Mathematics 4MB3/6MB3 Mathematical Biology
2018 ASSIGNMENT 3

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This assignment was due in class on Wednesday 28 February 2018 at 11:30am.

Analysis of the standard SIR model with vital dynamics

Consider the standard SIR model with vital dynamics,

$$\frac{dS}{dt} = \mu N - \frac{\beta}{N} SI - \mu S \tag{1a}$$

$$\frac{dI}{dt} = \frac{\beta}{N} SI - \gamma I - \mu I \tag{1b}$$

$$\frac{dR}{dt} = \gamma I - \mu R \tag{1c}$$

where S, I and R denote the numbers of susceptible, infectious and removed individuals, respectively, and $N = S + I + R$ is the total population size. The *per capita* rates of birth and death are the same (both are equal to μ). As usual, β is the transmission rate and γ is the recovery rate.

(a) Prove that the population size N is constant and that equations (1) are biologically well-defined, *i.e.,* the set Δ of biologically meaningful states is forward-invariant. (Note that you will need to begin by defining precisely the set Δ and the term "forward-invariant".)

Proof. Summing the three equations we have

$$\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0\,, \tag{2}$$

implying that $N = S + I + R =$ constant. Note that this is really a two-dimensional system, since the $\dot{S}$ and $\dot{I}$ equations depend only on S and I (not R). The set of biologically meaningful states is the triangle

$$\Delta = \{(S, I) : S \geq 0,\ I \geq 0 \text{ and } S + I \leq N\}\,. \tag{3}$$

Proving that Δ is forward-invariant means that if $(S(0), I(0)) \in \Delta$ then $(S(t), I(t)) \in \Delta$ for all $t > 0$. To establish this, consider $\partial\Delta$ (the boundary of Δ). If points on $\partial\Delta$ do not leave Δ then Δ is forward-invariant. First consider points on the S axis, *i.e.,* where $I = 0$; on this set

$$\frac{dI}{dt} = 0\,, \tag{4}$$

hence I always remains non-negative. Similarly, on the I axis, where $S = 0$, we have

$$\frac{dS}{dt} = \mu N > 0\,, \tag{5}$$

so S always remains non-negative. Finally, suppose $S + I = N$, so the state lies on the diagonal segment of $\partial\Delta$. Then

$$\frac{d(S+I)}{dt} = \mu(N - S - I) - \gamma I \tag{6a}$$

$$= -\gamma I \leq 0\,, \tag{6b}$$

so $S + I$ is non-increasing and hence cannot exceed N. □

(b) Show that equations (1) are equivalent dynamically to equations for the proportions (rather than numbers) of individuals in each disease state. For the remainder of this problem, use the equations in proportional form.

Proof. From the previous part we know N is constant. Let $S' = S/N$, $I' = I/N$, and $R' = R/N$. Then

$$\frac{dS'}{dt} = \mu - \beta S' I' - \mu S' \tag{7a}$$

$$\frac{dI'}{dt} = \beta S' I' - \gamma I' - \mu I' \tag{7b}$$

$$\frac{dR'}{dt} = \gamma I' - \mu R' \tag{7c}$$

Since the equations are identical up to scaling, they yield the same dynamics. □

(c) Re-express the equations for proportions in *dimensionless* form using the dimensionless time coordinate

$$\tau = (\gamma + \mu)t\,, \tag{8a}$$

and the dimensionless parameters

$$\mathcal{R}_0 = \frac{\beta}{\gamma + \mu}\,, \tag{8b}$$

$$\varepsilon = \frac{\mu}{\gamma + \mu}\,. \tag{8c}$$

What are the biological meanings of τ, $\mathcal{R}_0$ and ε? Why are they good choices for non-dimensionalizing the equations? For a few diseases that you are familiar with, what is the order of magnitude of ε?

Solution. Dropping primes for convenience, we have

$$\frac{dS}{d\tau} = \varepsilon\big(1 - S\big) - \mathcal{R}_0 SI \tag{9a}$$

$$\frac{dI}{d\tau} = (\mathcal{R}_0 S - 1)I \tag{9b}$$

$$\frac{dR}{d\tau} = (1 - \varepsilon)I - \varepsilon R \tag{9c}$$

τ is time in units of the mean time an individual is infectious. $\mathcal{R}_0$ is the basic reproduction number, the average number of secondary cases resulting from a primary case in a wholly susceptible population[1]. (Hopefully everyone knows that by now!) ε is the mean time an individual is infectious as a proportion of the mean host lifetime. For typical childhood respiratory infections, $\varepsilon \ll 1$. It turns out, though it is certainly not obvious, that without an explicit latent period in the model, more accurate dynamics are obtained by considering $1/\gamma$ to be the mean *infected* period, opposed to the mean *infectious* period. With this in mind, we have for measles (which has a mean latent period of 8 days and mean infectious period of 5 days[2]),

$$\varepsilon \simeq \frac{13 \text{ days}}{75 \text{ years}} \simeq 4.7 \times 10^{-4}. \tag{10}$$

For other common respiratory infections, $10^{-4} \lesssim \varepsilon \lesssim 10^{-3}$. □

An  *note:* I did not type in the final value in equation (10). Instead, I let `knitr` do the computation and typeset it appropriately via `\Sexpr{signif(13/(75*365),2)}`.

(d) Show that there are exactly two equilibria: the disease free equilibrium (DFE) at $(S, I) = (1, 0)$ and an endemic equilibrium (EE) at $(S, I) = (\hat{S}, \hat{I})$, where $\hat{S}$ and $\hat{I}$ can be expressed compactly in terms of $\mathcal{R}_0$ and ε. Are both equilibria always biologically relevant?

Proof. The DFE, $(S, I) = (1, 0)$, always exists and is always biologically relevant. The EE is

$$\hat{S} = \frac{1}{\mathcal{R}_0}, \tag{11a}$$

$$\hat{I} = \varepsilon\left(1 - \frac{1}{\mathcal{R}_0}\right). \tag{11b}$$

If $\mathcal{R}_0 < 1$ then $\hat{I} < 0$, so this equilibrium can be relevant only if $\mathcal{R}_0 \geq 1$. Note, however, that if $\mathcal{R}_0 = 1$ then the EE coincides with the DFE. So assume $\mathcal{R}_0 > 1$, which implies

[1] See Anderson and May [1, Table 4.1, p. 70] for a list of $\mathcal{R}_0$ estimates for common diseases.

[2] See Anderson and May [1, Table 6.1, p. 129, Table 9.2, p. 181, and Table 14.2, p. 378] for lists of estimated latent and infectious periods for a variety of diseases.

$\hat{S} > 0$ and $\hat{I} > 0$. Further, note that

$$\hat{S} + \hat{I} \leq 1 \iff \frac{1}{\mathcal{R}_0} + \varepsilon\left(1 - \frac{1}{\mathcal{R}_0}\right) \leq 1 \tag{12a}$$

$$\iff \varepsilon\left(1 - \frac{1}{\mathcal{R}_0}\right) \leq 1 - \frac{1}{\mathcal{R}_0} \tag{12b}$$

$$\iff \varepsilon \leq 1 \quad (\because \mathcal{R}_0 > 1). \tag{12c}$$

Thus, the EE is biologically meaningful if and only if $\mathcal{R}_0 > 1$ and $\varepsilon \leq 1$. The condition $\mathcal{R}_0 > 1$ is natural, biologically, since it is the minimal condition for an infection to spread in the population. The condition $\varepsilon \leq 1$ says that the mean time infected must be no more than the mean lifetime, which is also biologically reasonable! Of course, the definition of ε [equation (**??**)] actually implies $\varepsilon < 1$ because $\mu \geq 0$ and $\gamma > 0$. □

(e) Show that the DFE is locally asymptotically stable (LAS) if $\mathcal{R}_0 < 1$ and the EE is LAS if $\mathcal{R}_0 > 1$.

Proof. At any point (S, I), the Jacobian matrix of the system is

$$J = \begin{pmatrix} -(\mathcal{R}_0 I + \varepsilon) & -\mathcal{R}_0 S \\ \mathcal{R}_0 I & (\mathcal{R}_0 S - 1) \end{pmatrix}. \tag{13}$$

At the DFE, $(S, I) = (1, 0)$, the Jacobian matrix is

$$J = \begin{pmatrix} -\varepsilon & -\mathcal{R}_0 \\ 0 & (\mathcal{R}_0 - 1) \end{pmatrix}. \tag{14}$$

Hence, the eigenvalues of J at the DFE are

$$\lambda_1 = -\varepsilon\,, \tag{15a}$$

$$\lambda_2 = \mathcal{R}_0 - 1\,. \tag{15b}$$

Both eigenvalues are negative if $\mathcal{R}_0 < 1$, so the DFE is LAS if $\mathcal{R}_0 < 1$, as was to be shown. Note, however, that $\lambda_2 = 0$ for $\mathcal{R}_0 = 1$, so the DFE is non-hyperbolic if $\mathcal{R}_0 = 1$ (and only if $\mathcal{R}_0 = 1$).

At the EE [equation (11)], the Jacobian matrix is

$$J = \begin{pmatrix} -\varepsilon\mathcal{R}_0 & -1 \\ \varepsilon(\mathcal{R}_0 - 1) & 0 \end{pmatrix}, \tag{16}$$

and the eigenvalues of J, are

$$\lambda_\pm = -\frac{\varepsilon\mathcal{R}_0}{2} \pm \frac{1}{2}\sqrt{\varepsilon^2\mathcal{R}_0^2 - 4\varepsilon(\mathcal{R}_0 - 1)} \tag{17}$$

λ_- always has a strictly negative real part, but this need not be so for λ_+. Indeed,

$$\lambda_+ = 0 \iff \frac{1}{2}\sqrt{\varepsilon^2\mathcal{R}_0^2 - 4\varepsilon(\mathcal{R}_0 - 1)} = \frac{\varepsilon\mathcal{R}_0}{2} \tag{18a}$$

$$\iff \sqrt{\varepsilon^2\mathcal{R}_0^2 - 4\varepsilon(\mathcal{R}_0 - 1)} = \varepsilon\mathcal{R}_0 \tag{18b}$$

$$\iff \varepsilon^2\mathcal{R}_0^2 - 4\varepsilon(\mathcal{R}_0 - 1) = (\varepsilon\mathcal{R}_0)^2 \tag{18c}$$

$$\iff -4\varepsilon(\mathcal{R}_0 - 1) = 0 \tag{18d}$$

$$\iff \mathcal{R}_0 = 1. \tag{18e}$$

Thinking through the above steps in reverse order is helpful for showing that $\mathcal{R}_0 > 1$ implies that $\Re(\lambda_+) < 0$. To begin with, note that

$$\mathcal{R}_0 > 1 \implies -4\varepsilon(\mathcal{R}_0 - 1) < 0 \tag{19a}$$

$$\implies \varepsilon^2\mathcal{R}_0^2 - 4\varepsilon(\mathcal{R}_0 - 1) < (\varepsilon\mathcal{R}_0)^2\,. \tag{19b}$$

There are two cases to consider at this point. First, suppose $\varepsilon^2\mathcal{R}_0^2 - 4\varepsilon(\mathcal{R}_0 - 1) \geq 0$. Then we can take the positive square root on both sides of inequality (19b) to obtain

$$\sqrt{\varepsilon^2\mathcal{R}_0^2 - 4\varepsilon(\mathcal{R}_0 - 1)} < \varepsilon\mathcal{R}_0 \implies \frac{1}{2}\sqrt{\varepsilon^2\mathcal{R}_0^2 - 4\varepsilon(\mathcal{R}_0 - 1)} < \frac{\varepsilon\mathcal{R}_0}{2} \tag{19c}$$

$$\implies -\frac{\varepsilon\mathcal{R}_0}{2} + \frac{1}{2}\sqrt{\varepsilon^2\mathcal{R}_0^2 - 4\varepsilon(\mathcal{R}_0 - 1)} < 0 \tag{19d}$$

$$\implies \lambda_+ < 0 \tag{19e}$$

Now suppose $\varepsilon^2\mathcal{R}_0^2 - 4\varepsilon(\mathcal{R}_0 - 1) < 0$. Then $\sqrt{\varepsilon^2\mathcal{R}_0^2 - 4\varepsilon(\mathcal{R}_0 - 1)}$ is a (pure) imaginary number and equation (17) implies

$$\Re(\lambda_+) = -\frac{\varepsilon\mathcal{R}_0}{2} < 0\,. \tag{19f}$$

Thus, the EE is LAS if $\mathcal{R}_0 > 1$, as was to be shown. □

Remark 1. *Note that equation* (18) *shows that the EE is non-hyperbolic for* $\mathcal{R}_0 = 1$*, the same value for which the DFE is non-hyperbolic. Furthermore, recall that if* $\mathcal{R}_0 = 1$ *then the EE is* $(\hat{S}, \hat{I}) = (1, 0)$*, identical to the DFE,* i.e., *the two equilibria collide at this point on the* $\mathcal{R}_0$ *parameter line. In addition, we can easily show that stability of the two equilibria is exchanged at this point, indicating a* transcritical bifurcation.

See, e.g., *page 149 of Guckenheimer and Holmes [2] for a brief discussion of transcritical bifurcations and the associated genericity conditions. Unlike a fold, a transcritical bifurcation can occur only if there are constraints of some sort in the system that force two distinct equilibria to collide (this is highly non-generic geometrically, since two random lines will not intersect in a space of dimension 3 or more). In the present case, the biological meaning of* $\mathcal{R}_0 = 1$ *makes clear the origin of the special condition: below this threshold an epidemic cannot take off, whereas above the threshold it is guaranteed to take off, hence the nature of the equilbrium must change from DFE to EE at this point.*

(f) Prove that the DFE is, in fact, globally asymptotically stable (GAS) if $\mathcal{R}_0 \leq 1$. *Hint:* This requires some careful analysis. Begin by using the function $L(S, I) = I$, and Theorem 2 stated in Assignment 1 under "Notes on Lyapunov Functions", to prove that all initial states in Δ are attracted to the S axis.

Proof. Observe that the bottom boundary of Δ, *i.e.,* the closed interval $[0, 1]$ on the S axis (on which $I = 0$), is a closed invariant set of the SIR model (9). Call this set $\mathcal{C}$. To see that $\mathcal{C}$ is invariant, note that if $I = 0$ then $dI/d\tau = 0$, so solutions that start in $\mathcal{C}$ must remain on the S axis; moreover, we know Δ is invariant, so solutions do not escape $\mathcal{C}$ by leaving the interval $[0, 1]$ on the S axis.

We can now apply Theorem 2 from Assignment 1 to the set $\mathcal{C}$, taking the open set $\mathcal{O}$ to be all of Δ, so the set that will be proved to be attracted to $\mathcal{C}$ is the remainder of Δ, *i.e.,* $\mathcal{O} = \Delta \setminus \mathcal{C}$. Verifying the hypotheses of Theorem 2 is easy for the set $\mathcal{C}$ and $L(S, I) = I$. Hypothesis (a) simply says "$I = 0$ if $I = 0$ and $I > 0$ if $I > 0$". Hypothesis (b) is slightly less trivial. We have

$$\dot{L} = \dot{I} = (\mathcal{R}_0 S - 1)I \,. \tag{20}$$

On the set $\mathcal{C}$, $I > 0$ so $S \leq 1 - I < 1$. Therefore, if $\mathcal{R}_0 \leq 1$ then $\mathcal{R}_0 S < 1$ so $\dot{L} < 0$. Thus, $L(S, I) = I$ is a Lyapunov function for $\mathcal{C}$ on all of Δ, so $\mathcal{C}$ is GAS, *i.e.,* every point of $\Delta \setminus \mathcal{C}$ converges to *some point* in $\mathcal{C}$.

That is progress, but we actually need to prove that all points of $\Delta \setminus \mathcal{C}$ converge to the *same point* in $\mathcal{C}$, namely the DFE, $(S, I) = (1, 0)$. To that end, consider the fate of initial states that lie in $\mathcal{C}$. Given $I(0) = 0$, the system [equation (9)] reduces to the one-dimensional equation

$$\frac{dS}{d\tau} = \varepsilon\big(1 - S\big) \,. \tag{21}$$

The solution of this equation is

$$S(\tau) = 1 - (1 - S_0)e^{-\varepsilon\tau} \,, \tag{22}$$

which implies $S(\tau) \to 1$ as $\tau \to \infty$ regardless of the initial proportion susceptible (S_0). Thus, all of $\mathcal{C}$ is attracted to the DFE.

Now, since all of $\Delta \setminus \mathcal{C}$ is attracted to $\mathcal{C}$ and all of $\mathcal{C}$ is attracted to the DFE, it follows (by continuity of the flow of a C^1 differential equation) that all of Δ is attracted to the DFE, *i.e.,* the DFE is GAS. □

(g) Prove that the EE is GAS if $\mathcal{R}_0 > 1$. *Hint:* Consider

$$L(S, I) = S - \hat{S}\log S + I - \hat{I}\log I \,, \tag{23}$$

and convince yourself that condition (a) in Theorem 1 stated in Assignment 1 under "Notes on Lyapunov Functions" can be replaced with

$$L(X) > L(X_*) \quad \text{for all} \quad X \in \mathcal{O} \setminus \{X_*\} \,. \tag{24}$$

Note: By GAS we mean here that *almost all* initial states are attracted to the EE. One way of making this precise is to say that the *basin of attraction* of the EE is an open, dense subset of Δ. You should completely describe the basins of attraction of both the EE and the DFE. Do your results make biological sense?

Proof. We will show L as defined in equation (23) is a strict Lyapunov function on an open set $\mathcal{O} \subset \Delta$ (in the sense of Theorem 1 from Assignment 1). First, to see that the first part of Theorem 1 can be replaced by the statement

(a′) $L(X) > L(X_*) \quad \forall X \in \mathcal{O} \setminus \{X_*\}$

note that we could define a new function $\tilde{L}(X) = L(X) - L(X_*)$ and use that in the theorem. Thus, our first step is to show that L has a global minimum (on some open set $\mathcal{O} \subset \Delta$) at $X_* = (\hat{S}, \hat{I})$. To that end, note that $L \in C^\infty(\mathbb{R}^2_+)$ and

$$\frac{\partial L}{\partial S} = 1 - \frac{\hat{S}}{S}, \tag{25a}$$

$$\frac{\partial L}{\partial I} = 1 - \frac{\hat{I}}{I}, \tag{25b}$$

so $\nabla L = 0 \iff (S, I) = (\hat{S}, \hat{I})$. Thus, L has a unique critical point in $\mathbb{R}^2_+$ at $(\hat{S}, \hat{I})$. This critical point must be a minimum because, for example,

$$L(S, \hat{I}) \to \infty \quad \text{as} \quad S \to \infty. \tag{26}$$

Continuing to restrict attention to $(S, I) \in \mathbb{R}^2_+$, consider

$$\dot{L} = \frac{\partial L}{\partial S}\frac{dS}{d\tau} + \frac{\partial L}{\partial I}\frac{dI}{d\tau} \tag{27a}$$

$$= \left(1 - \frac{\hat{S}}{S}\right)\Big(\varepsilon(1 - S) - \mathcal{R}_0 SI\Big) + \left(1 - \frac{\hat{I}}{I}\right)(\mathcal{R}_0 S - 1)I \tag{27b}$$

$$= (S - \hat{S})\Big(\varepsilon\frac{(1 - S)}{S} - \mathcal{R}_0 I\Big) + (I - \hat{I})(\mathcal{R}_0 S - 1) \tag{27c}$$

Now using $\mathcal{R}_0 = 1/\hat{S}$ and $\varepsilon = \hat{I}/(1-\hat{S})$, we have

$$\dot{L} = (S-\hat{S})\Big(\frac{\hat{I}(1-S)}{S(1-\hat{S})} - \frac{I}{\hat{S}}\Big) + (I-\hat{I})\Big(\frac{S}{\hat{S}} - 1\Big) \tag{27d}$$

$$= (S-\hat{S})\Big(\frac{\hat{I}(1-S)}{S(1-\hat{S})} - \frac{I}{\hat{S}}\Big) + (I-\hat{I})\Big(\frac{S-\hat{S}}{\hat{S}}\Big) \tag{27e}$$

$$= (S-\hat{S})\left[\frac{\hat{I}}{1-\hat{S}}\cdot\frac{1-S}{S} - \frac{I}{\hat{S}} + \frac{I-\hat{I}}{\hat{S}}\right] \tag{27f}$$

$$= (S-\hat{S})\left[\frac{\hat{I}}{1-\hat{S}}\cdot\frac{1-S}{S} - \frac{\hat{I}}{\hat{S}}\right] \tag{27g}$$

$$= (S-\hat{S})\frac{\hat{I}}{1-\hat{S}}\left[\frac{1-S}{S} - \frac{1-\hat{S}}{\hat{S}}\right] \tag{27h}$$

$$= (S-\hat{S})\frac{\hat{I}}{1-\hat{S}}\left[\frac{1}{S} - \frac{1}{\hat{S}}\right] \tag{27i}$$

$$= (S-\hat{S})\frac{\hat{I}}{1-\hat{S}}\left[\frac{\hat{S}-S}{S\hat{S}}\right] \tag{27j}$$

$$= -(S-\hat{S})^2\frac{\hat{I}}{(1-\hat{S})\hat{S}S} \tag{27k}$$

$$< 0 \quad \text{for all } (S,I) \in \mathbb{R}^2_+. \tag{27l}$$

If we now let $\mathcal{O} = \Delta \cap \mathbb{R}^2_+$, then $\mathcal{O}$ is an open subset of Δ and we can apply Theorem 1 to infer that all initial states in $\mathcal{O}$ converge asymptotically to the EE $(\hat{S}, \hat{I})$.

It remains to consider initial states that lie in the intersections of the S and I axes with Δ. If $S(0) = 0$, then at time $t = 0$ we have

$$\frac{dS}{d\tau} = \varepsilon > 0\,, \tag{28}$$

so the solution must immediately enter $\Delta \cap \mathbb{R}^2_+$ and then (by the above argument) converge to the EE. (We know that the solution must stay within Δ since Δ is forward-invariant.) If $I(0) = 0$ then the argument given in the previous part concerning the situation with $\mathcal{R}_0 \le 1$ applies without change, allowing us to conclude that the S axis is attracted to the DFE, not the EE.

Summarizing: If $\mathcal{R}_0 > 1$ then the basin of attraction of the DFE is the intersection of the S axis with Δ and the basin of attraction of the EE is the remainder of Δ. □

(h) Prove that the approach to the EE occurs via damped oscillations if and only if $\varepsilon < \varepsilon^*$, where

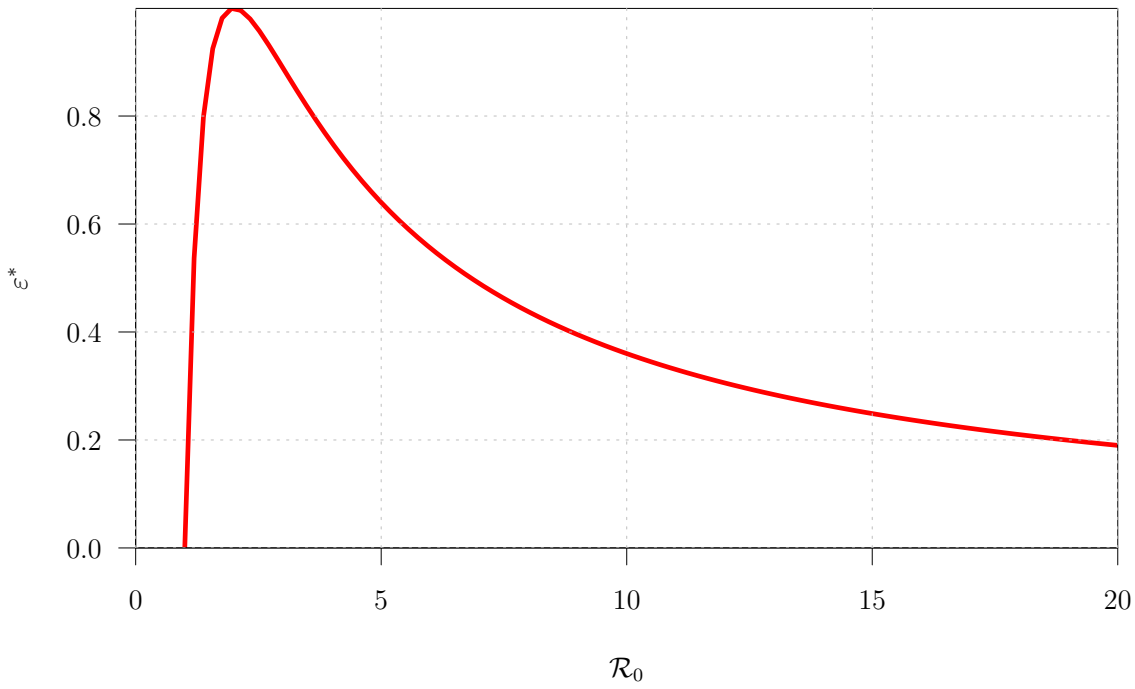
$$\varepsilon^* = \frac{4(\mathcal{R}_0 - 1)}{\mathcal{R}_0^2}\,. \tag{29}$$

For which diseases that you are familiar with would you expect damped oscillations versus monotonic convergence to the equilibrium?

Proof. The condition $\varepsilon < \varepsilon^*$ is easily derived from equation (17) by assuming the discriminant is negative.

The plot below shows that we can always expect damped oscillations for common respiratory infections (recall the comment immediately following equation (10)). The only situation in which we can expect *not* to have damped oscillations is if the infected period is of the same order of magnitude as the host lifetime, so for diseases like AIDS or TB (though for those diseases we might also wish to question the validity of the simple SIR model).

```
epsstarofR0 <- function(R0) {(4*(R0-1))/R0^2}
curve(epsstarofR0(x),1,20,xlim=c(0,20),lwd=5,col="red",xaxs="i",yaxs="i",
      las=1,xlab="$\\R_0$",ylab="$\\eps^*$")
grid(col="grey")
```



□

(i) For $\varepsilon < \varepsilon^*$, derive expressions for the period of damped oscillations onto the EE and the e-folding time for decay of the amplitude of oscillation. Use R to make a plot that

displays your results graphically for some biologically relevant and illustrative parameter values.

Solution. From equation (17), write

$$\lambda_{\pm} = -r \pm i\omega\,, \tag{30}$$

where

$$r = \frac{\varepsilon \mathcal{R}_0}{2} \tag{31a}$$

$$\omega = \frac{1}{2}\sqrt{4\varepsilon(\mathcal{R}_0 - 1) - \varepsilon^2 \mathcal{R}_0^2} \tag{31b}$$

Assuming $\varepsilon < \varepsilon^*$, the e-folding time is $1/r$ and the frequency of damped oscillations is ω (hence period of damped oscillations $2\pi/\omega$). If $\varepsilon \ll \varepsilon^*$, as for typical respiratory infections, then the second term under the square root can be neglected and we have

$$\omega \simeq \sqrt{\varepsilon(\mathcal{R}_0 - 1)}\,. \tag{31c}$$

Although not requested, note that if $\varepsilon \geq \varepsilon^*$ then the eigenvalues are purely real and the e-folding time for (monotonic) convergence to the EE is given by $|\lambda_-|$. In the graphs below, the time unit is the natural unit $1/(\gamma + \mu)$, *i.e.,* the mean time in the infected class. In the graph showing the damping period, the exact period is shown with a solid curve and the approximate period is shown with circles. Note that the approximation of the damping period is awful if $\varepsilon = 8/9$, as expected (the solid blue curve does not even overlap with with points plotted with blue circles). As discussed below in part (j), there are damped oscillations only if $1.5 < \mathcal{R}_0 < 3$ in this case. The damping period $\to \infty$ at the bifurcation points ($\mathcal{R}_0 = 1.5$ and 3), which is why R complains about the production of NaNs.

A few technical preliminaries make it easier to make clean plots of the e-folding time and damping period as a function of $\mathcal{R}_0$.

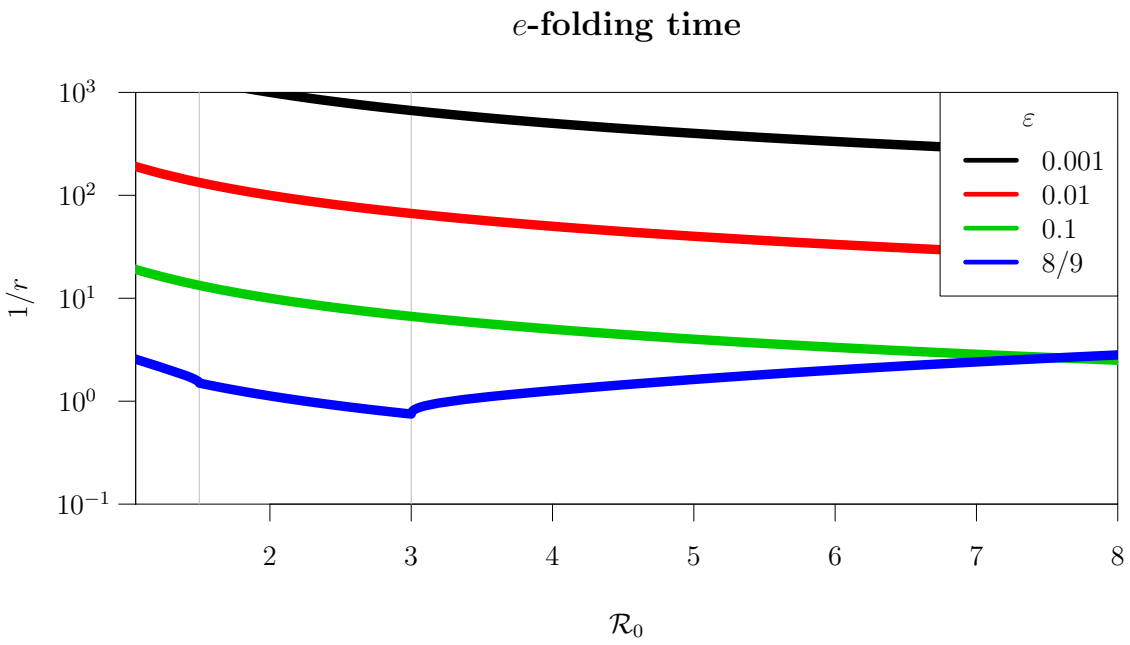
```
efoldtime <- function(R0,eps){
  discrim <- eps^2*R0^2 - 4*eps*(R0-1)
  return(as.vector(2/(R0*eps) + ifelse(discrim>0,(1/2)*sqrt(discrim),0)))
}
omega <- function(R0,eps) {
  discrim <- eps^2*R0^2 - 4*eps*(R0-1)
  return(as.vector(ifelse(discrim<0,(1/2)*sqrt(-discrim),0)))
}
omega.approx <- function(R0,eps) {sqrt(eps*(R0-1))}
## begin with a coarse set of R0 values:
R0.coarse <- seq(1.05,8,length=40)
## add more points where we need them:
R0 <- sort( c(R0.coarse, seq(1.01,1.1,length=100),
```

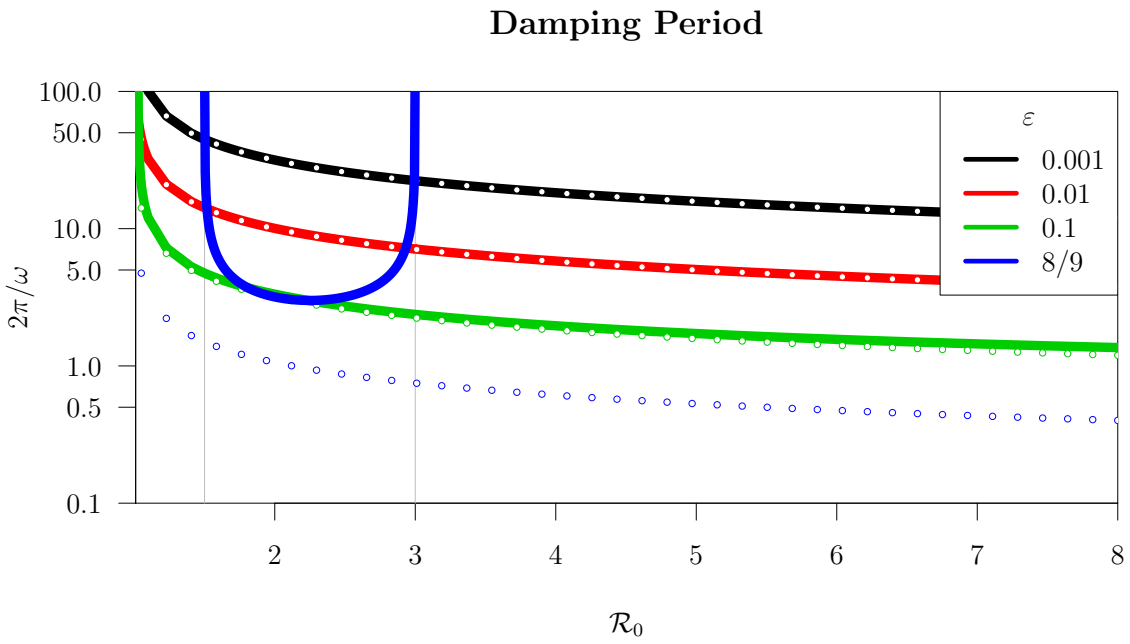
```
              seq(1.4,1.499,length=100),
              seq(1.5001,2.9999,length=300),
              seq(3.0001,3.1,length=100)
              )
            )
epsvalchar <- c("0.001", "0.01", "0.1", "8/9")
epsval <- c(0.001, 0.01, 0.1, 8/9)
epscol <- 1:length(epsval)
## This allows us to construct for() loops based on eps directly
## rather than via an index:
names(epscol) <- epsval
```

We align the two plots on the same graph so that corresponding values of $\mathcal{R}_0$ are evident when comparing e-folding time and damping period.

```
par(mfrow=c(2,1))
## e-FOLDING TIME PLOT:
plot(R0.coarse,efoldtime(R0.coarse,min(epsval)),typ="n",log="y",
     xlab="$\\R_0$",ylab="$1/r$",las=1,ylim=c(0.1,1000),xaxs="i",yaxs="i",
     main="$e$-folding time",yaxt="n")
axis(side=2,at=10^(-1:3), las=1,
     ## aligning nice-looking axis labels is a pain:
     labels=sprintf("$10^{%d}$%s",-1:3, ifelse((-1:3)>=0,"\\,","\\!\\!")))
abline(v=c(1.5,3),col="grey") # add vertical grey lines at R0=1.5, 3
for (eps in epsval) {
  lines(R0,efoldtime(R0,eps),lwd=10,col=epscol[as.character(eps)])
}

## Warning in sqrt(discrim):  NaNs produced
## Warning in sqrt(discrim):  NaNs produced

legend("topright",legend=epsvalchar,col=epscol[as.character(epsval)],lwd=5,
       bg="white",title="$\\eps$",cex=1)
## DAMPING PERIOD PLOT:
plot(R0,1/omega(R0,min(epsval)),typ="n",lwd=10,log="y",xaxs="i",yaxs="i",
     xlab="$\\R_0$",ylab="$2\\pi/\\omega$",las=1,ylim=c(0.1,100),
     main="Damping Period")
abline(v=c(1.5,3),col="grey") # add vertical grey lines at R0=1.5, 3
for (eps in epsval) {
  lines(R0,1/omega(R0,eps),typ="l",lwd=10,col=epscol[as.character(eps)])
  points(R0.coarse,1/omega.approx(R0.coarse,eps),pch=21,
         col=epscol[as.character(eps)],bg="white",cex=0.5)
}
```

```
## Warning in sqrt(-discrim):  NaNs produced
## Warning in sqrt(-discrim):  NaNs produced

legend("topright",legend=epsvalchar,col=epscol[as.character(epsval)],lwd=5,
       bg="white",title="$\\eps$",cex=1)
```





□

(j) Prove that as $\mathcal{R}_0$ is increased from 0 to ∞, three "bifurcations" occur. In addition, use R to make a four-panel plot that illustrates the different dynamics (phase portraits) in each of the four $\mathcal{R}_0$ intervals that have distinct dynamics. (*Hint:* I suggest you choose $\varepsilon = 8/9$ for this figure, but you should explain why this is a good choice.)

Theoretical note: The word "bifurcation" is in quotes above because many dynamicists would consider only one of the three transitions to be a genuine bifurcation (it happens to be a *transcritical bifurcation*). The other two dynamical transitions yield biologically relevant qualitative changes, but the phase portraits on either side of the "bifurcation point" are actually topologically conjugate.

R *note:* Computing phase portraits should be easy based on code you've written for solving ODEs in R "from scratch". However, if you wish, you can use the `phaseR` package (or another R package of your choice) to make the phase portraits.

Proof. The condition $\varepsilon < \varepsilon^*$ is equivalent to

$$\frac{2}{\varepsilon}\left(1-\sqrt{1-\varepsilon}\right) < \mathcal{R}_0 < \frac{2}{\varepsilon}\left(1+\sqrt{1-\varepsilon}\right) \tag{32}$$

so there are three bifurcations (two in addition to $\mathcal{R}_0 = 1$) if we fix ε and increase $\mathcal{R}_0$:

$$\mathcal{R}_0 = 1 \tag{33}$$

$$\mathcal{R}_0 = \frac{2}{\varepsilon}\left(1-\sqrt{1-\varepsilon}\right) \tag{34}$$

$$\mathcal{R}_0 = \frac{2}{\varepsilon}\left(1+\sqrt{1-\varepsilon}\right) \tag{35}$$

Note that

$$\lim_{\varepsilon\to 0^+} \frac{2}{\varepsilon}\left(1-\sqrt{1-\varepsilon}\right) = \lim_{\varepsilon\to 0^+} \frac{2}{\varepsilon}\left(1-\sqrt{1-\varepsilon}\right)\cdot\frac{1+\sqrt{1-\varepsilon}}{1+\sqrt{1-\varepsilon}} \tag{36}$$

$$= \lim_{\varepsilon\to 0^+} \frac{2}{\varepsilon}\frac{1-(1-\varepsilon)}{1+\sqrt{1-\varepsilon}} \tag{37}$$

$$= \lim_{\varepsilon\to 0^+} \frac{2}{\varepsilon}\frac{\varepsilon}{1+\sqrt{1-\varepsilon}} \tag{38}$$

$$= \lim_{\varepsilon\to 0^+} \frac{2}{1+\sqrt{1-\varepsilon}} = 1 \tag{39}$$

so the lower bound in (32) increases from 1 to 2 as ε is increased from 0 to 1. The upper bound decreases from ∞ to 2 as ε is increased from 0 to 1. Thus, since ε is typically small in practice ($\ll 0.01$) the only practically important bifurcation is at $\mathcal{R}_0 = 1$ (*i.e.*, the second bifurcation occurs so close to $\mathcal{R}_0 = 1$ that it appears to occur at $\mathcal{R}_0 = 1$ and the third bifurcation occurs at absurdly large $\mathcal{R}_0$).

For the purpose of qualitative understanding, it is convenient to choose an unrealistic value of ε that yields numerically convenient values of the "bifurcation points". With $\varepsilon = 8/9$, the three points are $\mathcal{R}_0 = 1$, $\mathcal{R}_0 = 1.5$ and $\mathcal{R}_0 = 3$. Note that $\varepsilon \sim 1$ is

biologically absurd for most diseases, since it means the mean time infectious is of the same order as the mean lifetime.

Recall from part (e) that the only non-hyperbolic equilibrium is at $\mathcal{R}_0 = 1$, so the other two "bifurcation points" above do not correspond to topologically distinct phase portraits. They do, however, yield different behaviour of the system, since they determine ranges of $\mathcal{R}_0$ over which there are, or are not, damped oscillations in the approach to the EE (spiral vs node).

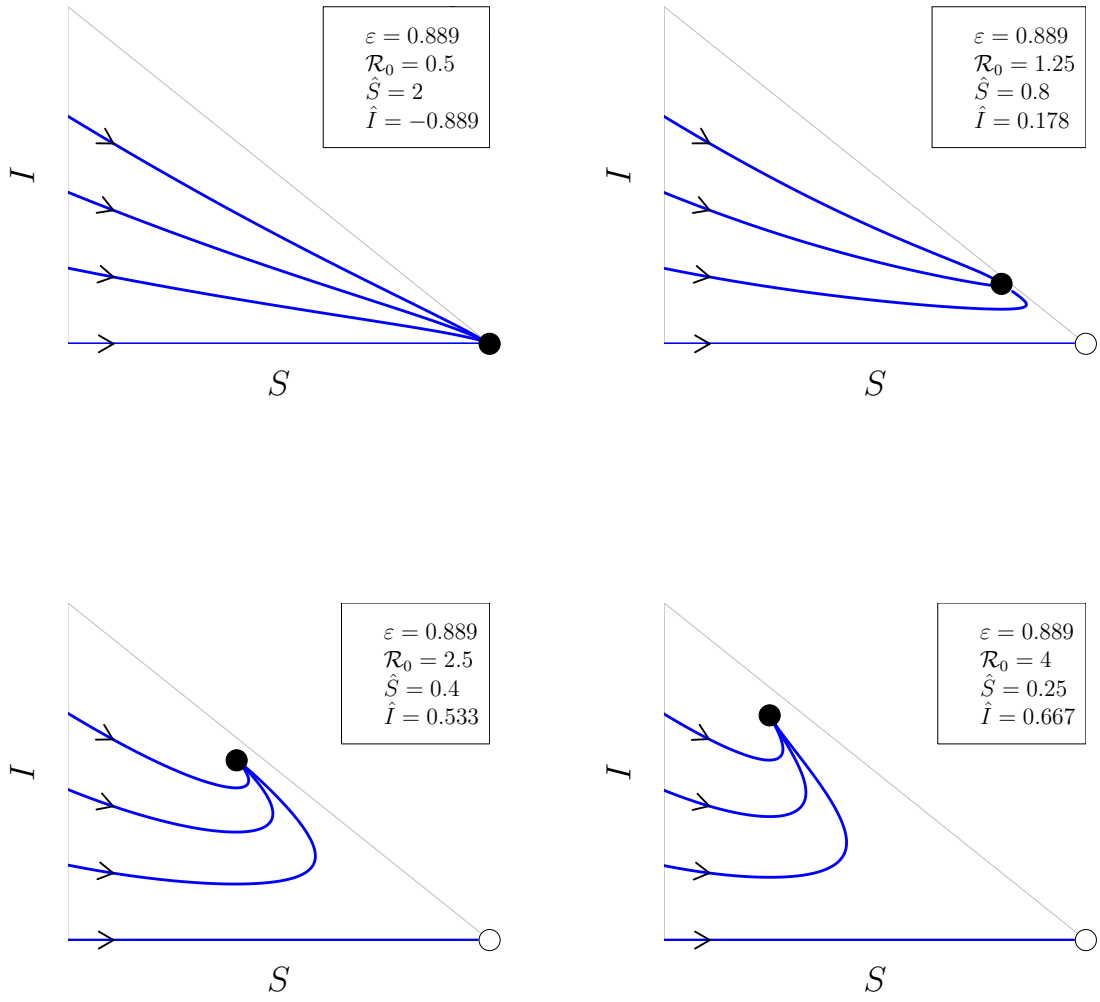
Since we need to draw four SIR phase portraits, it will be useful to have a function that plots a single SIR phase portrait.

```
source("phaseportrait.R")
draw.SIR.phase.portrait <- function(eps=8/9,R0=2,zoom=0,...) {
  ## calculate endemic equilibrium point:
  Shat <- 1/R0
  Ihat <- eps*(1-1/R0)
  if (zoom) { # zoom in around stable equilibrium point
    setup.type <- ""
    origin <- if (R0 > 1) c(Shat,Ihat) else c(1,0)
    rmax <- 1/zoom
  } else { # show all of Delta
    setup.type <- "positivei"
    origin <- c(0,0)
    rmax <- 1
  }
  draw.phase.portrait(func=SIR, parms=c(eps=eps,R0=R0),
                      setup.type=setup.type, origin=origin,
                      xlab="$S$",ylab="$I$",
                      rmax=rmax,
                      ictype="point", icpoint=c(0.1,0.6),
                      ...)
  abline(a=1,b=-1,col="grey")
  draw.phase.portrait(func=SIR, parms=c(eps=eps,R0=R0), add=TRUE,
                      ictype="point", icpoint=c(0.1,0.4))
  draw.phase.portrait(func=SIR, parms=c(eps=eps,R0=R0), add=TRUE,
                      ictype="point", icpoint=c(0.1,0.2))
  draw.phase.portrait(func=SIR, parms=c(eps=eps,R0=R0), add=TRUE,
                      ictype="point", icpoint=c(0.1,0))
  if (R0 > 1) {
    show.equilibrium(point=c(Shat,Ihat),cex=2,xpd=TRUE)
    show.equilibrium(point=c(1,0),type="unstable",cex=2,xpd=TRUE)
  } else {
    show.equilibrium(point=c(1,0),cex=2,xpd=TRUE)
  }
  legend("topright",bg="white",
```

```
          legend=sprintf("$%s=%g$",
            c("\\eps","\\R_0","\\Shat","\\Ihat"),
            signif(c(eps,R0,Shat,Ihat),3)) )
}
```

Now we can call the function just defined, once for each panel of our plot.

```
par(mfrow=c(2,2))
draw.SIR.phase.portrait(eps=8/9,R0=0.5)
draw.SIR.phase.portrait(eps=8/9,R0=1.25)
draw.SIR.phase.portrait(eps=8/9,R0=2.5)
draw.SIR.phase.portrait(eps=8/9,R0=4)
```



Note that for $\mathcal{R}_0 = 2.5$, we know the solution is a spiral sink (damped oscillations), but the oscillations are not evident in the phase portrait because they are too slow and damp out too fast (damping period about 5 mean infected periods, which is about 4.5 mean lifetimes, and e-folding time about 1 mean infected period; see figure in previous part).

□

(k) Are there real diseases that display recurrent epidemics for which the standard SIR model that you have studied in this problem might be adequate to explain the observed epidemic dynamics? If so, which diseases? If not, why not?

Answers. The infectious diseases that we have discussed display recurrent epidemics that do not damp out. Something else is needed in order to explain these diseases. However, as discussed in class, it is not necessary to start from scratch. We can build from the SIR model, adding seasonal forcing and demographic stochasticity. □

— END OF ASSIGNMENT —

References

[1] Anderson RM, May RM. Infectious Diseases of Humans: Dynamics and Control. Oxford: Oxford University Press; 1991.

[2] Guckenheimer J, Holmes P. Nonlinear Oscillations, Dynamical Systems, and Bifurcations of Vector Fields. vol. 42 of Applied Mathematical Sciences. New York: Springer-Verlag; 1983.

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