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↪ We now look at the *population dynamics* of *infectious diseases*.

↪ There is an interesting historical background to this topic in section 10.1 of J.D. Murray's *Mathematical Biology: I. An Introduction, Third Edition*.

↪ Mathematical modelling of diseases in the "modern" era could be said to begin with Hamer and Ross (the latter winning a Nobel prize in 1902 for demonstrating that malaria was transmitted by mosquitoes) who used mathematical models to test their theories.

↪ The other significant development was the 1927 publication of a paper by William O. Kermac and Anderson G. McKendrick, *A Contribution to the Mathematical Theory of Epidemics*, in which they outlined the SIR model we will look at later. The model in that classic paper was the basis for much further work on the topic.

▶ Now the mathematical modelling of the spread of infectious diseases is a flourishing field and informs public health policy.

Some Key Terminology

↪ **ENDEMIC** diseases are constantly (or usually) present in a population.

↪ **EPIDEMIC** diseases are prevalent in a population only at certain times or under certain circumstances (hence are NOT *endemic*).

↪ An individual's disease status[1] could, broadly speaking, be:
  — **Susceptible** if they can get the disease;
  — **Latent** or **exposed** if they are infected but not yet infectious;
  — **Infectious** or **infective** if they can infect others;
  — **Removed** if they are no longer infectious, whether due to acquired immunity, quarantine (isolation), or death;
  — **Carrier** if they are infectious for long periods but show no symptoms.

↪ A disease is **CONTAGIOUS** if it is spread by contact between an *infected* and a *susceptible* individual.

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[1] We will only look at the disease statuses highlighted in yellow above and discuss models which predict how the proportion of a population with each disease status changes with time.

Some Key Terminology, continued

$\hookrightarrow$ Infective diseases fall into two main categories dependent on the (original) infective agent:

A. **MICROPARASITIC**: These are diseases caused by very small infectious agents - too small to be seen with the naked eye. These agents can be viruses *(e.g., Measles, Ebola, Zika)*, bacteria *(e.g. Tuberculosis, Streptococcus, Escheria [E.] coli)*, protozoan *(e.g., Malaria, Giardia)*, etc. Here, essentially someone either has the disease or does not have the disease.

B. **MACROPARASITIC**: These are diseases caused by larger infectious agents - large enough to be seen with the naked eye. These agents can be helminths *(e.g. tapeworms, nematodes)*, arthopods *(e.g., ticks, lice)*, etc. Here, the level of infestation might be relevant to the progress of the disease.

Some Key Terminology, continued

↪ The **PREVELANCE** of a disease is the proportion of the population infected.

↪ The **INCIDENCE** of an infectious disease is the rate at which infection occurs.

↪ A **COMPARTMENTAL MODEL** is often used to model the "flow" of diseases within a population.

  ▶ Each disease status is assigned a separate compartment within which the number of individuals typically changes over time.

  ▶ The "flow" of individuals from one compartment to another is indicated by arrows often accompanied by the transition rates between those compartments. An arrow leaving or entering a compartment, respectively, indicates a negative or positive, respectively, rate of change of the number of individuals in that compartment.

$\hookrightarrow$ For example,

$$S \to I \to R \quad or \quad \boxed{S} \to \boxed{I} \to \boxed{R}$$

is a compartment model (omitting the rates of transition between compartments) which indicates that in a population, individuals are first *susceptible*, then some are *infected*, then some infected people are *removed*.

$\mapsto$ Other key terminology will be defined when needed.

▶ One assumption in the *Kermac-McKendrick* model and other early models is that population is well-mixed and the likelihood of any two individuals coming into contact with each other follows the **law of mass action**. This would indicate that each person in the population is equally likely to meet someone else in the population. While this is a reasonable first approximation, it is typically not the case in reality *(for example, think about the relatively small number of and largely predictable/fixed group of people many people encounter in a typical day)* and hence this assumption is usually refined to be more realistic in later models.

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SIS Epidemic - Introduction

$\hookrightarrow$ We will model a disease in which each individual in a population can either be *Susceptible (S)*, *Infected (I)* or can recover and immediately be *Susceptible (S)* again.

$\rightsquigarrow$ This can be the case with diseases such as tuberculosis or gonnorrhoea (bacterial), or the common cold or the flu (viruses), in which people who recover are frequently not immune and could get that disease again if they are not careful.

$\hookrightarrow$ We will however briefly begin by looking at the simpler model in which we assume that an infected person remains infected indefinitely. This is representative of what happens near the start of an infectious outbreak but clearly is not a realistic model for the longer-term dynamics of the disease. Thus the model we consider first is

$$S \to \text{I}.$$

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SI Epidemic (The Simple Epidemic)

$\hookrightarrow$ Let $S(t)$ be the number of individuals who are susceptible at time t and $I(t)$ be the number of individuals who are infected at time t.

$\hookrightarrow$ In this simple epidemic model, we assume that the overall population is constant, call it N, and that everyone is either susceptible or infected. So

$$S(t) + I(t) = N.$$

$\hookrightarrow$ One could now derive a form of the differential equation(s) directly using this *conservation law* for the total population:

$$\frac{dS}{dt} + \frac{dI}{dt} = \frac{dN}{dt} = 0 \quad \Rightarrow \quad \frac{dS}{dt} = -\frac{dI}{dt}.$$

$\blacktriangleright$ The problem is that this is one differential equation with two unknown functions, so it would be helpful to have a better idea of what $\frac{dS}{dt}$ or its negative $\frac{dI}{dt}$ looked like.

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$\hookrightarrow$ In general, if we make the simplifying assumption that both $\frac{dS}{dt}$ and its negative $\frac{dI}{dt}$ depended only on S and I (and not directly on t), then

$$\frac{dS}{dt} = -F(S, I), \qquad \frac{dI}{dt} = F(S, I)$$

where, based on earlier definitions, $F(S, I)$ is known as the
                    of the disease.

$\succ$ Clearly, we are assuming here that $F(S, I)$ is a positive function given the direction of the arrow in the compartment model $S \to I$.

$\hookrightarrow$ We will assume that the rate of infection follows the **law of mass action** with proportionality constant β: Thus

$$\frac{dS}{dt} =$$

▶ NOTE that last set of ODEs could also be summarised by the following compartment model diagram:

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↪ The constant β is called the `(pairwise) infectious contact rate` and is the *rate of infection per infectious and per susceptible individual*.

↪ More generally, the *incidence* of the disease, $F(S, I)$ is often given as a *function* of I multiplied by S:

$$F(S, I) = \lambda(I)S$$

where $\boldsymbol{\lambda(I)}$ is called the `force of infection`. So in the current SI model, we have taken the simplest, non-constant force of infection, a linear functon βI.

↪ Crucially, the *conservation law* $S(t) + I(t) = N$ means that we can write one of the functions, $S(t)$ or $I(t)$, in terms of the other and easily reduce the system of two ODEs to a single ODE.

↪ For example, since $S(t) = N - I(t)$

$$\frac{d\mathbf{I}}{d\boldsymbol{t}} = \beta\mathbf{I}(\boldsymbol{N} - \mathbf{I})$$

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↪ This is just a **Logistic** ODE   with
   intrinsic growth rate $r =$ and carrying capacity
   $K =$.

↪ Clearly, in the long term, this model predicts that the
   entire population will become infectious (the stable
   equilibrium solution is $I = N$, $S = 0$). This is unrealistic
   and is why this model is not relevant for the long-term
   progress of a disease.

  ▶ Instead, the SIS model is more realistic.

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SIS Epidemic

$\hookrightarrow$ Compared to the SI model, the only additional assumption in the SIS model is that infectious individuals *can* recover; but they acquire no immunity and are immediately again susceptible.

$\hookrightarrow$ We will assume that the *rate* at which the infected individuals recover (and become susceptible again) is proportional to the number of infected individuals with proportionality constant μ. Hence the compartment model (with rates of transition) is

$\hookrightarrow$ NOTE that, $\frac{dN}{dt} = \frac{d(S+I)}{dt} = \hspace{4cm} 0,$
so as expected the total population $N = S(t) + I(t)$ is a constant.

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$\hookrightarrow$ NOTE again as in the SI case, we can use the fact that $S + \mathrm{I} = N$ to write this as a single ODE for $\frac{dS}{dt}$ or $\frac{d\mathrm{I}}{dt}$. For example, eliminating $S(t)$ from that equation we come up with the equivalent single ODE

$$
\frac{d\mathrm{I}}{dt} \;=\; \beta\mathrm{I}(N - \mathrm{I}) - \mu\mathrm{I} \;=\; \beta N\mathrm{I}\left(1 - \frac{\mathrm{I}}{N}\right) - \mu\mathrm{I}.
$$

$\hookrightarrow$ At this stage, we have a choice of whether to analyse this SIS model as a system or as a single equation. I will leave you to analyse it as a system using the techniques of **Lecture 4** and **Lecture 5** and verifying that you come to the same conclusions that we do when analysing it as a single equation in what follows.

$\hookrightarrow$ First, to reduce the number of parameters, we will non-dimensionalise the equation (using the 5-step procedure from **Lecture 5**).

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REMINDER: $\dfrac{d\mathrm{I}}{dt} = \beta\mathrm{I}(N-\mathrm{I}) - \mu\mathrm{I} = \beta N\mathrm{I}\left(1 - \dfrac{\mathrm{I}}{N}\right) - \mu\mathrm{I}$

1. We will non-dimensionalise $\mathrm{I}(t)$ and t, calling the new variables x and τ respectively.

$$\frac{dx}{d\tau} = \frac{\beta N}{\mu}x\left(1 - x\right) - x$$

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REMINDER: $\dfrac{dx}{d\tau} = \dfrac{\beta N}{\mu} (1 - x) x - x$

5. If we let $R_0 = \dfrac{\beta N}{\mu}$ then the non-dimensionalised ODE with only one parameter, R_0 is

$$\frac{dx}{d\tau} = R_0 (1 - x) x - x.$$

$\hookrightarrow$ In these dimensionless variables, $x(\tau) = \frac{I}{N}$, is just the proportion of the population that is infected. Hence, the proportion of the population at time τ which is susceptible is $1 - x(\tau)$ and we shall call it y.

$\hookrightarrow$ Given that μ is the rate at which infected people recover (and become susceptible), then $1/\mu$ is the typical (average) recovery time *(which is the same as the typical time an individual is infectious)*. Hence the dimensionless time variable $\tau = \mu t = \dfrac{t}{1/\mu}$ measures time as a proportion of the average recovery time.

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REMINDER: $\dfrac{dx}{d\tau} = \dfrac{\beta N}{\mu} (1 - x) x - x = R_0 (1 - x) x - x$

$\hookrightarrow$ Exercise: Use dimensional analysis to interpret the parameter R_0.

▶ Answer:

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[2] See Sec. 3.2 of *Essential Mathematical Biology* by Britton or Example 6.7 of *A Primer on*

Mathematical Methods in Biolgy by Edelstein-Keshet for alternative routes to the same interpretation of R_0

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REMINDER: $\dfrac{dx}{d\tau} = \dfrac{\beta N}{\mu}(1-x)x - x = R_0(1-x)x - x$

$\hookrightarrow$ The equilibrium solutions to this ODE are solutions to

$$R_0 x - R_0 x^2 - x = 0 \quad \Rightarrow \quad x(R_0 - R_0 x - 1) = 0 \quad \Rightarrow$$

$$x = 0 \quad (so \ y = 1) \quad or \quad x = \frac{R_0 - 1}{R_0} = 1 - \frac{1}{R_0} \quad (so \ y = \frac{1}{R_0}).$$

$\hookrightarrow$ We use calculus to classify these equilibrium solutions:

$$\frac{d}{dx}\{R_0(1-x)x - x\} = R_0 - 2R_0 x - 1.$$

− Evaluated at $x = 0$ this derivative is $R_0 - 1$ which is < 0 if $R_0 < 1$ and > 0 if $R_0 > 1$ so that if $R_0 < 1$ ($x = 0, y = 1$) is a stable steady state and if $R_0 > 1$ ($x = 0, y = 1$) is an unstable steady state.

− Evaluated at $x = 1 - \frac{1}{R_0}$ the derivative is $-R_0 + 1$ so if $-R_0 + 1 < 0 \Rightarrow R_0 > 1$ then $\left(x = 1 - \frac{1}{R_0}, y = \frac{1}{R_0}\right)$ is a stable steady state and if $-R_0 + 1 > 0 \Rightarrow R_0 < 1$ then $\left(x = 1 - \frac{1}{R_0}, y = \frac{1}{R_0}\right)$ is an unstable steady state.

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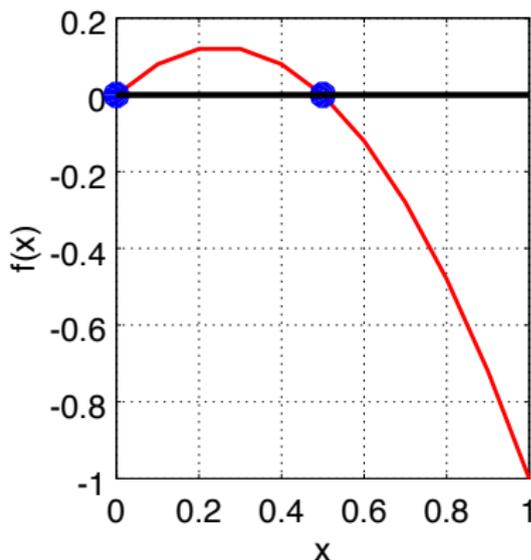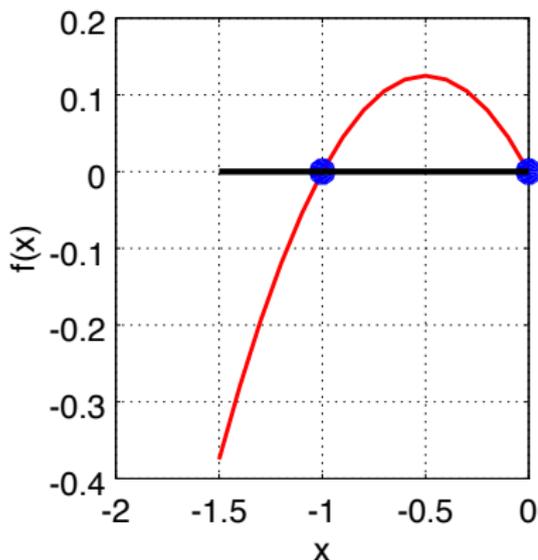
$f(x) = R_0 (1 - x) x - x$ versus x for the case $\boldsymbol{R_0 < 1}$ *(LEFT)* and $\boldsymbol{R_0 > 1}$ *(RIGHT)*. The equilibrium solutions $x = 0$ and $x = 1 - 1/R_0$ are clearly indicated by blue dots. We see that when $R_0 < 1$ *(LEFT)* the non-zero equilibrium solution is in the biologically infeasible region of $x < 0$. The value of R_0 used in the left plot was 0.5, corresponding with equilibrium $x = -1$, and the value of R_0 used in the right plot was 2, corresponding to equilibrium solution $x = 0.5$ (and $y = 0.5$).

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↪ In summary then, if $R_0 = (\beta N)/\mu < 1$ then the *disease-free* equilibrium solution $x = 0$ ($y = 1$) is stable while the *endemic* equilibrium solution $x = 1 - 1/R_0$ ($y = 1/R_0$) is unstable. HOWEVER note in this case that the *endemic* equilibrium solution $x = 1 - 1/R_0$ ($y = 1/R_0$) is not biologically realistic since $x < 0$, so we can ignore this case.

↪ Meanwhile, if $R_0 = (\beta N)/\mu > 1$ then the *disease-free* equilibrium solution $x = 0$ ($y = 1$) is unstable while the *endemic* equilibrium solution $x = 1 - 1/R_0$ ($y = 1/R_0$) is stable.

▶ Thus at the threshold value of 1, the basic reproductive ratio $R_0 = (\beta N)/\mu$ causes a very dramatic shift in the progress of the disease:

– If $R_0 < 1$ then each infected individual infects, on average, fewer than one other individual hence after an initial time the disease dies out. *The disease cannot "reproduce" fast enough to survive.*

– If $R_0 > 1$ then each infected individual infects more than one other individual hence the disease becomes endemic in the population. *The disease "reproduces" quickly enough to survive in the long term.* NOTE that unless R_0 is massive or starts growing, there will still remain a certain proportion of the population which does not get the disease (in this model).

▶ So the importance of R_0 is clear. Much research goes into identifying this type of critical parameter in more complicated disease models.

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A Few Comments Related to R_0

$\hookrightarrow$ If we write $\dfrac{dx}{d\tau} = \dfrac{\beta N}{\mu}(1-x)x - x = R_0(1-x)x - x$ in the equivalent *(CHECK!)* form

$$\frac{dx}{d\tau} = (R_0 - 1)x\left(1 - \frac{x}{1 - 1/R_0}\right),$$

we recognise it as a **Logistic** ODE with intrinsic growth rate $r = (R_0 - 1)$ and carrying capacity $K = 1 - 1/R_0$

▶ Thus R_0 tells us whether or not the disease will spread and r tells us how quickly it will do so.

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SIR Epidemic

$\hookrightarrow$ The key difference between this and the SIS model is that infectious individuals who "recover" either acquire immunity from the disease or are otherwised no longer capable of catching or transmitting the disease (they may be dead or quarantined) and are therefore *Removed* from the population with regard to the disease progress.

$\hookrightarrow$ This pattern is typical of many chilhood diseases such as smallpox or measles; once one recovers from the disease, one is immune. There was also a similar pattern in the early days of the HIV virus (before medical breakthroughs) since people who got HIV typically died shortly afterwards hence did not infect others when they were *removed* from the disease cycle.

$\hookrightarrow$ If we assume the rate at which individuals are removed from the infected category is proportional to the population of the infected category, γI, then the relevant compartment model diagram is
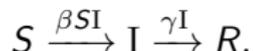
$$S \xrightarrow{\beta S I} I \xrightarrow{\gamma I} R.$$

REMINDER: $S \xrightarrow{\beta S \mathrm{I}} \mathrm{I} \xrightarrow{\gamma \mathrm{I}} R$.

$\hookrightarrow$ The corresponding system of differential equations is

$$\frac{dS}{dt} = -\beta S\mathrm{I}, \qquad \frac{d\mathrm{I}}{dt} = \beta S\mathrm{I} - \gamma \mathrm{I}, \qquad \frac{d\mathrm{R}}{dt} = \gamma \mathrm{I}.$$

$\hookrightarrow$ Observe that, again, the total population is constant:
$N = S(t) + \mathrm{I}(t) + R(t)$.

$\hookrightarrow$ We non-dimensionalise as before *(I'll leave the details to you)*.
Letting

$$u = \frac{S}{N}, \quad v = \frac{\mathrm{I}}{N}, \quad w = \frac{R}{N}, \quad and \quad \tau = \gamma t,$$

the dimensionless equations become

$$\frac{du}{d\tau} = -R_0 uv, \qquad \frac{dv}{d\tau} = (R_0 u - 1)v, \qquad \frac{dw}{d\tau} = v,$$

where $R_0 = (\beta N)/\gamma$ is again the intrinsic reproductive rate.

REMINDER: $\dfrac{du}{d\tau} = -R_0 uv$, $\quad \dfrac{dv}{d\tau} = (R_0 u - 1)v$, $\quad \dfrac{dw}{d\tau} = v$

$\hookrightarrow$ One can now use the fact that
$N = S(t) + I(t) + R(t) \Rightarrow u + v + w = 1$ to eliminate one of the
variables - for example w - and perform analysis on two of the
variables in a phase plane. This is done in many standard textbooks
on the subject - for example, Britton's `Essential Mathematical
Biology`. For variety, we will study the full system of 3 equations.

$\hookrightarrow$ The steady states are obtained by solving

$$-R_0 uv = 0, \quad (R_0 u - 1)v = 0, \quad v = 0.$$

▶ The last equation is only ever 0 when $v = 0$. The first equation is 0
when $u = 0$ or $v = 0$ and the second equation is 0 when $v = 0$ or
$u = 1/R_0$.

▶ So if $v = 0$ ALL three equations are 0 so the entire plane $v = 0$
(the $u - w$ plane) is effectively a steady state. Let us pause to
recover from this shocking news.

$\hookrightarrow$ At this point, it is worth noting that the region in which we can get biologically realistic solutions is a simplex (3d planar triangular region) given by
$\{(u, v, w) : 0 \leq u \leq 1, 0 \leq v \leq 1, 0 \leq w \leq 1, u + v + w = 1\}$. The intersection of this triagular plane with the plane $v = 0$ is the line segment $u + w = 1 \Rightarrow u = 1 - w$. *This is where we expect all of our feasible steady states to lie.*

▶ Thus there is no feasible steady state in which all three categories, Susceptible, Infectious, and Removed, co-exist (at least, not with this model).

$\hookrightarrow$ If you think about this, it makes sense: In the long term, the disease should be finished and there should only remain either susceptible people or people who have been removed (immune, quarantined, dead); there should be no more infectious people left.

▶ We can measure the size/impact of a disease by measuring the size of the Removed category, $w(\tau)$, once the steady state ($v = 0$) has been achieved.

$\hookrightarrow$ I will show time plots for several scenarios in which the SIR system is solved using the fourth order Runge-Kutta method. Initial conditions will be set so that $w = 0$ (hence the disease is just starting out with no removed individuals) and then u will be set to a large and then small value (so v will be small then large). I will also consider relatively small and large R_0 values less than 1 and relatively small and large R_0 values greater than 1. The table below summarises the different combinations of parameters in the following plots in the order in which the plots are presented:

R_0	u_0	v_0	w_0	R_0	u_0	v_0	w_0
0.05	0.8	0.2	0	0.05	0.2	0.8	0
0.5	0.8	0.2	0	0.5	0.2	0.8	0
1.5	0.8	0.2	0	1.5	0.2	0.8	0
15	0.8	0.2	0	15	0.2	0.8	0

NOTE time is denoted t in the following plots but should be interpreted as the dimensionless time τ.
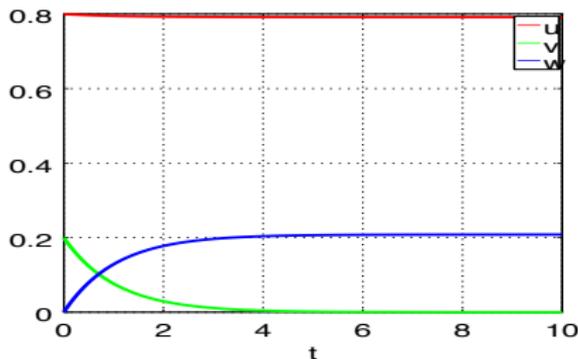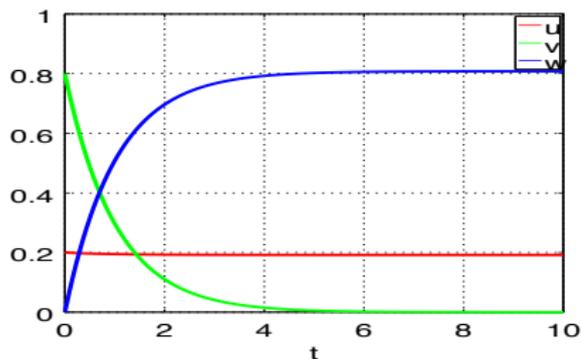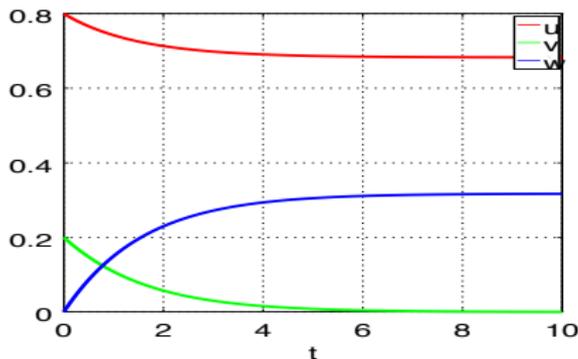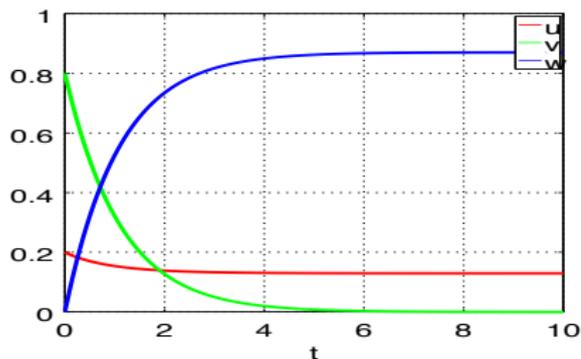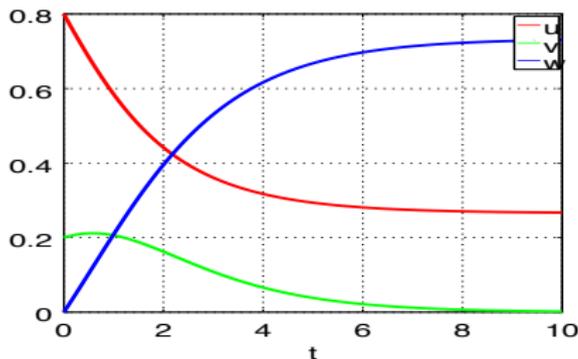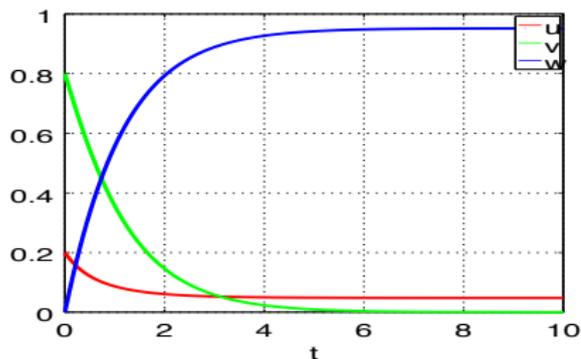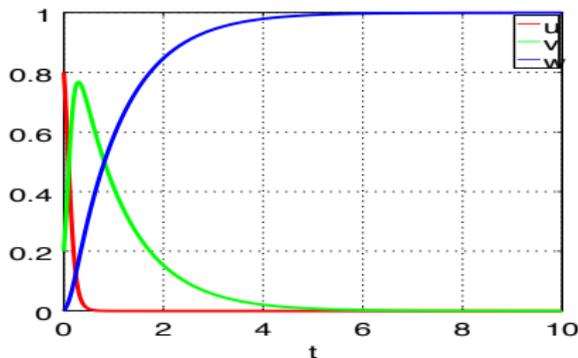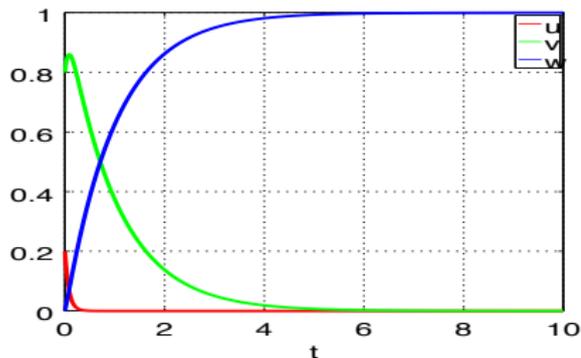
$\hookrightarrow$ The patterns of solutions are largely self-explanatory and logical. Some highlights are:

- ▶ As expected, the steady state for the infectious category is 0.
- ▶ It seems that there is only an epidemic (the number of infectious individuals grows at least for a time) when $R_0 > 1$. So again as in the SIS model the value of R_0, the basic reproductive ratio of the diseases, relative to 1 seems important.
- ▶ If R_0 is sufficiently large, even with a relative small initial infectious population, the whole population gets the disease (as measured by the steady state of the Removed population, w).

$\longmapsto$ We will now briefly analyse steady states with the aid of the Jacobian matrix of the transformation.

REMINDER: $\frac{du}{d\tau} = -R_0 uv, \quad \frac{dv}{d\tau} = (R_0 u - 1)v, \quad \frac{dw}{d\tau} = v$

$\hookrightarrow$ The Jacobian matrix is

$$J(u, v, w) = \begin{pmatrix} -R_0 v & -R_0 u & 0 \\ R_0 v & R_0 u - 1 & 0 \\ 0 & 1 & 0 \end{pmatrix}$$

$\hookrightarrow$ So at steady states, when $v = 0$, the Jacobian is

$$\begin{pmatrix} 0 & -R_0 u & 0 \\ 0 & R_0 u - 1 & 0 \\ 0 & 1 & 0 \end{pmatrix} \Rightarrow J - \lambda \mathrm{I} = \begin{vmatrix} -\lambda & -R_0 u & 0 \\ 0 & R_0 u - 1 - \lambda & 0 \\ 0 & 1 & -\lambda \end{vmatrix}$$

▶ From this, it is not too difficult to see that the eigenvalues are $\lambda_1 = 0$ and $\lambda_2 = R_0 u - 1$.

$\hookrightarrow$ Note we did not discuss zero eigenvalues in **Lecture 4**. The time progression of the disease in this model is more interesting than a detailed analysis of steady states, so for further discussion of this zero eigenvalue see other sources such as Example 3.2 of Nicholas Britton's book `Essential Mathematical Biology`.

$\hookrightarrow$ If we take $u = 1$ we have one of the many disease-free steady states and the eigenvalue $\lambda_2 = R_0 u - 1$ becomes $\lambda_2 = R_0 - 1$. This steady state is stable if $R_0 < 1$ but unstable if $R_0 > 1$. This unstable steady state suggests that the disease may not die out and an epidemic is possible. This analysis confirms what the earlier set of graphs of solutions, u, v, w, versus time showed.

Disease Control/Eradication/Vaccination

(This is largely a summary of the discussion in Section 3.5 of Nicholas Britton's
`Essential Mathematical Biology`*)*

$\hookrightarrow$ One of the key benefits of modelling the progress of diseases is that it gives the possibility of enacting policies which can cause the eradication of or at least control the spread of diseases.

$\hookrightarrow$ We have, for example, seen in the SIS and SIR models that having the basic reproduction ratio, $R_0 < 1$ can be crucial to suppressing epidemics and causing a disease to die out quickly, so strategies to reduce R_0 below a value of 1 are among those which can be deployed to control the spread of an infectious disease.

$\hookrightarrow$ Given that for closed models like the SIR model, $\boldsymbol{R_0 = (\beta N)/\lambda}$, there are three strategies to reduce R_0 and thus control such a disease.

$$R_0 = (\beta N)/\lambda$$

$\hookrightarrow$ In no particular order, those strategies are:

– Increase λ, which is the rate of removal of infectious individuals from the population. This could be done, for example, by quarantining infected individuals or culling infected animals (as was done to cows during the foot-and-mouth disease epidemic in 2001 in the UK).

– Decrease β, which is the pairwise infectious contact rate. Again this could be accomplished by quarantining infected individuals or restricting movement of individuals.

– Decrease the *effective* value of N. This does NOT mean killing of the population (although that would essentially work) but moving more people from the *susceptible* population in N into the *removed* category, with **vaccination** being one of the key ways in which this can be accomplished.

$\hookrightarrow$ We can use knowledge about the value of R_0 to determine what proportion, **p**, of a population would need to be (successfully) vaccinated to control the growth of a disease or to cause it to die off.

$\hookrightarrow$ NOTE vaccinating a fraction **p** of the (susceptible) population is equivalent to moving that fraction from the *susceptible* to the *removed* category, leaving the fraction $1 - p =$ **q** of the population in the *susceptible* category.

$\succ$ For example, in an SIR epidemic, the eigenvalue $\lambda_2 = R_0 u - 1$ would become $R_0 q - 1$ corresponding to a steady state of $(u, v, w) = (q, 0, p)$ in the disease-free equilibrium.

$\succ$ An epidemic might occur if that new disease-free steady state is unstable, meaning $R_0 q - 1 > 0$. On the other hand, for it to be a stable equilibrium, we require
$R_0 q - 1 < 0 \Rightarrow q < 1/R_0 \Rightarrow 1 - p < 1/R_0 \Rightarrow$ **$p > 1 - 1/R_0$**.

➢ In summary, if we vaccinate at least the proportion $p* > 1 - 1/R_0$ of individuals susceptible to an infectious disease in a population, we remove the threat of that disease becoming an epidemic.

↪ Here are some sample R_0 (hence threshold $p*$) values for selected infectious diseases (various sources). The data is largely for developed countries; R_0 is typically larger for developing countries.

Infectious Disease	R_0 Estimate	$p*$
Smallpox	$3 - 5$	$0.67 - 0.80$
Chickenpox	$9 - 10$	$0.89 - 0.90$
Measles	$12 - 18$	$0.92 - 0.94$
Malaria	> 100	> 0.99

End of Section

$\hookrightarrow$ There are many other disease models one can consider such as SIRS. See some books on the reading list for this course for more details. Two other useful sources (books) are:

- ▶ `Mathematical Models in Population Biology and Epidemiology`, second edition, by Fred Brauer and Carlos Castillo-Chavez *(Springer, 2011)*.
- ▶ `Mathematical Models for Communicable Diseases`, by Fred Brauer and Carlos Castillo-Chavez *(SIAM, 2013)*.

$\hookrightarrow$ Remember again you now have the tools and skills to explore *ANY* scenario modelled by a single or system of first order ordinary differential equations. Go forth and do so!