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MODELS AND METHODS IN MATHEMATICAL EPIDEMIOLOGY

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Introduction

1 Principles of mathematical modelling

By a **mathematical model** we understand an equation, or a set of equations, that describe some phenomenon that we observe in science, engineering, economics, or some other area, that provides a quantitative explanation and, ideally, prediction of observations.

Mathematical modelling is the process by which we formulate and analyze model equations and compare observations to the predictions that the model makes.

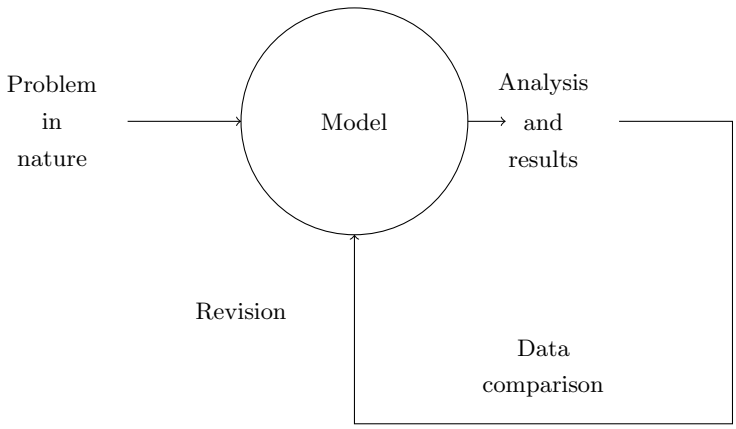


Fig. 1.1. The process of mathematical modelling.

Note:

- Modelling is not mathematics – it is impossible to prove that a model is correct;
- One counterexample disproves the model. However, this not always means that the model is useless – it may just require corrections.

A good model:

- has predictive powers – a model based on available observations gives correct answers in other cases:
  – General Theory of Relativity – light deflection, perihelion precession of Mercury, gravitational waves,
  – Dirac equations – existence of positrons;
- contains earlier working models as subcases:
  – Newton's mechanics is contained in Special/General Theory of Relativity for small velocities and away from

large masses,
– Quantum mechanics yields the same results as Newton's mechanics for large distances, large energies.

Descriptive versus explanatory models. Abundance of data often leads to statistical fitting the data with formulae. One can get a variety of statistical information such as expectations, medians, variance, correlations...
Remember: do not mistake correlations for causation!

Example: it has been observed that since the 1950s, both the atmospheric CO_2 levels and obesity levels in the US have increased sharply. Hence, obesity is caused by high levels of CO_2.

We shall focus on models which try to understand the underlying reasons for the phenomena we observe. Nevertheless, statistical analysis of the data is important as it separates their significant part from the noise.

Statistical (descriptive) models must not be mixed up with **stochastic models**. Stochastic modelling aims to explain the underlying mechanisms of the observed phenomena taking into account inherent(?) randomness of nature. Such models give probabilities of certain events and are indispensable in modeling small populations. We shall focus, however, on **deterministic models** that sometimes can be thought as stochastic models averaged over many individual trajectories (Law of Large Numbers) and giving answers in terms of the evolution of the densities of the populations. Nevertheless, stochastic models are often used explicitly to derive a deterministic model.

1.1 Conservation principles and constitutive relations

Conservation principles

Mathematical biology and epidemiology must obey laws of physics; in particular the balance law. Let Q be a quantity of interest (the number of animals, mass of a pollutant, amount of heat energy, number of infected individuals) in a fixed domain Ω. Over any fixed time interval in Ω we have

$$\begin{aligned}\text{The change of } Q = {} & \text{Inflow of Q} - \text{Outflow of Q} \\ & + \text{Creation of Q} - \text{Destruction of Q}.\end{aligned} \tag{1.1.1}$$

In probabilistic approach this is the same as saying that the probability that one of all possible events occurs equals one.

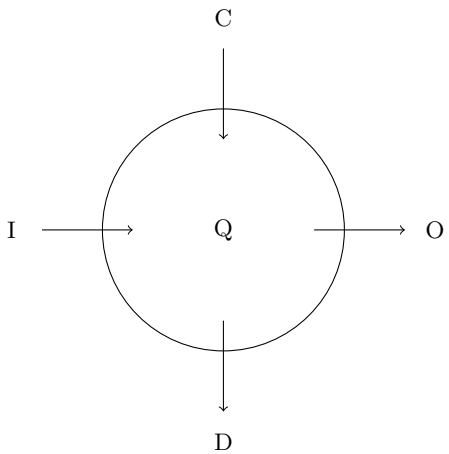


Fig. 1.2. Conservation law for the substance Q.

Continuous and discrete time

Before we proceed, we must decide whether we model with the **continuous time**, or with the **discrete time**.

We use **discrete time models** if we believe that significant changes in the system only occur during evenly spaced short time intervals, or we only can observe the system at evenly spaced time instances and have a reason to believe that essential parameters of the system remain unchanged between successive observations.

Then we use the time between the events/observations as the time unit and count time using the number of elapsed events/observations and (1.1.1) can be written as

$$Q(k+1) - Q(k) = I(k) - O(k) + C(k) - D(k). \tag{1.1.2}$$

Quantities $I(k), O(k), C(k), D(k)$ are the amounts of Q, respectively, that inflows, outflows, is created and destroyed in the time interval $[k, k+1]$.

Examples. Many plants and animals breed only during a short, well-defined, breeding season. Also, often the adult population dies soon after breeding. Such populations are ideal for modelling using discrete time modelling. Let us consider a few typical examples.

(i) Monocarpic plants flower once and then die. Such plants may be annual but, for instance, bamboos grow vegetatively for 20 years and then flower and die. (ii) Animals with such a life cycle are called semelparous.
a) Insects typically die after lying eggs but their life-cycle may range from several days (e.g. house flies) to 13–17 years (cicads).
b) Similar life cycle is observed in some species of fish, such as the Pacific salmon or European eel. The latter lives 10-15 years in freshwater lakes, migrates to the Sargasso Sea, spawns and dies.
c) Some marsupials (antechinus) ovulate once per year and produce a single litter. There occurs abrupt and total mortality of males after mating. The births are synchronized to within a day or two with a predictable 'bloom' of insects.

(iii) A species is called iteroparous if it is characterized by multiple reproductive cycles over the course of its lifetime. Such populations can be modelled by difference equations if the breeding only occurs during short, regularly spaced breeding periods. It is typical for birds. For instance, females of the Greater Snow Geese lay eggs between 8th–20th of June (peak occurs at 12th–17th of June) and practically all eggs hatch between 8th and 13th of July.

If the assumptions allowing us to use discrete time modelling are not satisfied, we use **continuous time**. This, however requires some preparation, as all quantities may change at any instance of time. Thus, I, O, D, C should be considered as the **rates** of inflow, outflow, destruction or creation, respectively; in other words, the amount of Q at a given time t will be given by

$$Q(t) = Q(t_0) + \int_{t_0}^{t} I(s)ds - \int_{t_0}^{t} O(s)ds + \int_{t_0}^{t} C(s)ds - \int_{t_0}^{t} D(s)ds,$$

where $Q(t_0)$ is the initial amount of Q.

Hence, assuming that I, O, D, C are continuous functions, so that Q is differentiable, we obtain the conservation law in differential form,

$$\frac{dQ}{dt}(t) = I(t) - O(t) + C(t) - D(t). \tag{1.1.3}$$

Note 1. The meaning of I, O, C and D (and the dimension) in (1.1.3) is different than in (1.1.2).
Note 2. If we consider populations, then the value of Q always is a nonnegative integer. Such a function can never be continuous. Thus already (1.1.3) is an approximation the validity of which requires that Q be so large that it can be considered a continuum.

In epidemiology we are predominantly concerned with continuous time models.

Constitutive relations.

Real modelling consists in determining the form of Q, I, O, C and D and the relations between them – these are known as **constitutive relations**.

We try to build the functions I, O, D, C to encompass all we know about the process. However, this is usually impossible.

> There are known knowns. These are things we know that we know. There are known unknowns. That is to say, there are things that we know we don't know. But there are also unknown unknowns. There are things we don't know we don't know.
> Donald Rumsfeld

Nevertheless, let us try. The functions I, O, D, C may depend on

- other unknown quantities – this leads to systems of equations that are the main topic of the lectures;
- space or other independent quantities – this leads to partial differential equations that will be discussed in the third lecture;
- explicitly on time – this results in non-autonomous equations which will be discussed later in this lecture;
- the unknown Q in
  a) a nonlinear way, such as $I(t) = I(Q(t)) = Q^2(t)$, or
  b) a linear way, such as $I(t) = I(Q(t)) = 2Q(t)$,
  in which case we talk, respectively, about autonomous nonlinear or linear equations.

Note. It is important to realize that non-autonomous equations often are derived from a larger systems of autonomous nonlinear equations in which the coefficients depend on partial solutions of this system which can be determined explicitly.

1.2 Basic unstructured continuous population models

Malthusian model.

If births and death rates are constant then, denoting the net growth rate by r we obtain

$$\frac{dP}{dt} = rP. \tag{1.1.4}$$

which has a general solution given by

$$P(t) = P(0)e^{rt}, \tag{1.1.5}$$

where $P(0)$ is the size of the population at $t = 0$. The U.S. Department of Commerce estimated that the Earth population in 1965 was 3.34 billion and that the population was increasing at an average rate of 2% per year during the decade 1960-1970. Thus $P(0) = 3.34 \times 10^9$ with $r = 0.02$, and

$$P(t) = 3.34 \times 10^9 e^{0.02t}. \tag{1.1.6}$$

Then the population will double in

$$T = 50 \ln 2 \approx 34.6 \text{ years},$$

which is in a good agreement with the estimated value of 6070 billion inhabitants of Earth in 2000. It also agrees relatively well with the observed data if we don't go too far into the past. On the other hand, if we try to extrapolate this model then in, say, 2515, the population would reach $199980 \approx 200000$ billion giving each of us area of $(86.3\,\text{cm} \times 86.3\,\text{cm})$ to live on.

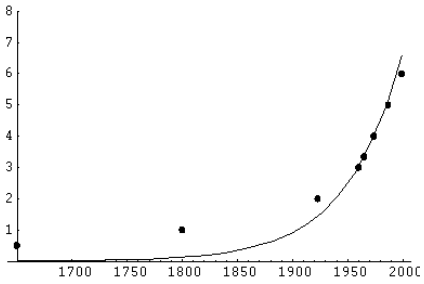


Fig 1.1. Comparison of actual population figures (points) with those obtained from equation (1.1.6).

Nevertheless, the Malthusian model has its uses for short term prediction. It also provides a useful link about the death rate and the expected life span of an individual.

Consider a population in which individuals die at a constant rate μ

$$P' = -\mu P.$$

Then the probability that an individual dies in a time interval Δt is approximately equal to $\mu \Delta t$. Let $p(t)$ be the probability that the individual is alive at time t. Then the probability $p(t + \Delta t)$ of it being alive at $t + \Delta t$ provided he/she was alive at t is $p(t + \Delta t) = (1 - \mu \Delta t)p(t)$ which, as above, yields

$$p' = -\mu p$$

with $p(0) = 1$ (expressing the fact that the individual was born, and thus alive, at $t = 0$) yielding $p(t) = e^{-\mu t}$. The average life span is given by

$$L = \int_0^\infty sm(s)ds,$$

where $m(s)$ is the probability (density) of dying exactly at age s. Since the probability of dying at the age between t and $t + \Delta t$ is

$$1 - p(t + \Delta t) - (1 - p(t)) = -\int_t^{t+\Delta t} \frac{d}{ds} p(s)ds$$

(one should be alive at t and dead at $t + \Delta t$, we have $m(s) = -\frac{d}{ds}p(s)$ and

$$L = -\int_0^\infty s\frac{d}{ds}e^{-\mu s}ds = \mu \int_0^\infty se^{-\mu s}ds = \frac{1}{\mu}. \tag{1.1.7}$$

Nonlinear models with size controlled growth

Logistic equation.

Passing to the limit in the discrete logistic equation valid between t and $t + \Delta t$,

$$P(t + \Delta t) - P(t) = r\Delta t \left(1 - \frac{P(t)}{K}\right)$$

we obtain the continuous logistic model

$$\frac{dP}{dt} = rP(t)\left(1 - \frac{P}{K}\right), \tag{1.1.8}$$

which proved to be one of the most successful models for describing a single species population. The equation has two constant solutions, $P(t) = 0$ and $P(t) = K$, with the latter being the carrying capacity of the environment. Other solutions can be obtained by separation of variables:

$$P(t) = \frac{P(0)K}{P(0) + (K - P(0))e^{-rt}}. \tag{1.1.9}$$

We have

$$\lim_{t\to\infty} P(t) = K, \quad P(0) > 0,$$

hence our model correctly reflects the initial assumption that K is the carrying capacity of the habitat. Next, we obtain

$$\frac{dP}{dt} > 0 \text{ if } 0 < P(0) < K,$$
$$\frac{dP}{dt} < 0 \text{ if } P(0) > K,$$

thus, if $P(0) < K$, the population monotonically increases, whereas if $P(0) > K$, then such a population will decrease until it reaches K. Also, for $0 < P(0) < K$,

$$\frac{d^2P}{dt^2} > 0 \text{ if } 0 < P(t) < K/2,$$
$$\frac{d^2P}{dt^2} < 0 \text{ if } P(0) > K/2,$$

thus, as long as the population is small (less then half of the capacity), then the rate of growth increases, whereas for larger population the rate of growth decreases. This results in the famous *logistic* or *S-shaped* curve that describes saturation process. On the other hand, Verhulst in 1845 predicted, on the basis of the

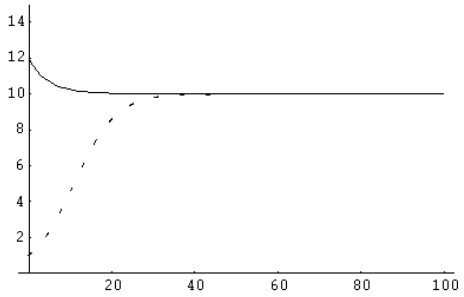


Fig. 1.3. Logistic curves with $P_0 < K$ (dashed line) and $P_0 > K$ (solid line) for $K = 10$ and $r = 0.02$.

logistic equation, that the maximum population of Belgium is 6 600 000. However, already in 1930 it was close to 8 100 000. This is attributed to the global change that happened for Belgium in the XIX century - acquisition of Congo that provided resources to support increasing population (at the cost of the African population of Congo).

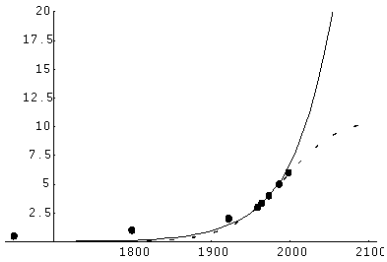


Fig. 1.4. Human population on Earth with $K = 10.76$ billion and $r = 0.029$ and $P(1965) = 3.34$ billion. Observational data (points), exponential growth (solid line) and logistic growth (dashed line).

	Actual	Predicted	Error	%
1790	3,929,000	3,929,000	0	0.0
1800	5,308,000	5,336,000	28,000	0.5
1810	7,240,000	7,228,000	− 12,000	− 0.2
1820	9,638,000	9,757,000	119,000	1.2
1830	12,866,000	13,109,000	243,000	1.9
1840	17,069,000	17,506,000	437,000	2.6
1850	23,192,000	23,192,000	0	0.0
1860	31,443,000	30,412,000	− 1,031,000	− 3.3
1870	38,558,000	39,372,000	814,000	2.1
1880	50,156,000	50,177,000	21,000	0.0
1890	62,948,000	62,769,000	− 179,000	− 0.3
1900	75,995,000	76,870,000	875,000	1.2
1910	91,972,000	91,972,000	0	0.0
1920	105,711,000	107,559,000	1,848,000	1.7
1930	122,775,000	123,124,000	349,000	0.3
1940	131,669,000	136,653,000	4,984,000	3.8
1950	150,697,000	149,053,000	− 1,644,000	− 1.1

Fig. 1.5. Comparison of actual and logistic model population in the United States

A simplified logistic model

We have considered two basic demographical models, the Malthusian model and the logistic model. The drawback of the Malthusian model is that it only can describes a very simple dynamics: the population either decays to zero, or exponentially grows to infinity. The drawback of the logistic model is that it is nonlinear and thus may create additional difficulties in analysis. For this reason an intermediate model is often used in analysis. The model takes the form

$$N' = \Lambda - \mu N, \tag{1.1.10}$$

where Λ is the total birth/recruitment rate and μ is per capita death rate. This is a linear nonhomogeneous equation in N with the solution

$$N(t) = N_0 e^{-\mu t} + \frac{\Lambda}{\mu}(1 - e^{-\mu t}). \tag{1.1.11}$$

It is easy to see that

$$N^* = \frac{\Lambda}{\mu} \tag{1.1.12}$$

is the only equilibrium (!). It is globally asymptotically stable.

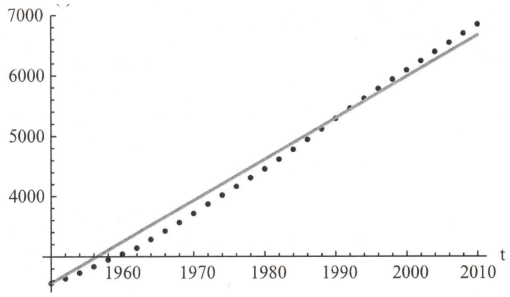


Fig. 1.6. World population alongside the simplified logistic model prediction. The value $P(1950) = 2556.5 \times 10^6$ billion people. The least square error is 703 483. However, the parameters are $\Lambda = 68.5 \times 10^6$ while $\mu = 5.54 \times 10^{-12}$. This give the average lifespan of 1.8×10^{11} years – completely unrealistic.

Holling II type argument.

Consider a sexually reproducing population. We begin with assumption, that over some time T, the number of offspring p per individual in the population of size/density P is proportional to it but we take into account the the reproduction happens only over some shorter period of time T_{as} over which individuals are sexually active:

$$p = rPT_{as}.$$

However, we have to take into account that adult individuals are not always sexually active, for instance during the gestation period. If an adult spends time T_g per offspring for gestation/rearing and, apart from that, it is ready for reproduction,

$$T = T_{as} + pT_r = T_{as} + rPT_{as}T_g$$

then

$$T_{as} = \frac{T}{1 + rT_rP}$$

and hence, the birth rate; that is, the number of offspring per unit time, is given as

$$\frac{dP}{dt} = \frac{pP}{T} = \frac{rP^2}{1 + rT_rP}.$$

A similar argument can be used to derive a Holling type death term used in Allee type models. Consider a population in which individuals must leave a refuge in order to mate. During this time they are exposed to dangers and thus the number of deaths induced by this activity in some period of time T is

$$d = \mu PT_{exp}, \tag{1.1.13}$$

where μ is the additional rate of death due to this activity. Now

$$T = T_s + T_{exp},$$

where T_s is the time spend in a shelter. We assume that the time of an individual is divided between searching for a mate and caring for the offspring. Hence, T_s can be obtained as the product of the number of successful matings in T times T_r that is the average time spend on looking after the offspring. Now, the number of successful matings is the product of the area searched in T_{exp}, the density of males and the efficiency of mating. We assume that the ratio of females and males is constant, so that the density of males is proportional to P. Further, the searched area is proportional to the search time T_{exp} so we can write

$$T_s = aPT_{exp}$$

for some constant a. Hence, as before, we have

$$\frac{dP}{dt} = -\frac{d}{T} = -\frac{\mu P}{1+aP}.$$

An analogous argument can be used in the population that is exposed to a generalist predator; that is, a predator that can eat other prey, so that its number is not affected by the presence of the prey from this particular population. In this case, (1.1.13) is replaced by

$$d = \mu N P T_{hunt}.$$

where N is the (constant) population of predators and T_{hunt} is the hunting time of the predator. Then $T = T_{hunt} + T_{handl}d/N$ where T_{handl} is the handling time of a single prey. Hence $T_{hunt} = T/(1+\mu T_{handl}P)$ and

$$\frac{dP}{dt} = -\frac{\mu N P}{1+\mu T_{handl}P}.$$

Gompertz model.

In the logistic equation we assumed $r(P) = 1 - P/K$, or

$$\frac{dr}{dP} = -\frac{1}{K}, \quad r(K) = 0.$$

A variety of models can be obtained by varying the equation for r. For instance if

$$\frac{dr}{dP} = -\frac{\alpha}{P}, \quad r(K) = 0, \tag{1.1.14}$$

then we have the so-called Gompertz model.

The above equation can be easily solved giving

$$r(P) = \alpha \ln\left(\frac{K}{P}\right)$$

and thus the population equation takes the form

$$\frac{dP}{dt} = \alpha \ln\left(\frac{K}{P}\right) P. \tag{1.1.15}$$

We see that the equation has two equilibria, with $P = K$ asymptotically stable.

One can derive another, possibly more instructive form of this equation. Using (1.1.14) and the Chain Rule, we get

$$\frac{dr}{dt} = \frac{dr}{dP}\frac{dP}{dt} = -\frac{\alpha}{P} rP = -\alpha r.$$

Hence the growth rate decays exponentially as $r(t) = r_0 e^{-\alpha t}$ and we can write (1.1.15) as

$$\frac{dP}{dt} = (r_0 e^{-\alpha t})P = r_0 \cdot (e^{-\alpha t}P). \tag{1.1.16}$$

Different places of brackets indicate different interpretations - the left one suggests that the growth rate is decreasing, while the right one suggests that the pool of fertile individuals is decreasing. That is why the model has been quite successful in modelling cancer. The equation can be solved by separation of variables, giving

$$P(t) = P(0) e^{\frac{r_0}{\alpha}} e^{-\frac{r_0}{\alpha} e^{-\alpha t}}. \tag{1.1.17}$$

Since K is the only asymptotically stable equilibrium,

$$\lim_{t\to\infty} P(t) = P(0) e^{\frac{r_0}{\alpha}} = K$$

we can rewrite the solution in terms of the carrying capacity as

$$P(t) = K e^{e^{-\alpha t} \ln \frac{P(0)}{K}}. \tag{1.1.18}$$

Allee type model

In all previous models with density dependent growth rates the bigger the population (or the higher the density), the slower was the growth. However, in 1931 Warder Clyde Allee noticed that in small, or dispersed, populations the intrinsic growth rate in individual chances of survival decrease which can lead to extinction of the populations. This could be due to the difficulties of finding a mating partner or more difficult cooperation in e.g., organizing defence against predators. Models having this property can also be built within the considered framework by introducing two thresholds: the carrying capacity K and a parameter $0 < L < K$ at which the behaviour of the population changes so that $P' < 0$ for $0 < P < L$ and $P > K$ and $P' > 0$ for $L < P < K$.

The simplest equation of this type has a cubic nonlinearity:

$$\frac{dP(t)}{dt} = r(L - P(t))(P(t) - K))P(t). \tag{1.1.19}$$

A more complex model

$$\frac{dP}{dt} = \lambda P \left(1 - \frac{P}{C} - \frac{A}{1 + BP}\right), \tag{1.1.20}$$

$\lambda, C, A, B > 0$, can be obtained by adding to the logistic growth the additional mortality term $-\lambda AP/(1 + BP)$ that, as we know from modelling of Holling type effects, can be caused by exposure to danger due to search for mates, or by a presence of a generalist predator.

We have to prove that it indeed describes a behaviour required from the Allee model. Let us recall that for this, the equation must have three equilibria, 0 and, say, $0 < L < K$ such that if the size of the population P satisfies $0 < P < L$, then P decreases to 0 and if $L < P < K$, then P increases to K. In the terminology of this section, 0 and K should be asymptotically stable equilibria of (1.1.20) and L should be its unstable equilibrium.

Since (1.1.20) is difficult to solve explicitly (though it is possible as it is a separable equation) we analyse it using the 'phase-plane' argument. The equilibria are solutions to

$$f(P) := P \left(1 - \frac{P}{C} - \frac{A}{1 + BP}\right) = 0. \tag{1.1.21}$$

Clearly, $P \equiv 0$ is an equilibrium so, in particular, any solution originating from $P(0) = P_0 > 0$ satisfies $P(t) > 0$. We see that

$$f'(P) = 1 - \frac{2P}{C} - \frac{A}{(1 + BP)^2} \tag{1.1.22}$$

and since $f'(0) = 1 - A$ we obtain that if $A > 1$, then $P = 0$ is an asymptotically stable equilibrium. By analysing the second derivative we can also state that if $A = 1$ and $BC < 1$, then $P = 0$ is semi-stable, that is, it attracts trajectories originating from positive initial conditions but this case is not relevant in studying the Allee type behaviour. Now we can focus on the other equilibria. For (1.1.20) to describe an Allee model first we must show that

$$1 - \frac{P}{C} - \frac{A}{1 + BP} = 0 \tag{1.1.23}$$

has two positive solutions. It could be done directly but then the calculations become little messy so that we follow a more elegant approach of [**?**] and use the above equation to define a function $A(P)$ by

$$A(P) = \frac{1}{C}(C - P)(1 + BP)$$

and analyse it. It is an inverted parabola satisfying $A(0) = 1$. $A(P)$ takes its maximum at the point P^*, where

$$A'(P) = -\frac{1}{C} + B - \frac{2B}{C}P = 0.$$

This gives

$$P^* = \frac{BC-1}{2B}$$

with the maximum

$$A^* = \frac{(BC+1)^2}{4BC}.$$

Now, the nonzero equilibria of (1.1.20) are the points at which the horizontal line $A = const$ cuts the the

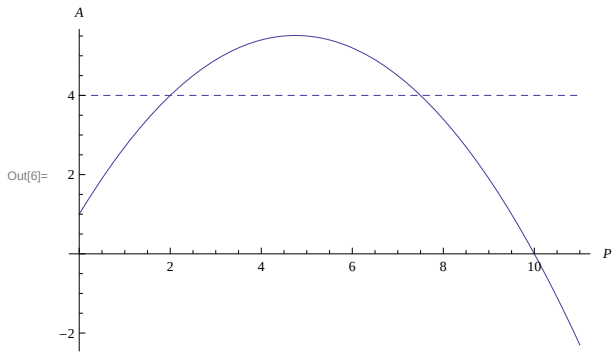


Fig. 1.7. The equilibria as a function of A.

graph of $A(P)$, see Fig. 1.7. First, we note that if $BC < 1$, then the stationary point P^* is negative and thus there is a positive and a negative solution for $0 < A < 1$, a negative and 0 solution for $A = 1$, two negative solutions if $1 < A < A^*$, one (double) negative solution if $A = A^*$ and no solutions if $A > A^*$. If $BC = 1$, then we have one positive, one negative solution for $0 < A < 1$, double 0 solution for $A = A^* = 1$ and no solutions for $A > 1$. Thus, in none case with $BK \leq 1$ we can expect the Allee type behaviour. Let us focus then on the case $BK > 1$. Since $A > 0$, we have the following cases

(a) If $0 < A < 1$, then there are two solutions to (1.1.23), but only one is positive while the other is negative;

(b) If $A = 1$, then there is one 0 and one positive solution to (1.1.23);

(c) If $1 < A < A^*$, then there are two distinct positive solutions to (1.1.23);

(d) If $A = A^*$, then there is a double positive solution to (1.1.23);

(e) If $A > A^*$, then there are no solutions to (1.1.23).

To determine the stability of the equilibria, we re-write (1.1.20) in the following form

$$\begin{aligned}\frac{dP}{dt} &= \lambda P\left(1 - \frac{P}{C} - \frac{A}{1+BP}\right) = \frac{\lambda BP}{C(1+BP)}\left(-P^2 + P\frac{BC-1}{B} + \frac{C(1-A)}{B}\right)\\ &= \frac{\lambda BP}{C(1+BP)}(P-L)(K-P). \qquad (1.1.24)\end{aligned}$$

Using the results of the first part of this section, we can describe the dynamics of (1.1.20) as follows. Let $BC > 1$. Then

(i) For $0 < A < 1$, there is one negative, L, and two nonnegative equilibria of (1.1.20), 0 and K. Zero is unstable and K is asymptotically stable;

(ii) At $A = 1$, the negative equilibrium L merges with 0. Zero becomes semi-stable (unstable for positive trajectories) and K is asymptotically stable;

(iii) For $1 < A < A^*$, there are three nonnegative equilibria, 0 and $0 < L < K$. 0 becomes a stable equilibrium, L is unstable and K is asymptotically stable negative equilibrium L merges with 0. Zero becomes semi-stable (unstable for positive trajectories) and K is asymptotically stable;

(iv) At $A = A^*$, there are two nonnegative equilibria, 0 and double $L = K$. 0 is stable and $L = K$ becomes semistable attracting trajectories from the right and repelling those from the left.

(v) For $A > A^*$, there is only one equilibrium at 0 which is globally attracting.

If $BC \leq 1$, then we cannot have two positive equilibria so that the Allee effect cannot occur in this case. However, to complete analysis, we note that if $0 < BC \leq 1$ then the only case in which there is a positive equilibrium K is for $0 < A < 1$ and in this case K is asymptotically stable while 0 is unstable. For all other cases the only biologically relevant equilibrium is 0 and it is stable if $1 < A$, semistable (attracting positive trajectories) if $A = 1$ and $BC < 1$ and stable if $A = 1 = BC$. Summarizing, (1.1.20) describes the Allee effect if and only if

$$BC > 1 \quad \text{and} \quad 1 < A < \frac{(BC+1)^2}{4BC}. \tag{1.1.25}$$

In any other case with a positive equilibrium the dynamics described by (1.1.20) is similar to the dynamics described by the logistic equation.

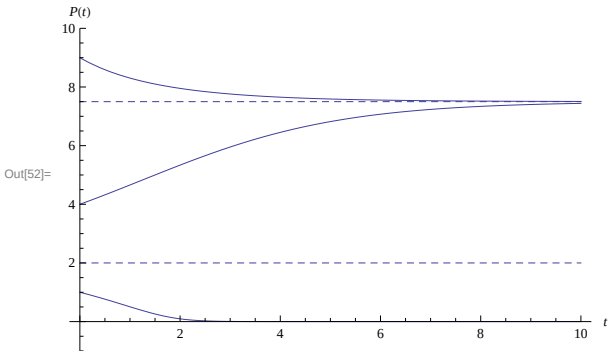


Fig. 1.8. Trajectories $P(t)$ of (1.1.20) for various initial conditions. Here $A = 4$, $C = 10$, $B = 2$, $L = 2$ (lower dashed line), $K = 7.5$ (upper dashed line).

Another way of looking at the problem is to consider the number and stability of the equilibria as a function of a parameter. This approach is known as the *bifurcation theory*. Here we focus on the case $BC > 1$ and we select the parameter A, which can be regarded as representing the extra mortality, over the mortality due to the overcrowding characteristic for the logistic model. Then, for small $A \in (0, 1)$, 0 is an unstable equilibrium and K is stable, as in the logistic model. When A moves through 1, a new positive equilibrium L 'bifurcates' from 0 and the latter changes from being repelling to being attractive; K stays attractive and we are in the 'Allee region'. Finally, when A moves across A^*, K vanishes and 0 becomes globally attractive – large mortality drives the population to extinction. The Allee phenomenon is of concern in many practical applications. For instance, if we try to eradicate a pest whose population can be modelled by an Allee type equation, then it is enough to create conditions if which the size of the population will be below L; the population will then die out without any external intervention. Similarly, if by overhunting or overfishing we drive a population below L, then it will become extinct even if we stop its exploitation.

1.3 Modelling interacting populations

Usually we split the system as

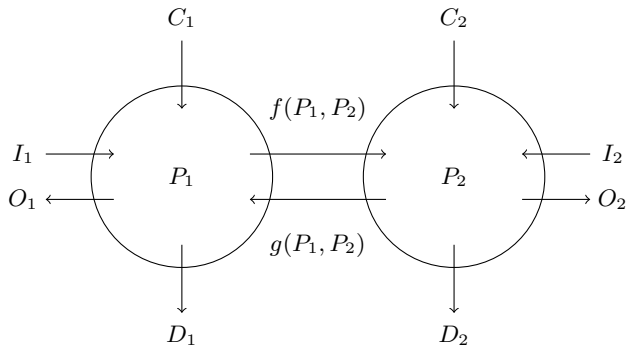


Fig. 1.9. Interactions between populations P_1 and P_2.

$$\frac{dP_1}{dt} = \text{Rate of change of} P_1 \text{ without } P_2 + \text{Impact of } P_2 \text{ on } P_1,$$
$$\frac{dP_2}{dt} = \text{Rate of change of} P_2 \text{ without } P_1 + \text{Impact of } P_1 \text{ on } P_2.$$

P_1 and P_2 typically will be densities of the populations over a certain area. For the first terms in each line we can use any suitable model from the first part.

The second terms require attention.

The simplest terms would be linear terms, e.g.

$$\frac{dP_1}{dt} = r_1 P_1 \left(1 - \frac{P_1}{K_1}\right) + a_{12} P_2$$
$$\frac{dP_2}{dt} = r_2 P_2 \left(1 - \frac{P_2}{K_2}\right) + a_{21} P_1.$$

This system could describe two populations with logistic vital dynamics each, where the additional rate of change of one population due to the other would be directly proportional to the density of the later.
Understanding the interaction terms.
We could distinguish:

- competition, when $a_{12}, a_{21} < 0$, as the presence of each species has a negative impact on the other;
- predator-prey interaction, when $a_{12} > 0$, $a_{21} < 0$, as the presence of species 2 (prey) has a positive impact on the species 1 (predator) and the as the presence of species 1 has a negative impact on the species 2;
- mutualism, when $a_{12}, a_{21} > 0$, as the presence of each species has a positive impact on the other.

In, say, the predator-prey case, the model above implies that the predator eats the same amount of prey per unit time, irrespective of the prey density. This is unrealistic so the coefficients a_{ij} should depend on the density of the ith species. Again, the simplest assumption is that a single predator will consume a proportion of the available prey, leading to the term

$$\alpha_{21} P_1 P_2, \quad \alpha_{21} < 0$$

that is called the *mass action law*, the term borrowed from chemical kinetics where it is assumed that the rate of reactions is proportional to the product of the concentrations of substrats. Hence we have a family of mass-action models of interactions between two species:

$$x' = x(\beta_1 + \mu_1 x + \alpha_{12} y),$$
$$y' = y(\beta_2 + \mu_2 y + \alpha_{21} x),$$

using which we can model various types of interactions and vital dynamics. For instance, assuming predator-prey interactions, $(\alpha_{12} > 0, \alpha_{21} < 0)$, we have $\beta_2 > 0, \mu_2 < 0$ if the prey population follows the logistic vital dynamics in the absence of predator. On the other hand, if we are to model a specialist predator, $\beta_1 < 0, \mu_1 \leq 0$ but for a generalist we can have $\beta_1 > 0, \mu_1 < 0$.

It is clear that the mass action law also is not realistic: for instance, it implies that the predator could eat an arbitrary amount of prey in a unit time (if the density of prey is large enough). To be more realistic, we must incorporate at least some saturation effect. We describe one such model, called Holling type 2 functional response (mass action is termed Holling type 1 response).

We assume that the amount P of prey consumed by a single predator in time T is proportional to the prey density and the time spent on hunting T_h

$$P = cyT_h.$$

The mass action law assumes that $T_h = T$; that is, the predator does not stop hunting while devouring the prey. While it is sometimes possible (e.g. adult salmon eating its offspring), in most cases the time T_e spent on eating the prey is positive. If P is the number of prey caught in time T, then the time used on consuming it is PT_s and thus $T_h = T - PT_s$. Thus

$$P = cy(T - PT_s)$$

and the density of prey eaten per unit time per predator is

$$\frac{P}{T} = \frac{cy}{1 + cT_s y}.$$

Adjusting the number of prey P to the density, we obtain the predation term of the form

$$-c_1 \frac{xy}{1 + c_2 y},$$

with positive constants c_1, c_2.

2

Basic Epidemiological Models

1 Basic epidemiological terminology

An infectious disease is an evident illness caused by microbial agent. The microbial agent can be:

bacteria: tuberculosis, pneumonia;

virus: HIV, influenza;

fungus: dermatomycosis;

parasite: malaria, bilharzia;

toxic protein or prions: Creutzfeldt-Jakob disease (mad cow disease).

Communicable disease are infectious disease that can be transmitted from one infectious person to another, directly or indirectly. There are infectious disease, such as tetanus, is infectious but not communicable. Transmittable diseases are infectious diseases that can be transmitted from one person to another by unnatural routes. For instance, mad cow disease can be passed from one person to another only through a surgical intervention.

For modelling purposes we distinguish the following types of transmission:

direct: when the pathogen is transmitted from one person to another by personal contact, such as sexually tramitted diseases, influenza, smallpox, measles, chickenpox, TB;

vector: when the pathogen is transmitted by a vector such as mosquito, tick or snail, that include malaria, dengue, zika, Lyme disease;

environmental: when a human is infected by a pathogen present in environment, water or food, such as cholera, salmonella, stomach flu;

vertical: mother-to-child transmission, such as HIV.

The following terminology is essential in epidemiological research:

Susceptible individuals: a member of a population who is at risk of becoming infected;

Exposed individuals: susceptible individuals that made a potentially disease-transmitting contact and may, or may not, develop the disease;

Infected and infectious individuals: if a pathogen establishes itself in an individuals, then the individual becomes infected. An infected individual who can transmit the disease is called infectious;

Latent individuals: individuals who are infected but not yet infectious;

Latent period: the time from infection to the moment the individual becomes infectious;

Incubation period: period between exposure to the pathogen to the onset of symptoms of the disease;

Incidence: the number of individuals who become ill during a specified time;

Prevalence: the number of people who have the disease at a specific time.

2 First models

2.1 SIR model

We begin with a simple model of a nonlethal disease in a homogeneous population divided into three classes: susceptible S, infective I and recovered R. Let us denote

λ = the force of infection; that is the rate at which susceptibles become infected,

μ = the death rate,

ν = the recovery rate,

γ = the rate of immunity loss,

B = the birth rate of the population.

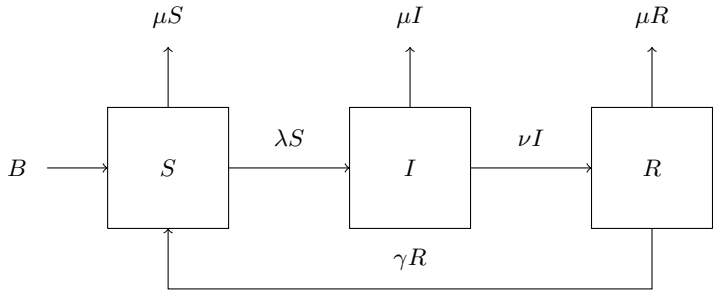


Fig. 2.1. Compartments in the SIRS model

On the basis of the above diagram, we build the following system of equations

$$\begin{aligned} S' &= B(N) - \lambda S + \gamma R - \mu S, \\ I' &= \lambda S - \nu I - \mu I, \\ R' &= \nu I - \gamma R - \mu R. \end{aligned} \tag{2.2.1}$$

S, I and R typically denote the number densities of, respectively, susceptibles, infectives and recovered, B maybe any function describing the vital dynamics of a healthy population (here we tacitly assumed that there is no vertical transmission of the disease or immunity).

Parameter interpretation

We showed that $1/\mu$ gives the expected lifespan of an individual; in the same way $1/\nu$ is the average duration of the disease and $1/\gamma$ is the average period of the acquired immunity.

For a directly transmitted pathogen the force of infection λ is the product of

1. the contact rate;

2. the proportion of these contacts that are with infective;
3. the proportion of such contacts that actually result in infection.

How should we model λ?

A simple assumption would be the mass action law - a single infective meets a fraction $c_1 S$ of susceptibles in a unit time and infects a fraction c_2 of those met:

$$c_2 c_1 SI.$$

As before, this may be a reasonable assumption if the densities are low but for large densities we need to take into account that contacts take time so that there can be only a finite time of contacts in a unit time interval. Also, saturation may be caused by satiation which playes a role in sexual transmission, but also in blood meals taken by mosquitoes.

In a population of size/density N we define $C(N)$ to be the fraction of the population engaged in a contact at any given time. Then $NC(N)$ (precisely $0.5NC(N)$) is the number of pairs in the population at any given time. Since the probability of choosing at random a pair consisting of a susceptible and an infective is

$$\frac{S}{N}\frac{I}{N},$$

the density of pairing that potentially can lead to infection is

$$C(N)\frac{SI}{N}.$$

The function $C(N)$ should be nonnegative, nondecreasing, linear in N for small N and having a limit (≤ 1) as $N \to \infty$. Let us try to derive such a function using a Holling type argument. First, in a population of size N we introduce the number of singles X and pairs P so that

$$N = X + 2P \tag{2.2.2}$$

so that

$$S' = -\beta 2P\frac{SI}{N^2}.$$

Assume that an individual can be either an available single, or form a pair, and that the contact lasts some time T_h. Denote by Z the total number contacts over some time T and let $Y = Z/T$, the number of contacts per unit time. As in the Holling derivation, we have

$$Z = \rho X T_s = \rho X(T - ZT_h), \tag{2.2.3}$$

where ρ is a constant. However, contrary to the predator-prey, where prey was unlimited, here the number of available singles is limited by time - singles are available only when they are not engaged in another contact. For a given single, in T it is available for $T - ZT_h$, and thus for the fraction $1 - YT_h$ of time. In other words, any given single at any given time is available for contact with probability $p = 1 - YT_h$. Thus the expected number of available singles at any given time is given by

$$\begin{aligned} X &= \sum_{k=0}^{N} k\binom{N}{k}p^k(1-p)^{N-k} = Np\sum_{l=0}^{N-1}\binom{N-1}{l}p^l(1-p)^{N-1-l} \\ &= Np = N(1 - YT_h). \end{aligned}$$

Hence, from (2.2.3),

$$\frac{Z}{T} = Y = \rho N(1 - YT_h)^2.$$

Using again (2.2.3), we find that the average number of pairs is

$$P = \frac{1}{2}NYT_h = \frac{\rho T_h}{2}N^2(1 - YT_h)^2 = \frac{\nu}{2}X^2.$$

By (2.2.2),

$$P = \frac{\nu}{2}(N - 2P)^2,$$

or

$$P = \frac{2\nu N + 1 \pm \sqrt{4\nu N + 1}}{4\nu}$$

and we have to select the negative sign,

$$P = \frac{2\nu N + 1 - \sqrt{4\nu N + 1}}{4\nu},$$

to keep $2P < N$. We re-write this as

$$P = \frac{\nu N^2}{2\nu N + 1 + \sqrt{4\nu N + 1}}.$$

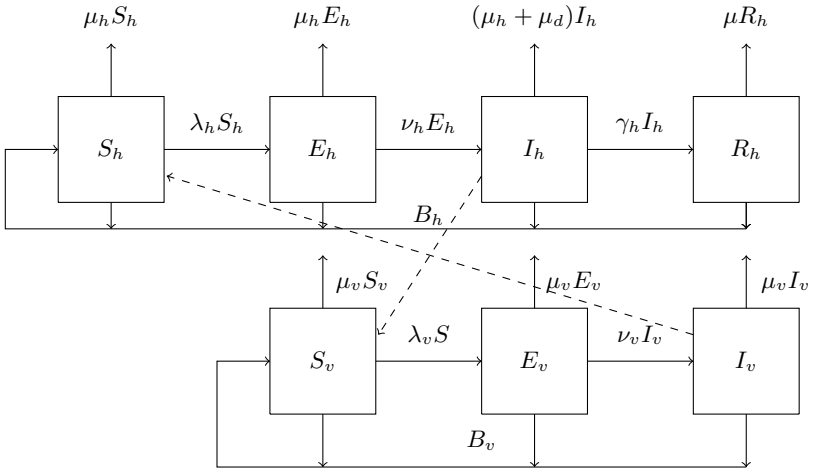
Finally,

$$S' = -C(N)\frac{SI}{N} = -\frac{2\nu\beta N}{2\nu N + 1 + \sqrt{4\nu N + 1}}\frac{SI}{N}.$$

However, most often we use one of the following simplifications:

- low density approximation $C(N) \sim N$ that leads to the mass action transmission rate βIS;
- constant approximation $C(N) \sim 1$ that gives frequency dependent transmission rate $\beta IS/N$.

2.2 A malaria model



$$\begin{aligned}
S_h' &= B_h(N_h) - \lambda_h S_h + \rho_h R_h - \mu_h(N_h)S_h,\\
E_h' &= \lambda_h S_h - \nu_h E_h - \mu_h(N_h)E_h,\\
I_h' &= \nu_h E_h - \gamma_h I - \mu_h(N_h)I_h - \delta_h I_h,\\
R_h' &= \gamma_h I_h - \rho_h R_h - \mu_h(N_h)R_h,\\
S_v' &= B_v(N_v) - \lambda_v S_v - \mu_v(N_v)S_v,\\
E_v' &= \lambda_v S_v - \nu_v E_v - \mu_v(N_v)E_v,\\
I_v' &= \nu_v E_v - \mu_v(N_v)I_v.
\end{aligned} \tag{2.2.4}$$

Here, we have the state variables

S_h: number of susceptible humans,

E_h: number of exposed humans,

I_h: number of infectious humans,

R_h: number of recovered (immune and asymptomatic, but slightly infectious) humans,

S_v: number of susceptible mosquitoes,

E_v: number of exposed mosquitoes,

I_v: number of infectious mosquitoes,

N_h: total number of humans,

N_v: total number of mosquitoes,

and parameters

σ_v: number one mosquito could bite a human per unit time, if humans were freely available. This is a function of the mosquito gonotropic cycle, its preference for human blood and time used for feeding. Time^{-1}.

σ_h: the maximum of mosquito bites a human can have per unit time. This is a function of the human's exposed area, awareness, etc. Time^{-1}.

β_{hv}: probability of infection from an infectious mosquito to a susceptible human, given that a contact between the two occurs. Dimensionless.

β_{vh}: probability of infection from an infectious human to a susceptible mosquito, given that a contact between the two occurs. Dimensionless.

$\tilde{\beta}_{hv}$: probability of infection from a recovered human to a susceptible mosquito, given that a contact between the two occurs. Dimensionless.

ν_h: per capita rate of progression of humans from the exposed state to the infectious state. $1/\nu_h$ is the average duration of the latent period. Time^{-1}.

ν_v: per capita rate of progression of mosquitoes from the exposed state to the infectious state. $1/\nu_v$ is the average duration of the latent period. Time^{-1}.

γ_h: per capita recovery rate of. $1/\gamma_h$ is the average duration of the infectious period. Time^{-1}.

ρ_h: per capita rate of the immunity loss of humans. $1/\rho_h$ is the average duration of the immune period. Time^{-1}.

δ_h: per capita disease induced death rate for humans. Time^{-1}.

Modelling the infection rates

The infection rates are given by

$$\lambda_h = b_h(N_h, N_v)\beta_{hv}\frac{I_v}{N_v}, \quad \text{and} \quad \lambda_v = b_v(N_v, N_h)\left(\beta_{vh}\frac{I_h}{N_h} + \tilde{\beta}_{vh}\frac{R_h}{N_h}\right). \tag{2.2.5}$$

In other words, λ_h is the product of the number of mosquito bites a human can have per unit time, b_h, the probability of the transmission of the infection, $\beta_h v$ and the probability that the bite comes from an infected mosquito, I_v/N_v. Similarly, λ_v is the product the number of human bites a mosquito has per unit times and the sum of probabilities that the bite comes from an infectious human and the transmission occurs. To

model the numbers of bites we first define the total number of bites that occur per unit time, $b(N_h, N_v)$ so that

$$b(N_h, N_v) = b_h(N_h, N_v)N_h = b_v(N_h, N_v)N_v.$$

To derive the formula, we use Holling type argument. In time T the total number of bites received by humans can be written as

$$b(N_h, N_v)T = \sigma_h T_{av} N_h$$

where T_{av} is the time available for mosquitoes to bite. Thus

$$T = T_{av} + T_{nav}.$$

Now, a mosquito cannot bite if it had a meal, and in time T a mosquito has $\sigma_h N_h T_{av}/N_v$ meals and the mosquito is not available for $1/\sigma_v$ after each meal. Thus

$$T = T_{av} + \frac{\sigma_h N_h T_{av}}{\sigma_v N_v}$$

and hence

$$b(N_h, N_v) = \frac{\sigma_v N_v \sigma_h N_h}{\sigma_v N_v + \sigma_h N_h}. \tag{2.2.6}$$

This gives

$$\lambda_h = \frac{\sigma_v \sigma_h}{\sigma_v N_v + \sigma_h N_h}\beta_{hv} I_v, \quad \text{and} \quad \lambda_v = \frac{\sigma_v \sigma_h}{\sigma_v N_v + \sigma_h N_h}\left(\beta_{vh} I_h + \tilde{\beta}_{vh} R_h\right). \tag{2.2.7}$$

2.3 Warm-up – analysis of a simple SIR model

For short lasting diseases, such as flu or common cold, it is customary to discard demographical processes. If the diseases induces immunity, at least in the time covered by the model, one of the simplest models is the SIR Kermack-McKendrick model

$$\begin{aligned} S' &= -\beta SI, \\ I' &= \beta SI - \nu I, \\ R' &= \nu I. \end{aligned} \tag{2.2.8}$$

As we see, we use the mass action transmission rate. The total population at time t is given by $N(t) = S(t) + I(t) + R(t)$ and, by adding the equations in (2.2.8), we obtain

$$N' = 0$$

hence $N(t) = N(0)$, reflecting the assumption that there is no demographic processes included in the model.

The dynamics of the model can be fully analysed without any sophisticated tools.

Step 1. The model is well-posed; that is, for every $(S(0), I(0), R(0)) = (S_0, I_0, R_0)$ there is exactly one solution defined at least on some interval $(-\tau, \tau)$, $\tau > 0$. This follows from the Picard theorem. We shall be interested in $t \geq 0$. Once we know that there is a solution $(S(t), I(t), R(t)), t \in (0, \tau)$, we can prove that it is positive provided $S(0), I(0)$ and $R(0)$ are positive. Indeed, for instance for S, we see that it satisfies the linear equation

$$S'(t) = -\beta I(t) S(t),$$

and hence $S(t) = S(0)e^{-\int_0^t I(s)ds}$, where I is a known function. Hence, $S(t) \geq 0$ as long as $I(t)$ is defined.

Thus, we have $0 \leq S(t), I(t), R(t) \leq N(0)$ for t in any interval on which the solution is defined and hence the solution is defined globally for $t \geq 0$.

Step 2. We see that $S' < 0$; that is, S is decreasing and bounded from below. Since it is defined for all $t \geq 0$, we have

$$\lim_{t\to\infty} S(t) = S_\infty.$$

Similarly, R is growing and satisfies

$$\lim_{t\to\infty} R(t) = R_\infty.$$

Further, since $S(t) + I(t) + R(t) = N(0)$, we must have

$$\lim_{t\to\infty} I(t) = I_\infty.$$

However, the number of infected individuals can increase or decrease depending on the sign of $\beta S(t) - \nu$. In particular, if $\beta S(0) - \nu > 0$, or

$$\frac{\beta S(0)}{\nu} > 1,$$

then the number of infectives initially will increase. Then we say that we have an outbreak or epidemic.

The number $\beta S(0)/\nu$ has an important interpretation. The coefficient β gives the number of infections per unit time induced by one infective, whereas $1/\nu$ is the average time an infective remains infectious. The number of susceptibles at the beginning is $S(0)$. Thus, we have arrived at the common interpretation of $\mathcal{R}_0$

Definition 2.1. *The basic reproduction number $\mathcal{R}_0$ is the number of infections that one infectious individual will introduce in a population consisting only of susceptible individuals.*

Next we estimate the limits. First, observe that $I(t) \neq 0$ for any finite t_0. Otherwise $(S(t_0), 0, R(t_0))$ would be a constant solution to the problem taking the same value as $(S(t), I(t), R(t))$ at $t = t_0$, contradicting the uniqueness of solutions. Hence $t \to R(t)$ is strictly increasing and we can consider $t = t(R)$ on $[R_0, R_\infty)$. Thus, we can consider $S(R) = S(t(R))$ and, using the Chain Rule and the derivative of the inverse function formula, we get

$$\frac{dS}{dR} = \frac{dS}{dt}\frac{dt}{dR} = -\frac{\beta SI}{\nu I} = -\frac{\beta}{\nu}S.$$

Hence

$$S(R) = S(R_0)e^{-\frac{\beta}{\nu}(R-R_0)} \geq S(R_0)e^{\frac{\beta}{\nu}R_0}e^{-\frac{\beta}{\nu}N(0)} > 0$$

for any R. Therefore we must have $S_\infty > 0$ which shows that no epidemic can infect all susceptibles.

Let us consider I_∞. Integrating the first equation in (2.2.8) we obtain

$$S_\infty - S_0 = \int_0^\infty S'(t)dt = -\beta \int_0^\infty S(t)I(t)dt \leq -\beta S_\infty \int_0^\infty I(t)dt.$$

In other words

$$\int_0^\infty I(t)dt \leq \frac{S_0 - S_\infty}{\beta S_\infty} < \infty.$$

Since we know that I_∞ exists and is nonnegative, we must have $I_\infty = 0$.

Step 3. We note that (2.2.8) is really a two-dimensional system and we can find orbits in the (S, I) plane. The two first equations are independent of R and can be solved separately yielding $R = N - S - I$. So, let us focus on

$$\begin{aligned} S' &= -\beta SI, \\ I' &= \beta SI - \nu I. \end{aligned} \tag{2.2.9}$$

From the above discussion, we know that S is a monotonic function of t for $S, I > 0$ and hence it can be inverted $t = t(S)$ allowing for writing $I = I(S)$

$$\frac{dI}{dS} = \frac{dI}{dt}\frac{dt}{dS} = \frac{\beta SI - \nu I}{-\beta SI} = -1 + \frac{\nu}{\beta S}.$$

Separation of variables and integration yields

$$I - I_0 = S_0 - S + \frac{\nu}{\beta} \ln \frac{S}{S_0}.$$

In particular, using that fact that $\lim_{t\to\infty} I(t) = 0$ and $\lim_{t\to\infty} S(t) = S_\infty$ we obtain

$$-I_0 = S_0 - S_\infty + \frac{\nu}{\beta} \ln \frac{S_\infty}{S_0}$$

or

$$\frac{\beta}{\nu} = \frac{\ln \frac{S_0}{S_\infty}}{S_0 + I_0 - S_\infty}. \tag{2.2.10}$$

Let us draw a few conclusions. First, since we know that S is a decreasing function, $S(t) \geq S_\infty$ for any $t \geq 0$. Thus we obtain

$$S_\infty \leq S_0 + I_0.$$

An important information is the maximum number of infectives. This occurs for $I' = 0$ or at $I(0) = I_0$. $I' = 0$ if $S = \nu/\beta$ (and hence this can occur if $S(0) > \nu/\beta$ since S is decreasing). Thus

$$I_{max} = I_0 + S_0 - \frac{\nu}{\beta} + \frac{\nu}{\beta} \ln \frac{\nu}{\beta S_0}. \tag{2.2.11}$$

2.4 (Mis)-matching models

In 1978 there was a report with detailed statistics of a flu epidemic in a boys boarding school with a total of 763 boys. Of these, 512 were confined to bed during the epidemic, which lasted from 22nd January to 4th February 1978. It seems that one infected boy initiated the epidemic. When a boy was infected he was put to bed and so we have $I(t)$ directly from the data.

A best fit numerical technique was used directly on the equations

$$\begin{aligned} S' &= -\beta IS, \\ I' &= \beta IS - \nu I, \\ R' &= \nu I. \end{aligned} \tag{2.2.12}$$

for comparison of the data. These gave $\beta = 2.1810^{-3}$/day, $\nu = 0.44$; that is, infectious period of 2.27 days, and

$$\mathcal{R}_0 = 2.18 \cdot 10^{-3} \cdot 762 \cdot 2.27 \approx 3.77.$$

However, the above approach ignores that flu, like most other diseases, has a latent period – there is a delay of 1 to 4 days in an infected becoming infective. The simplest way of incorporating the delay is to introduce the exposed class(es). In the case discussed here

$$\begin{aligned} S' &= -\beta IS, \\ E' &= \beta IS - \sigma E, \\ I' &= \sigma E - \nu I, \\ R' &= \nu I. \end{aligned} \tag{2.2.13}$$

How different can be SIR and SEIR models resulting from fitting the same data? We compare the estimated $\mathcal{R}_0$ numbers. First, observe that for the SEIR model, $\mathcal{R}_0^{SIR}$ is given by the same formula

$$\mathcal{R}_0^{SEIR} = \frac{\beta S(0)}{\nu}.$$

We use A. Lloyd approach. If SIR and SEIR models give the same data, their initial growth rate of I should be the same. For SIR initially

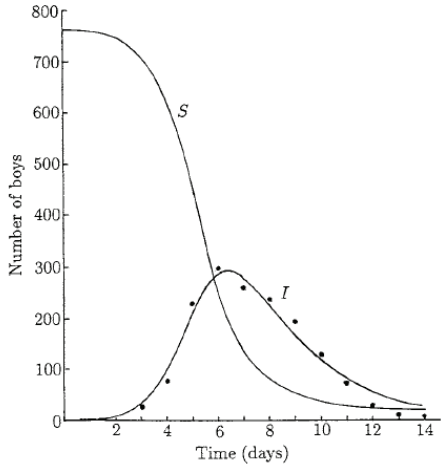


Fig. 2.2. Parameter values are $N = 763$, $S_0 = 762$, $I_0 = 1$ and, fitted, $\beta = 2.1810^{-3}$/day, $\nu = 0.44$.

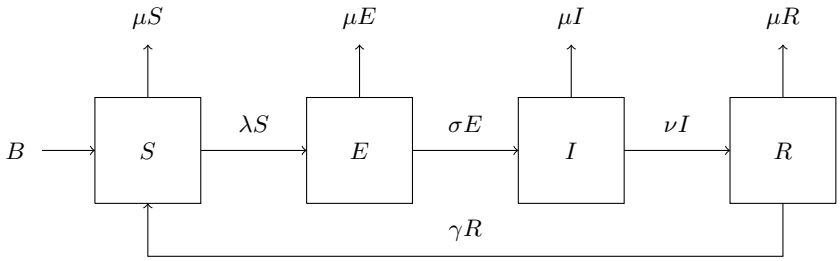


Fig. 2.3. Compartments in SEIRS model

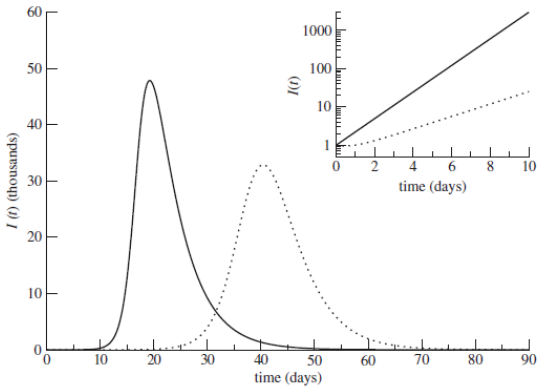


Fig. 2.4. Solid curve: SIR model, dotted curve: SEIR model. The inset compares the initial behavior of the two outbreaks. $1/\nu = 5$ days, $\mathcal{R}_0 = 5$, $1/\sigma = 2$ days, $N_0 = S_0 = 10^6$.

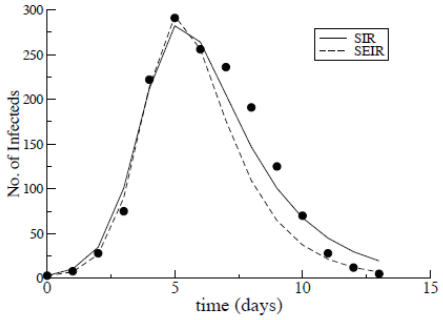


Fig. 2.5. SIR and SEIR models fitted to the available data (first half).

$$I' = \beta S(0)I - \nu I = \nu(\mathcal{R}_0^{SIR} - 1)I$$

initially $I(t) = I_0 e^{rt}$ with $r = \nu(\mathcal{R}_0^{SIR} - 1)$ or $\mathcal{R}_0^{SIR} = 1 + \nu^{-1} r$. On the other hand, for SEIR

$$E' = \beta S(0)I - \sigma E,$$
$$I' = \sigma E - \nu I.$$

and the initial growth rate r is the biggest root of the eigenvalue equation

$$r^2 + (\nu + \sigma)r - \sigma\nu(R_0^{SEIR} - 1) = 0$$

or

$$\mathcal{R}_0^{SEIR} = (1 + \nu^{-1} r)(1 + \sigma^{-1} r) = \mathcal{R}_0^{SIR}(1 + \sigma^{-1} r).$$

If we observe the same data and try to use SIR and SEIR models, the observable r

$$r = r^{SIR} = r^{SEIR} = 1.22.$$

If we add the latency period of 1 day

$$\mathcal{R}_0^{SEIR} = \mathcal{R}_0^{SIR}(1 + r) = 3.77 \cdot 2.22 = 8.37.$$

2.5 Models reducible to one-dimensional problems

The SIS model

If the disease does not induce immunity but, instead, after recovery the infected individuals become again susceptible, then the SIR model turns into the SIS model

$$S' = -\beta SI + \alpha I,$$
$$I' = \beta SI - \alpha I, \tag{2.2.14}$$

where α is the rate of recovery. Here, again, if we add the equations, we will find that the total population $N = S + I$ is constant in time. Thus, we can write

$$S = N - I$$

and thus (5.5.81) reduces to

$$I' = \beta I(N - I) - \alpha I = (\beta N - \alpha) I \left(1 - \frac{I}{\frac{\beta N - \alpha}{\beta}}\right) = rI\left(1 - \frac{I}{K}\right). \tag{2.2.15}$$

This is the logistic equation that was analysed earlier. In particular, we have the following cases