quite well).

Thieme (1982) presents results on stable distributions which apply to many linear and nonlinear models (for instance in epidemiology).

In my opinion these examples underline the need for a general qualitative and quantitative mathematical theory of nonlinear first order partial differential equations with nonlocal terms. At the moment such a theory seems still far-off, but I hope that it will ultimately arise.

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