

Mathematical Modeling of Infectious Diseases, UCSF

Version 1.0—modified 13 Feb 2016

1 Quick review

We need a bit of calculus. The first derivative of the function f of the argument x (say), is defined as

$$\lim_{\Delta x \rightarrow 0} \frac{f(x + \Delta x) - f(x)}{\Delta x}.$$

There's mathematical fine print sometimes; the limit has to exist, for example.

Let's do one. Let $f(x) = x^2$, the simple parabola:

$$\frac{df}{dx} = \lim_{\Delta x \rightarrow 0} \frac{(x + \Delta x)^2 - x^2}{\Delta x}.$$

Then,

$$\frac{df}{dx} = \lim_{\Delta x \rightarrow 0} \frac{x^2 + 2x\Delta x + (\Delta x)^2 - x^2}{\Delta x},$$

$$\frac{df}{dx} = \lim_{\Delta x \rightarrow 0} \frac{2x\Delta x + (\Delta x)^2}{\Delta x},$$

$$\frac{df}{dx} = \lim_{\Delta x \rightarrow 0} (2x + \Delta x),$$

so

$$\frac{df}{dx} = 2x.$$

How about another one? Let $f(x) = x^n$, with n an integer (say).

$$\frac{df}{dx} = \lim_{\Delta x \rightarrow 0} \frac{(x + \Delta x)^n - x^n}{\Delta x}.$$

With the Binomial theorem: $(x + \Delta x)^n = \sum_{i=0}^n (\Delta x)^i x^{n-i}$, so

$$\frac{df}{dx} = \lim_{\Delta x \rightarrow 0} \frac{x^n + nx^{n-1}\Delta x + o(\Delta x) - x^n}{\Delta x}.$$

where $o(\Delta x)$ means something or other that has the property that $\lim_{\Delta x \rightarrow 0} o(\Delta x)/\Delta x = 0$ (stuff that goes to 0 faster than Δx does—for example, $k(\Delta x)^2$ where k is just some number that doesn't depend on Δx). So let's cancel:

$$\frac{df}{dx} = \lim_{\Delta x \rightarrow 0} \frac{nx^{n-1}\Delta x + o(\Delta x)}{\Delta x}.$$

$$\frac{df}{dx} = \lim_{\Delta x \rightarrow 0} \left(nx^{n-1} + \frac{o(\Delta x)}{\Delta x} \right).$$

So this is just nx^{n-1} .

We need two others: $\frac{d}{dx}e^x = e^x$, and $\frac{d}{dx}\log(x) = \frac{1}{x}$, but we won't prove these.

2 The Reed-Frost Process

We're going to now shift gears.

We've taken a good hard look at a classical stochastic model of an epidemic system, the Galton-Watson process. But it's missing something important. One thing it's missing is the idea of depletion of susceptibles. Another is the idea of change in behavior. People don't, for the most part, just mindlessly keep doing the same things in response to epidemic disease, but rather, change what they're doing to reduce their chance of getting the disease.

Remember the simple Galton-Watson model simply gave us

$$E[Y_{\tau+1}] = RE[Y_\tau]$$

where R is the reproductive number of the infection, i.e. the mean of the secondary case distribution. If $R > 1$ the mean of the epidemic simply increased without bound, as a geometric increase.

Exercise 1. Just in words, without writing any formulas or doing any math, explain how the mean can increase geometrically, even though sometimes the process goes extinct.

■

And we found that if $R < 1$, the mean geometrically declines to zero. But the stochastic analysis also gave us the fact that the probability of extinction is 1 whenever $R < 1$ but also whenever $R = 1$.

The equation $E[Y_{\tau+1}] = RE[Y_\tau]$ clearly shows that the mean goes to zero whenever $R < 1$, but on the critical case $R = 1$ the analysis of the mean alone doesn't give us much insight at all. Still, $R = 1$ is a special boundary case. It will often be valuable to just forego analysis of the random nature of the process, and just look at models where we have taken some sort of averaging process during the model development itself, leading to a *deterministic model*. The computational and analytical simplicity (relatively speaking) of such models often makes it worthwhile.

Now: the Galton-Watson analysis always assumed that each case could just make secondary cases from the same distribution, forever, till the bitter end of time if necessary. Today's goal is to lift that restriction. Soon we will need to move on to deterministic models as well as explore continuous time as well.

So let's get started. Let's consider the idea that the epidemic can "use up" susceptibles so to speak. So we will need to keep track of the number of susceptibles. Let's let X_τ be the number of susceptibles at time τ and Y_τ be the number of infectives.

In the Galton-Watson model we had each case give rise to a number of secondary cases, and we specified the distribution's probability generating function, g . Let's suppose we have the same thing now—almost. Before felt entitled to just use any old distribution we wanted, any count distribution at all supported on $0, 1, 2, \dots$. But this isn't all that great now, not now that we are trying to be crystal clear on the issue of the total count of susceptibles. You can't possibly have more new infections than there are susceptibles.

So let's approach this a different way. I'll show you one particular approach.

To start with, let's imagine that each case of disease lasts one time period, and that once you recover, you have recovered and you are totally immune. You are totally removed from the epidemic process. We're still going to follow generations the way we did with the Galton-Watson process, but we're going to toss the secondary case distribution g out the

window. Instead, focus on the susceptibles.

What we're going to have is a rule that is going to take the number of susceptibles and infectives now, X_τ and Y_τ , and generate from them the number of susceptibles and infectives next time, $X_{\tau+1}$ and $Y_{\tau+1}$.

Suppose we have a rule that says that *each* infective has a chance p of infecting *each* susceptible at any time period.

So what is the chance that a susceptible is infected? We have Y infectives and each of them has a chance p of doing the deed (causing infection). Remember the binomial risk models we looked at earlier? The logic is the same: the only way to *not* get infected is to not get infected by every single infective:

$$P(\text{not infected}) = (1 - p)^{Y_\tau}$$

So the risk of infection—chance a susceptible DOES get infected—is

$$r_\tau = 1 - (1 - p)^{Y_\tau}.$$

This changes over time. It depends on the number of infectives, which in general is changing.

Does this make sense? Let's check special cases. What if $Y_\tau = 0$? We would have $1 - (1 - p)^0 = 1 - 1 = 0$, so no infectives implies no risk. What if $p = 0$? Now we have $1 - (1 - 0)^Y = 1 - 1 = 0$; no transmission per infective, no risk.

So now, there are X_τ infectives, and each of them is at risk r . We assume independent and identical transmission risks from every infective to every susceptible. So here we have X Bernoulli trials, and each of them independent and identical. We know what to do with that:

$$P(Y_{\tau+1} = y) = \binom{X_\tau}{y} r_\tau^y (1 - r_\tau)^{X_\tau - y}.$$

and of course

$$X_{\tau+1} = X_\tau - Y_{\tau+1}.$$

whatever $Y_{\tau+1}$ happened to be.

So here we have a simple model of an epidemic that uses up its population. It's not hard to see that it must die out. There are only X_0 susceptibles; each time unit you either die out or use up at least one susceptible and there are only so many and they aren't replaced.

This particular model is called the *Reed-Frost* process in the literature.

Exercise 2. Assume $Y_0 = 1$, $X_0 = N - 1$. Compute the expected number of secondary cases for a single index case in an otherwise susceptible population. ■

Exercise 3. Suppose you wanted to allow superspreaders in the Reed-Frost model. How might you vary the model to allow this? ■

The Reed-Frost model is an example of a so-called chain binomial model.

Here is some R code to simulate the process. The function `reed.frost` takes the probability p as the first argument, the population size as the second, the initial number of infectives as the third, and finally the total amount of time.

```
reed.frost <- function(pp, nn, c1, t.end, cumul.only = FALSE) {  
  if (t.end > 1000) {  
    stop("t.end too big")  
  }  
  if (t.end <= 0) {  
    stop("negative or zero ending time")  
  }  
  if (c1 < 0 || abs(round(c1) - c1) > 1e-07) {  
    stop("invalid starting number of cases")  
  }  
  if (nn < 0 || abs(round(nn) - nn) > 1e-07) {  
    stop("invalid population size")  
  }  
  if (pp > 1 || pp < 0) {  
    stop("invalid transmission probability")  
  }  
}
```

```

}
if (nn < c1) {
  stop("more cases than people")
}
ss <- rep(0, t.end)
cc <- rep(0, t.end)
cumul <- 0
current.cc <- c1
current.ss <- nn-c1
if (!cumul.only) {
  cc[1] <- current.cc
  ss[1] <- current.ss
}
for (ii in 2:t.end) {
  rr <- 1-(1-pp)^current.cc
  current.cc.new <- rbinom(1,size=current.ss,prob=rr)
  current.ss.new <- current.ss - current.cc.new
  if (!cumul.only) {
    ss[ii] <- current.ss.new
    cc[ii] <- current.cc.new
  }
  current.ss <- current.ss.new
  current.cc <- current.cc.new
  cumul <- cumul + current.cc.new
  if (current.cc.new == 0) {
    if (!cumul.only) {
      for (jj in (ii+1):t.end) {
        ss[jj] <- current.ss
      }
    }
  }
}

```

```

    }
  }
  break
}
}
if (cumul.only) {
  cumul
} else {
  list(susc=ss,cases=cc,cumul.new.cases=cumul)
}
}

```

You can call it with the flag `cumul.only`, and have it just tell you how many infections there were. Otherwise, it returns a list containing the number of susceptibles over time (`susc`), the number of cases over time (`cases`), and the total outbreak size `cumul.new.cases`.

For fun, here is a function that will average a lot of different runs. As before, you can either use `cumul.only` to get the average final size, or you can average the replications.

```

reed.frost.average <- function(pp,nn,c1,t.end,nreps=1,cumul.only=FALSE) {
  if (cumul.only) {
    cumul <- reed.frost(pp,nn,c1,t.end,cumul.only)
    for (ii in 2:nreps) {
      cumul <- cumul + reed.frost(pp,nn,c1,t.end,cumul.only)
    }
    cumul/nreps
  } else {
    run <- reed.frost(pp,nn,c1,t.end)
    ss <- run$susc
    cc <- run$cases
  }
}

```

```

if (nreps >= 2) {
  for (ii in 2:nreps) {
    run <- reed.frost(pp,nn,c1,t.end)
    ss <- ss+ run$susc
    cc <- cc + run$cases
  }
}
list(susc=ss/nreps,cases=cc/nreps)
}

```

Let's do a few, with population size 64 say:

```

colrz <- c("black","red","blue","green","cyan","gray","magenta","orange")
multi.rf <- function(nsim,pp,nn,endtime,pcolors=colrz) {
  if (nsim<1) {
    stop("bad nsim")
  }
  ans <- reed.frost(pp=pp,nn=nn,c1=1,t.end=endtime)
  sims <- list(ans)
  for (jj in 2:nsim) {
    sims[[jj]] <- reed.frost(pp=pp,nn=nn,c1=1,t.end=endtime)
  }
  timez <- 1:endtime
  plot(timez,sims[[1]]$cases)
  points(timez,sims[[1]]$cases,type="l")
  for (ii in 2:nsim) {
    points(timez,sims[[ii]]$cases,col=pcolors[[(ii %% length(pcolors))+1]])
    points(timez,sims[[ii]]$cases,col=pcolors[[(ii %% length(pcolors))+1]],type="l")
  }
}

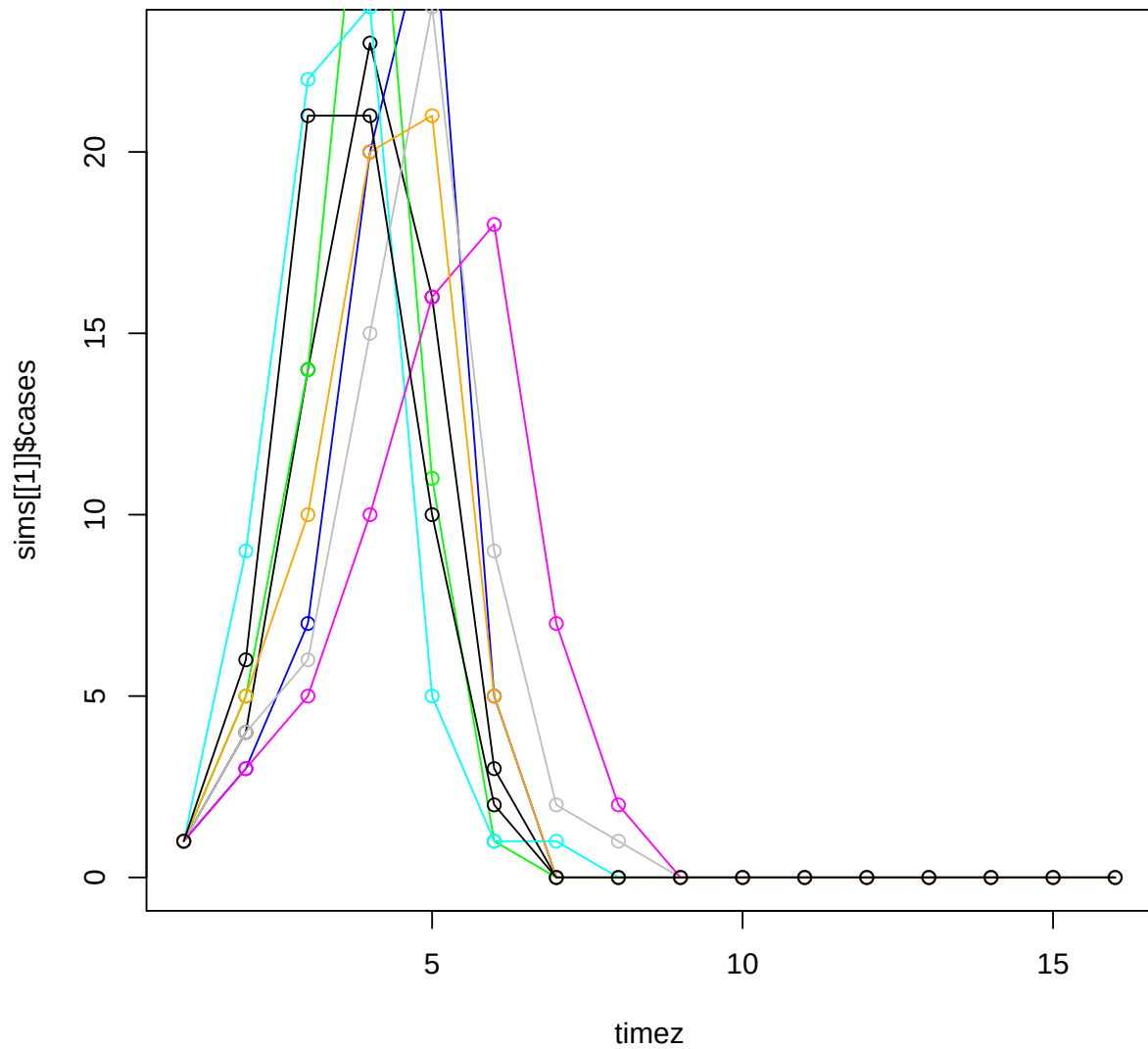
```



```

}
}
multi.rf(nsim=8,pp=1/16,nn=64,endtime=16)

```



Exercise 4. Repeat the above exercise for the same p ; can you find an epidemic where

it went extinct? Try it for smaller p , say for $p = 0.01$; try it for $p = 1/64$. Try different population sizes. What is the longest epidemic you see? What is the largest epidemic? How often do you see it drop near zero and then have a large epidemic? Try it dozens of times (but only turn in 3). You can use the `multi.rf` function. ■

3 Extending the model

The Reed-Frost process is a simple example of a so-called SIR model, because susceptibles become infective and then removed or recovered. But there is something a little special or unnatural about having the generations keep separated.

Let's take some steps to remedy this. How about this: allow, each time period, each infective to recover with probability q ? Now, you know that you will have a *geometric waiting time* until recovery. We're going to have to write it this way: let $\nu_\tau = \text{Binom}(X_\tau, r_\tau)$ be the random number of new infections.

$$Y_{\tau+1} = \text{Binom}(Y_\tau, 1 - q) + \nu_\tau,$$

with

$$X_{\tau+1} = X_\tau - \nu_\tau.$$

If we want to keep track of the recovered individuals Z , we can do it. Let $\zeta_\tau = \text{Binom}(Y_\tau, q)$. So

$$X_{\tau+1} = X_\tau - \nu_\tau$$

$$Y_{\tau+1} = Y_\tau - \zeta_\tau + \nu_\tau,$$

$$Z_{\tau+1} = Z_\tau + \zeta_\tau.$$

Now that we don't have to have the time interval match the length of infection or anything like that, we're free to let it become very small. Let's keep our head clear though:

$$X_{t+dt} = X_t - \nu_t$$

$$Y_{t+dt} = Y_t - \zeta_t + \nu_t,$$

$$Z_{t+dt} = Z_t + \zeta_t.$$

Let's pause a moment. This model is perfectly legitimate in its own right. It is a stochastic model in discrete time, and it makes sense for small dt . But we're going to move on and not explore it.

It does not make sense to allow the time step to become small, but p to stay the same. Instead, we're going to assume that $p = \beta dt$, so that the transmission probability were proportional to the time at risk. This does not work if dt is large.

Now, ν_t is a random variable. Let's approximate it with its mean, for a small time step. This is, at least in principle, a huge thing to do, but let's do it. The mean of ν is $X_t r_t$, but $r = 1 - (1 - p)^{Y_t}$. But this is $r_t = 1 - (1 - \beta dt)^{Y_t} \approx \beta Y_t dt$. So the mean of ν_t is $\beta X_t Y_t dt$. So:

$$X_{t+dt} = X_t - \beta X_t Y_t dt.$$

which we could write

$$X_{t+dt} - X_t = -\beta X_t Y_t dt.$$

Then, dividing by dt :

$$\frac{X_{t+dt} - X_t}{dt} = -\beta X_t Y_t.$$

If we take $dt \rightarrow 0$:

$$\frac{dX}{dt} = -\beta XY.$$

Here, I suppressed the subscript for time, t . We have gone from a discrete quantity, defined at various time steps, over to a continuous function of time.

Similarly, we could look at approximating the random variable ζ_t . We could say that the recovery probability q really equals a hazard ρ times dt . Then the mean of ζ_t would be $Y_t \rho dt$.

$$Y_{t+dt} = Y_t - Y_t \rho dt + \beta X_t Y_t dt.$$

which becomes

$$Y_{t+dt} - Y_t = -Y_t \rho dt + \beta X_t Y_t dt$$

and then

$$\frac{Y_{t+dt} - Y_t}{dt} = -Y_t \rho + \beta X_t Y_t$$

Finally,

$$\frac{dY}{dt} = -Y\rho + \beta XY.$$

The equation for Z is

$$\frac{dZ}{dt} = Y\rho.$$

At this point, we arrive at what is often called the *Kermack-McKendrick model* for an epidemic. In point of fact, the original paper of Kermack and McKendrick featured a fairly involved analysis of a more complex system, of which this is just a small special case.

Kermack-McKendrick model:

$$\frac{dX}{dt} = -\beta XY.$$

$$\frac{dY}{dt} = \beta XY - \rho Y$$

$$\frac{dZ}{dt} = \rho Y$$

Exercise 5. Let $N = X + Y + Z$. Assume the Kermack-McKendrick model. Prove $\frac{dN}{dt} = 0$; interpret. ■

A couple of things to notice: first, suppose we have a big population N , with 1 infective, all else susceptible. Then $X \approx N$. The second equation is then

$$\frac{dY}{dt} \approx Y (\beta N - \rho).$$

If $\beta N - \rho > 0$, then the time derivative of Y is positive. This means that Y is getting larger over time, right at the start. But if $\beta N - \rho < 0$, Y is decreasing. This is equivalent to saying if $\beta N/\rho > 1$, then Y increases at the beginning, and if $\beta N/\rho < 1$, then Y decreases at the beginning. Next time we will see that for this model, $\beta N/\rho = R_0$, the basic reproduction number.

Another thing: $\frac{dX}{dt} \leq 0$, always; the number of susceptibles can **never** increase at any instant. Intuitively, you are just using up susceptibles that can never be replaced.

Let's take a moment to look at what happens to our original cohort of infectives. Maybe this initial cohort is just $Y = 1$, a single person. A single person is kind of hard to interpret in this sort of deterministic model, where we have made Y a smooth continuous variable that can take any nonnegative value. (Not only did we go over to continuous time, we also moved over to a real valued variable!) For the *original cohort*,

$$\frac{dY^0}{dt} = -\rho Y^0.$$

There is no $+\beta XY$ or anything *right here*, because we're just looking at the original cohort of infectives. By definition, they just die away; we don't get any more. New infectives are not in the original cohort. Let's solve the equation:

$$\frac{dY^0}{Y^0} = -\rho dt$$

Integrate:

$$\int_{t=0}^{t=T} \frac{dY^0}{Y^0} = \int_{t=0}^{t=T} -\rho dt$$

$$\log(Y^0(T)) - \log(Y^0(0)) = -\rho(T - 0) = -\rho T.$$

$$\log(Y^0(T)/Y^0(0)) = -\rho T.$$

$$Y^0(T)/Y^0(0) = e^{-\rho T}.$$

$$Y^0(T) = Y^0(0)e^{-\rho T}.$$

This is a *very important* idea in the big picture. We found the original cohort *decays exponentially*. As time T gets larger, Y^0 goes to 0.

Note: the material for the next page or so logically belongs here, but will be covered next class.

Let's figure out how many new cases this first cohort can cause, in the otherwise completely susceptible population. I need to know the cumulative number of the next generation, the number of people newly infected by this first cohort. I don't need the actual size of the next cohort. The definition of R_0 is the number of new infections caused by an initial infection in an otherwise susceptible population. So: the number of *new* infectives resulting from the initial cohort follows

$$\frac{dU}{dt} = \beta XY^0.$$

But we're going to take $X \approx N$; that one infective shouldn't make much of a dent in X , at the beginning. So

$$dU = \beta NY^0 dt.$$

We know $Y^0(t) = Y^0(0)e^{-\rho t}$:

$$dU = \beta NY^0(0)e^{-\rho t} dt.$$

Integrate:

$$\int_{t=0}^{\infty} dU = \beta NY^0(0) \int_0^{\infty} e^{-\rho t} dt.$$

So:

$$U(\infty) - U(0) = \beta NY^0(0) \int_0^{\infty} e^{-\rho t} dt.$$

How many people have been cumulatively infected by the first generation of infectives at the beginning of the epidemic? None, by definition. So $U(0) = 0$, and

$$U(\infty) = \beta NY^0(0) \int_0^{\infty} e^{-\rho t} dt.$$

Now, we need to do the integral. Let $w = -\rho t$, so $dw = -\rho dt$ and the limits of integration become $w = 0$ to $w = -\infty$:

$$U(\infty) = \beta N Y^0(0) \frac{1}{-\rho} \int_0^{-\infty} e^w dw.$$

We know that the integral of e^w is e^w , so

$$U(\infty) = \beta N Y^0(0) \frac{1}{-\rho} (e^{-\infty} - e^0) = \beta N Y^0(0) \frac{1}{-\rho} (0 - 1) = \frac{\beta N Y^0(0)}{\rho}.$$

If $Y^0(0) = 1$, representing a single infective, we have

$$U(\infty) = \frac{\beta N}{\rho}.$$

So: a single infective, in an otherwise wholly susceptible population, will result in $\frac{\beta N}{\rho}$ new infections, and so this is why we can write $R_0 = \beta N / \rho$ for this model.

Every model will have different assumptions, and a different calculation for the basic reproduction number will have to be done. There is no single formula that just works for every model. The initial group of infectives dies away, but on the way, they cause a certain number of new infections.

Continuing then, with our analysis, we can see that intuitively, in the far future, you just shouldn't have any infectives. The ones you started with decay away; new infectives enter the Y box and are subject to the same process, and there are only a finite fixed number of people who can ever enter Y anyway. Epidemiologically, our instinct is that this process has to quieten down in the far future with no infections, but how do we know *these equations* actually behave that way? Well, we'll see next time. For the moment, however, please take for granted that in the far future, $Y = 0$ and $dY/dt = 0$.

We have $\frac{dX}{dt} = -\beta XY$. So $Y = -\frac{1}{\beta X} \frac{dX}{dt}$. So

$$\frac{dZ}{dt} = \rho Y = -\frac{\rho}{\beta X} \frac{dX}{dt}.$$

Integrate both sides:

$$\int_{t=0}^{t=T} \frac{dZ}{dt} = \int_{t=0}^{t=T} \left(-\frac{\rho}{\beta X} \frac{dX}{dt} \right)$$

$$Z(T) - Z(0) = -\frac{\rho}{\beta}(\log(X(T)) - \log(X(0))).$$

Assume there weren't any recovered/removed people at the beginning: $Z(0) = 0$.

Also, now divide both sides by N , the population size:

$$Z(T)/N = -\frac{\rho}{\beta N}(\log(X(T)) - \log(X(0))).$$

Call $Z(T)/N = z(T)$, the fraction who are removed. This is the fraction who were infected at some time.

$$z(T) = -\frac{1}{R_0} \log(X(T)/X(0)).$$

Rearrange a bit:

$$-R_0 z(T) = \log(X(T)/X(0)).$$

Then:

$$e^{-R_0 z(T)} = X(T)/X(0)$$

Let's also assume this: $X(0) \approx N$, so we're looking at basically everybody being susceptible at the beginning. We could write $X(T)/N = x(T)$ as the fraction susceptible.

$$e^{-R_0 z(T)} = x(T)$$

Let's go to the far future: $T \rightarrow \infty$:

$$e^{-R_0 z(\infty)} = x(\infty)$$

In the far future, you are either still susceptible, or recovered/removed, so $x(\infty) = 1 - z(\infty)$:

$$e^{-R_0 z(\infty)} = 1 - z(\infty).$$

And so

$$z(\infty) = 1 - e^{-R_0 z(\infty)}.$$

This is quite a strange formula in some ways. Think of it this way: we had $Z(\infty)$ people who were infected. They may not be infected at the end of time; they're removed. But they were infected at some point, or they wouldn't be Z s. Each person can infect R_0 other people in a fully susceptible population. So we had, all together, the capability of infecting $Z(\infty)R_0$ people. This much "infection capability" is spread over N people, so you have an average of $z(\infty)R_0$ exposures per person. Thinking of this as though these were distributed according to the Poisson distribution, the chance of having no "hits" or exposures is $e^{-z(\infty)R_0}$. The chance of having at least one such hit or exposure (one or more = **not zero**) is then $1 - e^{-z(\infty)R_0}$. So that is the fraction that ought to have been infected, and sure enough, the formula tells us that $z(\infty) = 1 - e^{-z(\infty)R_0}$.

This classic formula goes way back, and seems strange when you first see it. But it does relate the basic reproduction number R_0 to the final attack size of a simple epidemic. This **does** include exhaustion of susceptibles over the course of the epidemic, but it does not include other sources of change in transmission. It still assumes the people are mixing randomly and so on, so it is really only the first word, and not the last.

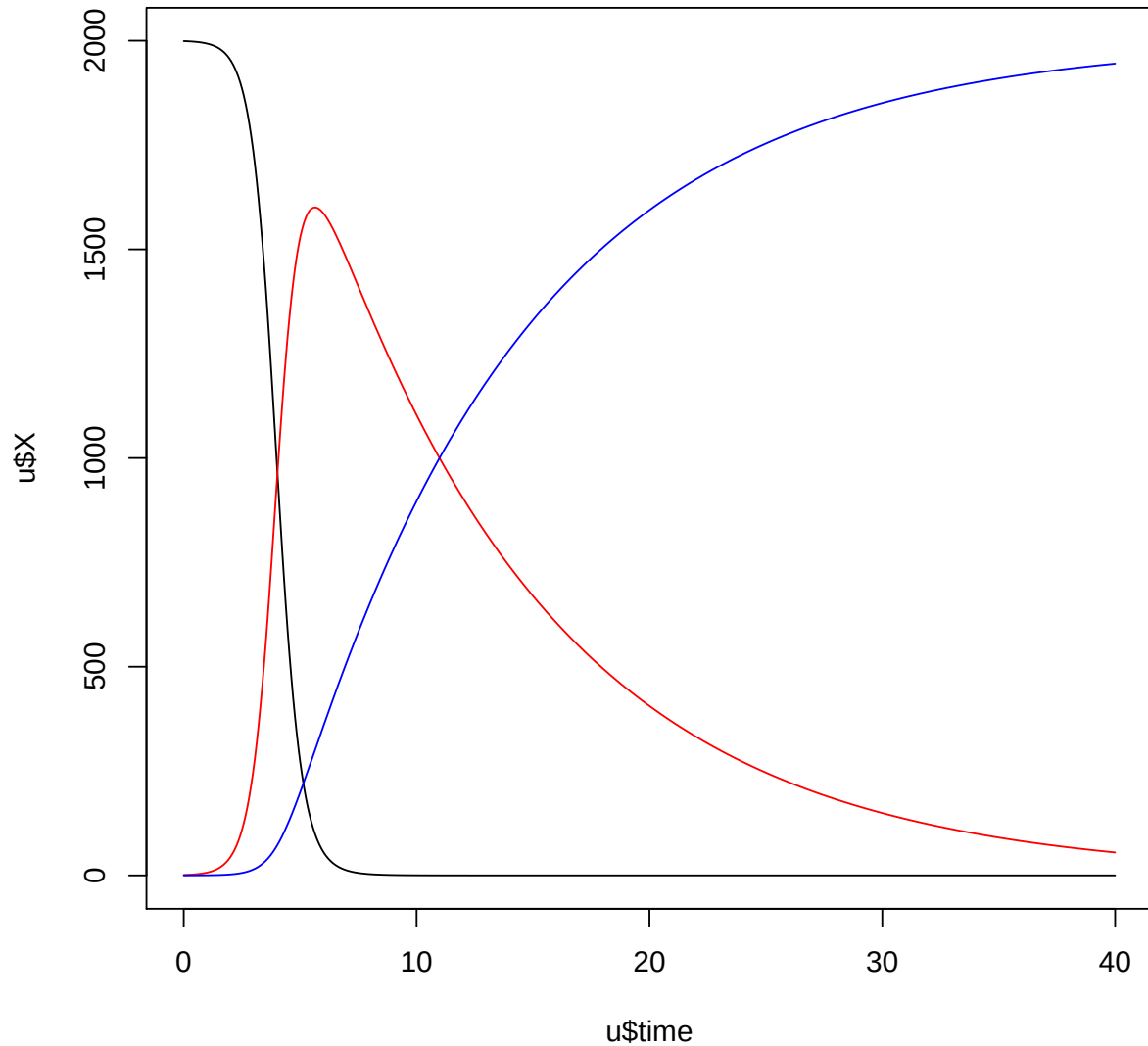
You will have to have the package `deSolve` installed in your R. Email if you have trouble with this.

```
require(deSolve)
km <- function(t,sys,params) {
  dsys <- sys[]
  c1 <- params[["beta"]] * sys[1] * sys[2]
  c2 <- sys[2]*params[["rho"]]
  dsys[1] <- -c1
  dsys[2] <- c1-c2
  dsys[3] <- c2
  list(dsys,NULL)
}
```

```

dosim <- function(pars,init,endtime) {
  tmp <- lsoda(init,seq(0,endtime,length=512),km,pars)
  ans <- as.data.frame(tmp)
  names(ans) <- c("time","X","Y","Z")
  ans
}
u <- dosim(pars=c(beta=0.001,rho=0.1),init=c(1999,1,0),endtime=40)
kmplot <- function(kmout) {
  plot(u$time,u$X,col="black",type="l")
  points(u$time,u$Y,col="red",type="l")
  points(u$time,u$Z,col="blue",type="l")
}
kmplot(u)

```



With the parameters I gave, $N = 2000$, and $R_0 = 0.001 * 2000 / 0.1 = 20$. We see an explosive epidemic that happens on the order of the timescale of a single infectious lifetime. In the plot, black is susceptible, red is infective, and blue is recovered.

Exercise 6. Play with the Kermack-McKendrick epidemic; vary β , vary ρ , vary N . To vary N , just change the sum of the number of people you use in `init`. You can do all your

explorations as one-liners:

```
kmplot(dosim(pars=c(beta=0.001,rho=0.1),init=c(1999,1,0),endtime=40))
```

where you just change β , ρ , etc. Do a bunch of them; make sure you look at one with $R_0 < 1$ and one with R_0 around 2 or so. Only turn in 3 graphs. ■

Exercise 7. How long did this assignment take you? ■