STOCHASTIC POPULATION MODELS (SPRING 2015)

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1. Introduction

1.1. **What the course is about.** This is a course about population models that cannot be properly described or analysed in a purely deterministic way because of the presence of noise. We consider two kinds of noise depending on its origin: The noise may be exogenous, i.e., due to autonomous processes external to the population itself and affecting it by causing population parameters to fluctuate in time. The noise may also be endogenous, i.e., due to stochastic demographic fluctuations in the number of births and deaths within any given interval of time.

The course addresses the following issues:

Basic notions in model formulation and analysis: the principle of mass-action; growth and development; equilibria and local stability; elements of the theory of Poincare and Bendixon.

The population as a filter of externally generated noise: ordinary differential equations and delay-differential equations; impulse response; frequency response; transfer function; filter characteristics of the population model.

The population as the source of noise: single-type and multi-type birth-death processes; demographic noise; stochastic processes and ergodicity; the Fokker-Planck equation; stochastic differential equations; autocorrelation function and spectral density.

1.2. **Exponential population growth.** Thomas R. Malthus (1766-1834) proposed the following differential equation equation for population growth:

$$\frac{dx}{dt} = ax \tag{1}$$

where x denotes population density, t the time and where $a := b - d$ (the so-called "Malthusian parameter") is the difference between the *per capita* birth and death rates. The solution of the differential equation is

$$(2) \qquad\qquad x(t) = x_0 e^{at}$$

where x_0 is the population density at time $t = 0$. If $a > 0$, the population grows exponentially and without bounds. If $a < 0$, the population dies out. The population stays constant if $a = 0$, i.e., if births and deaths exactly balance, which is very unlikely to happen in reality. The case of unbounded population growth, however, is also obviously absurd, while the case of the extinct population leaves us without study object.

Malthus was aware of these problems, of course. He therefore proposed that the population growth eventually has to slow down as crowding will increase competition and lead to disease and famine, which in turn will reduce the birth rate and increase the death rate such that at sufficiently high population densities the population growth may even be reverted to a population decline.

1.3. **The logistic equation.** Pierre F. Verhulst (1804-1849) proposed a simple linear declining Malthusian parameter as a function of the population density, i.e., $a = r(1 - x/K)$, so that

$$(3) \qquad\qquad \frac{dx}{dt} = rx\left(1 - \frac{x}{K}\right)$$

where $r > 0$ is called the "intrinsic rate of increase" and $K > 0$ the "carrying capacity". Notice that the explicit relation to the separate birth and death rates is no longer present. The equation is known under various names including the "Verhulst equation" and the "logistic equation".

The logistic equation can be solved, but we are not going to do that. Since most ODEs (=ordinary differential equations) cannot be solved explicitly, we make it a habit from the very start to see what information can be obtained directly from the equation without actually solving it.

First, notice from Figure 1 that $dx/dt = 0$ for $x = 0$ and $x = K$, which therefore are equilibrium values, i.e., if we start there, the population will neither grow nor decline, but simply stay at those values. Second, notice that $dx/dt > 0$ (population growth) for $0 < x < K$, and that $dx/dt < 0$ (population decline) for $x > K$. Hence, $x = 0$ is an unstable equilibrium and $x = K$ is a stable equilibrium. Third, dx/dt is maximal for $x = K/2$, i.e., the population growth accelerates on $0 < x < K/2$ and decelerates elsewhere. Without having to solve the ODE, we now nevertheless know that the orbits (=solutions) for different initial conditions must look like as depicted in Figure 2.
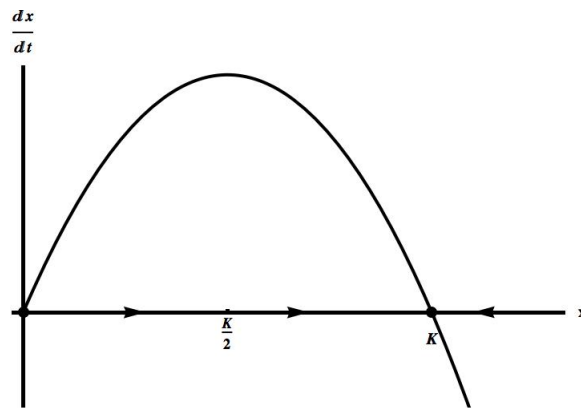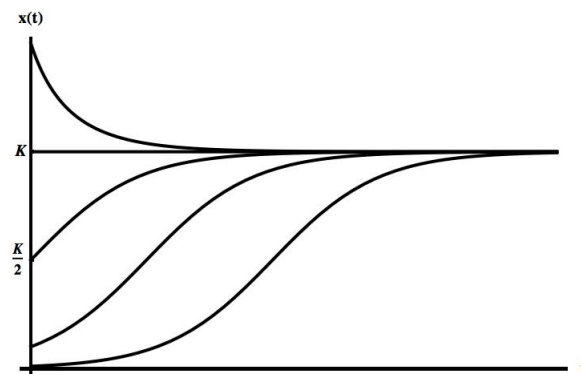
FIGURE 1. Plot of dx/dt as a function of x in the logistic equation.

FIGURE 2. Solutions of the logistic equation for different initial conditions.

1.4. **Mechanistic population models.** Now that we fully understand the qualitative behaviour of the logistic equation, we can "put noise on the system". We can do this by making r and K dependent on time. However, to do this in a meaningful way, we have to understand more of the structure of the model.

We have already seen that in the logistic equation there is no explicit relation between the population growth rate on the one hand and the separate birth and death rates on the other, i.e., we know that $a = r(1 - x/K)$, but we do not know how to split this into a separate birth term (b) and a separate death term (d) such that $r(1 - x/K) = b - d$. This structure simply is not there, not explicitly at any rate. There are infinitely many ways how to accomplish such a split. How we do it matters for the question whether r and K can vary independently or whether they are correlated in some specific way. How, then, do we make a choice?

A population is an ensemble of individuals, and its behaviour (i.e., change in population size and structure over time and space) ultimately is a consequence of

the behaviour of individuals. Therefore, when we make a population model, we should first describe the behaviour of the individuals and from that *derive* a model of the population. In this way all parameters as well as the structure of the model have an interpretation in terms of individual behaviour. In particular, we will know exactly how the individual birth and death rates enter the equations.

A population model that has an interpretation in terms of the behaviour of the individuals is called a "mechanistic population model". If such an interpretation is not given (as for the logistic equation), then we speak of a "phenomenological population model". If it were possible to derive the logistic equation as a mechanistic model (i.e., if we could find a mechanistic underpinning of the logistic equation), then it would be immediately clear how the separate birth and death rates enter the equation. As we shall see, the outcome is not unique and depends on the particular underlying mechanism. Consequently, whether r and K can vary independently or whether they are correlated in some specific way depends on the particular underpinning.

1.5. **The principle of mass-action.** How to make a mechanistic population model? There exist various modeling principles. One of the most useful (and certainly the most frequently used) is the "principle of mass-action" where individuals are modeled as molecules in a well-mixed medium that can undergo reactions just by themselves or by the interaction with other molecules.

Of course, there are many differences between real living individuals on the one hand and inanimate molecules on the other, but there are also similarities, and that is what we exploit. For example, compare the following 'chemical reactions":

$$(4) \qquad\qquad\qquad Hb + O_2 \xrightarrow{\alpha} HbO_2$$

describing a haemoglobin molecule in the blood binding to an oxygen molecule, and

$$(5) \qquad\qquad prey + hungry\ predator \xrightarrow{\beta} satiated\ predator$$

for a predator capturing and eating a prey. The first reaction happens after a chance collision between an Hb molecule with an O_2 molecule. The rate at which such collisions take place in a well-mixed medium is proportional to the Hb concentration as well as to the O_2 concentration. For the change in concentrations we thus have

$$(6) \qquad \begin{array}{rcl} \frac{d}{dt}[Hb] & = & -\alpha[Hb][O_2] \\ \frac{d}{dt}[O_2] & = & -\alpha[Hb][O_2] \\ \frac{d}{dt}[HbO_2] & = & +\alpha[Hb][O_2] \end{array}$$

where α is a constant of proportionality (also called the reaction constant) and where the square brackets denote the concentration. Notice that while $Hb + O_2 \rightarrow$

HbO$_2$ describes a process on the level of the individual molecules, the above system of ODEs describes the behaviour of a population of molecules.

Applying the same formalism to the second "reaction" we get

$$(7) \qquad \begin{array}{rcl} \frac{d}{dt}x & = & -\beta xy \\ \frac{d}{dt}y & = & -\beta xy \\ \frac{d}{dt}z & = & +\beta xy \end{array}$$

where x denotes the population density (=concentration) of the prey, y the population density of hungry predators and z that of satiated predators. Again, while the reaction prey + hungry predator $\to$ satiated predator describes individual behaviour, the ODEs describe changes on the population level.

A predator typically does not meet a prey merely by chance, and they also usually are not "well-mixed" but tend to live in groups, but when we apply the principle of mass-action that is exactly what we assume. A model is never the real thing but just an approximation. There are other modeling principles than mass-action that are more suitable to capture population structure in space. For now we stick to the mass-action principle only.

1.6. **Bimolecular and monomolecular reactions.** In chemistry there are two kinds of elementary reactions: the monomolecular reaction in which a single molecule undergoes a reaction and changes into one or more other kinds of molecules all by itself, and the bimolecular reaction in which two molecules react with one another to produce one or more other kinds of molecules. Tri-molecular reactions or reactions of an even higher order require a chance meeting of three or more molecules at the same time. Such meetings are very improbable and hence must be exceedingly rare.
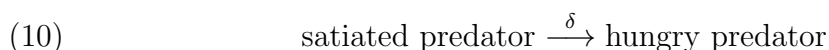
Examples of bimolecular reactions have already been given. An example of a monomolecular reaction is the release of oxygen into the blood by oxygenized haemoglobin:

$$(8) \qquad \qquad \mathrm{HbO_2} \xrightarrow{\gamma} \mathrm{Hb} + \mathrm{O_2}$$

This reaction happens with a constant probability per unit of time per HbO$_2$ molecule. The rate of change in the concentration of HbO$_2$ molecules is therefore proportional to the HbO$_2$ concentration itself:

$$(9) \qquad \begin{array}{rcl} \frac{d}{dt}[\mathrm{Hb}] & = & +\gamma[\mathrm{HbO_2}] \\ \frac{d}{dt}[\mathrm{O_2}] & = & +\gamma[\mathrm{HbO_2}] \\ \frac{d}{dt}[\mathrm{HbO_2}] & = & -\gamma[\mathrm{HbO_2}] \end{array}$$

And here is an example of a monomolecular reaction as a model for a predator digesting its prey:

$$(10) \qquad \qquad \text{satiated predator} \xrightarrow{\delta} \text{hungry predator}$$
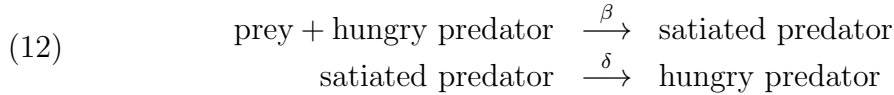
with corresponding population equations

$$
\begin{aligned}
\tfrac{d}{dt}y &= +\delta z \\
\tfrac{d}{dt}z &= -\delta z
\end{aligned}
\tag{11}
$$

Other examples of processes that can be modeled as a monomolecular reaction: the binary fission of a cell, cell death, asexual reproduction, maturation, and divorce.

1.7. Reaction networks.

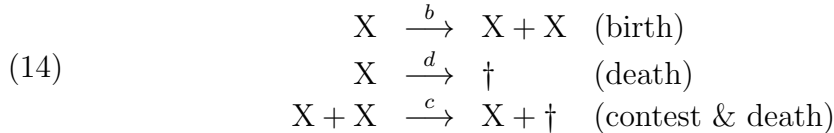Different reactions can be combined into a so-called reaction network:

$$
\begin{aligned}
\text{prey} + \text{hungry predator} &\xrightarrow{\beta} \text{satiated predator} \\
\text{satiated predator} &\xrightarrow{\delta} \text{hungry predator}
\end{aligned}
\tag{12}
$$

The total effect of the reactions on the population densities is equal to the sum of the effect of the individual reactions:

$$
\begin{aligned}
\tfrac{d}{dt}x &= -\beta xy \\
\tfrac{d}{dt}y &= -\beta xy + \delta z \\
\tfrac{d}{dt}z &= +\beta xy - \delta z
\end{aligned}
\tag{13}
$$

We now shall apply the principle of mass-action to derive the logistic equation.

1.8. A derivation of the logistic equation.

Let X denote a single individual and consider the reaction network

$$
\begin{aligned}
\text{X} &\xrightarrow{b} \text{X} + \text{X} \quad \text{(birth)} \\
\text{X} &\xrightarrow{d} \dagger \quad\quad\ \text{(death)} \\
\text{X} + \text{X} &\xrightarrow{c} \text{X} + \dagger \quad \text{(contest \& death)}
\end{aligned}
\tag{14}
$$

Let x denote the population density. Applying the principle of mass-action, the population equation is

$$
\frac{dx}{dt} = bx - dx - \frac{c}{2}x^2
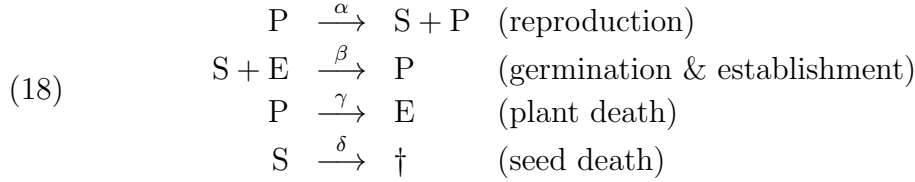\tag{15}
$$

which we can rewrite as

$$
\frac{dx}{dt} = rx\left(1 - \frac{x}{K}\right)
\tag{16}
$$

where

$$
\begin{aligned}
r &= b - d \\
K &= 2(b - d)/c
\end{aligned}
\tag{17}
$$

Returning to the question whether r and K can vary independently or whether they are correlated in some specific way, we see that if we vary r by varying the birth and death rates b and d, then K will vary in concert. However, if we vary K by varying the contest rate c, then r remains unaffected.

1.9. **Another derivation of the logistic equation.** Let P denote an individual plant, S an individual seed and E an empty (i.e., unoccupied) site, and consider the reaction network

(18)
$$\begin{array}{rcll}
\text{P} & \xrightarrow{\alpha} & \text{S} + \text{P} & \text{(reproduction)} \\
\text{S} + \text{E} & \xrightarrow{\beta} & \text{P} & \text{(germination \& establishment)} \\
\text{P} & \xrightarrow{\gamma} & \text{E} & \text{(plant death)} \\
\text{S} & \xrightarrow{\delta} & \dagger & \text{(seed death)}
\end{array}$$

Let p, s and e denote the densities of plants, seeds and empty sites. Applying the principle of mass-action we get the following population equations:

(19)
$$\begin{array}{rclll}
\frac{d}{dt}p & = & & +\beta se & -\gamma p \\
\frac{d}{dt}s & = & \alpha p & -\beta se & -\delta s \\
\frac{d}{dt}e & = & & -\beta se & +\gamma p
\end{array}$$

Notice that $dp/dt + de/dt = 0$, i.e., $p + e$ does not change, which makes a lot of sense, because sites are either occupied (p) or empty (e) so that the total site density $e_0 := p + e$ stays constant. We use this to eliminate the variable e from the above system:

(20)
$$\begin{array}{rclll}
\frac{d}{dt}p & = & & +\beta s(e_0 - p) & -\gamma p \\
\frac{d}{dt}s & = & \alpha p & -\beta s(e_0 - p) & -\delta s
\end{array}$$

Now suppose that both the rate of seed production (α) and the death rate (δ) of seed are very high compared to the colonization rate (β) and the death rate (γ) of plants. For many plant species this seems quite a realistic assumption. Then changes in s are fast compared to changes in p. If the difference is big enough, then s changes on a timescale where p stays virtually constant. Only retaining the large terms αp and δs in the equation for s and ignoring the small term $\beta s(e_0 - p)$, we have approximately

(21)
$$\frac{ds}{dt} = \alpha p - \delta s$$

where p can be treated as a constant.

Plotting ds/dt as a function of s gives Figure 3, from which it can be seen that $s = \alpha p / \delta$ is a stable equilibrium. However, since p is not really constant but changes (albeit on a much slower timescale) we call this not just an equilibrium but a *quasi*-equilibrium.

Substituting $s = \alpha p / \delta$ into the equation of the slow variable p we get

(22)
$$\frac{dp}{dt} = \beta \frac{\alpha p}{\delta}(e_0 - p) - \gamma p$$

which can be rewritten as

(23)
$$\frac{dp}{dt} = rp\left(1 - \frac{p}{K}\right)$$
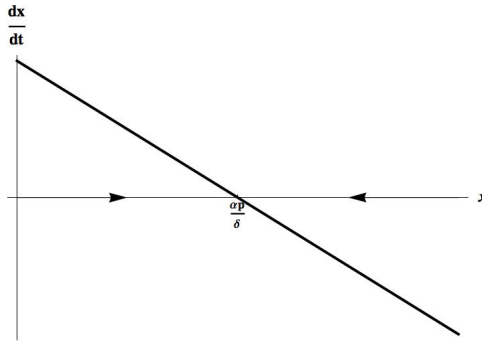
FIGURE 3. Plot of ds/dt as a function of s.

where

(24)
$$r = \frac{\alpha \beta e_0}{\delta} - \gamma$$

$$K = e_0 - \frac{\gamma \delta}{\alpha \beta}$$

Since all parameters occur in both the r and the K, we see that with the present mechanistic underpinning they cannot be varied independently. Not only must they vary together, they must do so in a specific way as they are both functions of the same parameters.

1.10. **Comparison of the two mechanisms.** Figure 4 illustrates some differences in how r and K co-vary as a consequence of changes in the birth and death rates for the mechanisms given in the two previous sections.
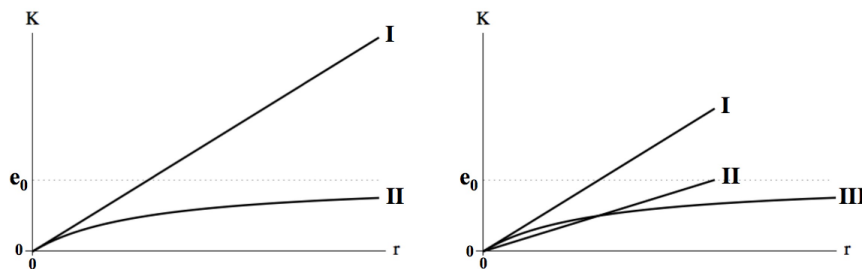
FIGURE 4. Relation between K and r in the logistic model depending on changes in birth and death rates.

In the figure on the left, varying birth rate b in (17) gives line **I** and varying birth rate α in (24) gives the curve **II**. In the figure on the right, varying death rate d in (17) gives line **I**, varying death rate γ (for established plants) in (24) gives line **II** and varying death rate δ (for seeds) in (24) gives curve **III**.

1.11. **Conclusion.** Someone once said that *a model is an equation with an interpretation.* If this is true, then putting noise on the logistic equation by varying the r and/or the K in an arbitrary way without accounting for the underlying mechanisms has no interpretation, and so the logistic equation would stop to be a model at all. How to vary r and/or K in a meaningful way depends, as we have seen, on the underlying individual processes, i.e., on the underlying mechanism.