A Report That Will Change Your Life Forever... How an Infectious Disease spreads through a Population

June 1, 2010

(Equipped with pictures, cool models, and a little chaos theory)

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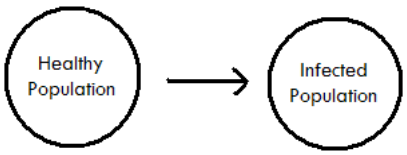
Abtract

In an effort to control the negative effect that disease can have an a population of people mathematicians and scientist try to tracks its progression of the disease as it spreads. They are able to do this by taking the characteristics of the disease, mapping the populations it could affect, and modeling it's progression using various techniques of solving complex differential equations. When mapping the spread of diseases, biologists can identify the steady states of the disease. They can also get an approximation as to the allotted time they have to prevent the epidemic or pandemic from reaching that point. This can be a difficult venture due to the wide variety of ways a disease can spread. We explore the various ways in which a disease can migrate, and the effectiveness modeling the disease is at predicting real life disease flow.

By modeling the movement of a particular disease, equipped with knowledge about all its relevant characteristics, we will simulate the movement of the disease in simplified models. Using simple models we should see how the different characteristics of a disease affect its how one models its progression. We will also notice the limitations that simple modeling has in predicting the outcome of complex, non-linear systems.

Introduction

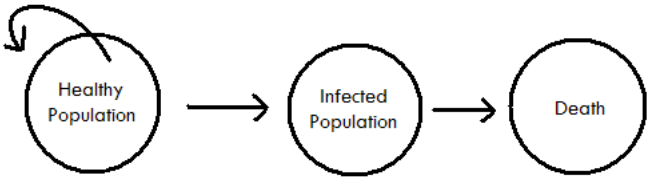
Disease can be defined as a type of illness that is caused by the infection of healthy (uninfected) people by disease causing pathogens. Different diseases have different characteristics that affect population dynamics. In general, diseases divide the total population into at least two subsets; infected and healthy.



The movement of members of the population into subsets (represented by arrows) of the total population occur from various dierences in the types of diseases and how they infect people. Disease may have any combiniation of these characteristics.

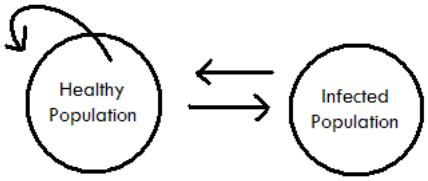
- It may be terminal:

  These diseases result in death, which remove people from the total population. Notice that the healthy population may reproduce new members into the population indicated by the curved arrow.



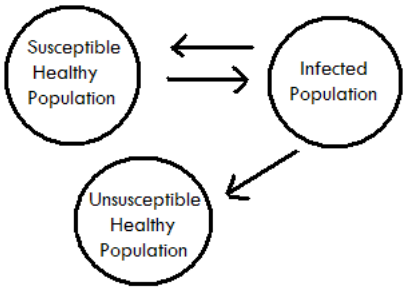
- It may be curable:

  These diseases have cures which enable members of the infected population to move back to the healthy population.



- It may be communicable:

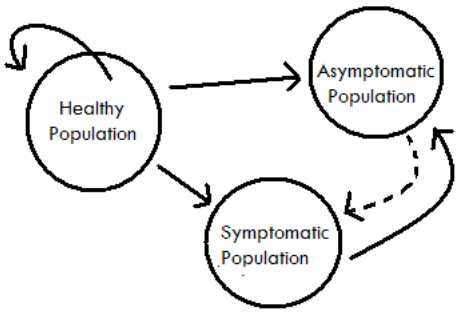
  These diseases spread through direct or indirect contact of the infected population with the suseptible population. In many cases, the entire healthy (uninfected) population is suseptible to infection. However, in some cases the healthy population are made of two subsets, susceptible and unsusceptible. The case of the unsusceptible population can arise from natural or articial immunities against an infection. This often occurs when an infected individual is cured or recovers, they may not be able to get the infection again due to a natural immunity.



- It may have Assymptomatic or Symptomatic populations:

  Diseases of this type divides the infected population into two subsets: asymptomatic and symptomatic. The symptomatic population are people who are infected by the disease and show signs of illness. Assymptomatic population are people who are infected but don't show any signs of illness.

Depending on the disease, members of the symptomatic population can move to the assymptomic population over some time or vice-versa.

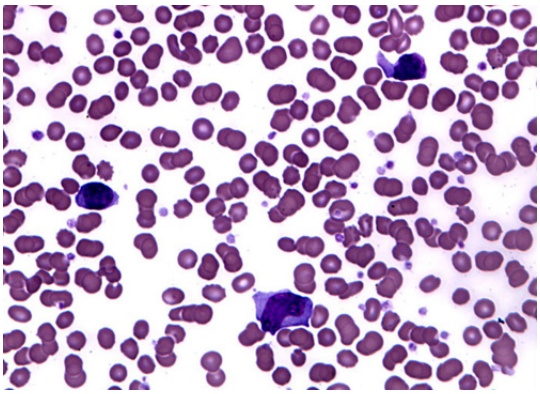


Infectious Mononucleosis

Mononucleosis (Mono) is a disease that has multiple characteristic which would aect the ow of the disease in the total population. Known as the kissing disease since it spreads through saliva, it is most common among young adults between the ages of 15-35. The most common symptoms among the symptomatic population is red and sore throat, headache, fever, nausea, abdominal pains, and white/red patched on tonsils.

It is a communicable disease, thus it spreads through the contact of the infected population with the suseptible population at the 15% infection rate after direct contact. However, after 12 to 18 months the diseases loses its ability to spread through contact makin it vurtually uncommunicable.

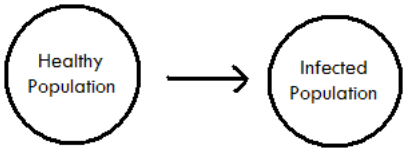
Also, in this disease there is no immunization thus the total healthy population is the suseceptible population. This disease has both a symptomatic and an assymptomatic population. The infected population is put into the symptomatic rst, then after a given time the infected members move to the asmyptomatic population. The infected population usually show symptoms 4 to 6 weeks before becoming asymptomatic. Finally, the rate of death due to Mono is very small, thus for the sake of simplicity we will consider it trivial enough to be disregarded.



Infected red blood cells look abmormal in this microscopic view of an infected individual's blood

Model Design and Implementation of Case 1

We will model the rst more general case of a disease similar to Mono. This diseases general case will be that of a disease which is communicable disease where infection spreads through the contact of the suseptible population with the infected. For the sake of simplicity we will disregard population growth, death, and the symptomatic/assymptomatic characteristics. We will focus on the communicable aspect of the disease (remember for Mono there is no recovery).



Let the total population be described as one, susceptible fraction of the population ($S(t)$), infected fraction of the popualtion ($I(t)$). We know $\beta_1 = .15$ is the rate of infection. In the general case the change in the infected population is a product of I and S:

$$I' = \beta_1(IS). \tag{1}$$

We know,

$$S + I = 1. \tag{2}$$

Thus,

$$I' = (1 - I)\beta_1 I. \tag{3}$$

Using Euler's Method of intertions we may estimate the progression of the disease over a year's time interval.

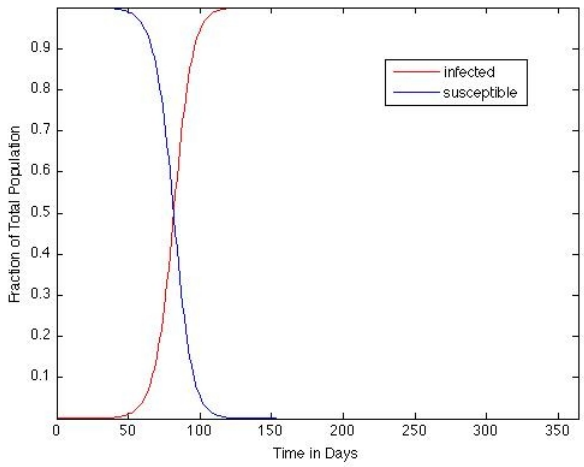
$$I_{n+1} = f(t_n, I_n)(\Delta t) + I_n, \text{ where } f(t, I) = (1 - I)\beta_1 I.$$

For the purposes of our real world population model, we assume initial conditions with one infected individual in a city where the population is about 100,000. We will use the MatLab code:

```
B=.15; days=365; dt=1;
y=zeros(days,1); %Infected Population
x=zeros(days,1); %Susceptible Population
t=zeros(days,1); %Time
x(1)=.99999; y(1)=.00001; t(1)=0.0;
for j=1:days-dt;
y(j+1)=y(j)+dt*B*(y(j)-y(j)*y(j));
x(j+1)=1-(y(j+1));
t(j+1)=t(j)+dt;
end
figure(1)
plot(t,y,'r')
hold on plot(t,x,'b')
legend('infected','susceptible')
xlabel('Time in Days'); ylabel('Fraction of Total Population');

axis tight
```

Then we obtain the plot:

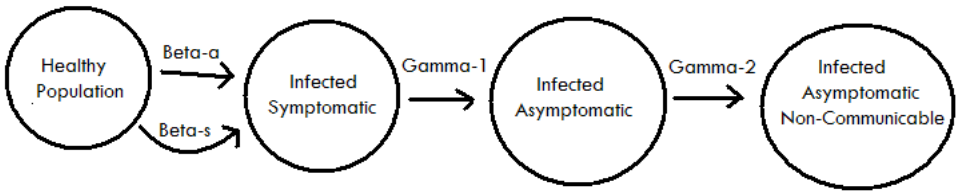


Notice that only after the first 100 days, the entire population moved from the healthy to the infected population. The rate at which the infected population grew was exponential.

Model Design and Implementation of Case 2

For Case 2 we will focus our model on more details about mononucleuosis in order to determine the affect that its assymptomatic behavior would have on the population.

Symptomatic infectious behaviour forces one to reinterpret the validity of β as the constant rate of illness. In the real world people who show signs of illness are often avoided by the general uninfected population to avoid getting infected. Thus, the real rate of infection for the symptomatic population, $\beta_s < \beta$ since it considers the propensity for healthy people to avoid sick people. However, existance of $\beta_s \Rightarrow \beta_a$; the rate of infection for asymptomatic people. Since the asymptomatic population doesn't look any dierent than the uninfected population, thier rate of contact with uninfected people would be slightly higher than the actual rate infection, $\beta_a > \beta$.



Let the total population be defined as one, infected symptomatic fraction of the population ($I(t)$), infected asymptomatic fraction of the population ($A(t)$), infected non-communicable fraction of the population ($N(t)$), suscpetible fraction of the population ($S(t)$), the rate of infection from the I population (β_s) is .03, the rate of infection from the A population (β_a) is .18, the rate of movement from I to A (γ_1) is 35^{-1}, and the rate of movement from A to N (γ_2) is 300^{-1}.

In this case we have the following system of equations and parameters:

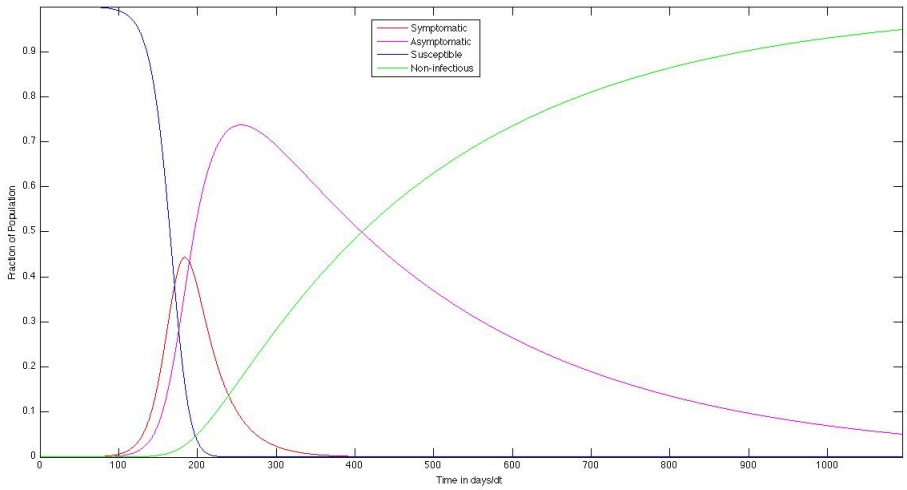
$$\begin{aligned} I' &= \beta_s IS + \beta_a AS - \gamma_1 I, \\ A' &= \gamma_1 I - \gamma_2 A, \\ N' &= \gamma_2 A, \\ S &= 1 - (I + A + N). \end{aligned}$$

We will use the following MatLab code in order to solve our system of differential equations:

```
Bs=0.03; Ba=0.18;
G1=1/35; G2=1/300;
days=365*3;
dt=0.01;
steps = days/dt+1;
t = (0:dt:days);
I=zeros(size(t)); A=zeros(size(t)); S=zeros(size(t)); N=zeros(size(t));
I(1)=0.00001; A(1)=0; S(1)=0.99999; N(1)=0;
for j=1:steps-1;
```

```
   I(j+1)=I(j)+dt*(Bs*I(j)*S(j)+Ba*A(j)*S(j)-G1*I(j));
   A(j+1)=A(j)+dt*(G1*I(j)-G2*A(j));
   N(j+1)=N(j)+dt*(G2*A(j));
   S(j+1)=1-(I(j+1)+A(j+1)+N(j+1));
   end
   figure(1)
   plot(t,I,'r')
   hold on plot(t,A,'m')
   plot(t,S,'b')
   plot(t,N,'g')
   legend('Symptomatic','Asymptomatic','Susceptible','Non-infectious');
xlabel('Time in days/dt'); ylabel('Fraction of Population');
   axis tight
```

We obtain the plot:



Conclusions

Notice that for the second model the progression of Mononucleosis follows the general movement predicted by Figure A. As time increased every member of the population moved to the non-infectous asymptomatic population. Both models of Mono accurately predict the real progression of the disease. As one may observe, many people are infected with the Mono disease just as decribed in model one. Of course, Model two seems to do a better job in painting a realistic picture of Mono since it has more parameters; notice that most people whom are infected are non-infectous asypmtomatic just as the second model predicted.

However, after examining the steady state one may releaze that despite the second model being more detailed, both cases have the same outcome.

If we set both A' and I'equal to zero we obtain $I(2\gamma_1 - \beta_s S) = A(\beta_a S + \gamma_2)$, and after we set N'equal to zero notice that $A = 0 \Rightarrow I = 0 \Rightarrow S = 0$. Since $S = 1-(I+A+N) \Rightarrow S = 1-N$, where S' is negative as it approaches zero, we essentially derive the same progression into the steady states as equations (1) and (2) predict from the first model. Ultimately, for both models the infected population will rise to one as the susceptible population falls to zero.

Despite the accuracy of the second model, its conculsions cannot be entirely real. The issue with the model is that it implys that the infection will die out over time, which of course is should not happen. This issue arises when a model does not account for population growth and migration. As the uninfectous population grows this allows for the susceptible population to grow as well due to the natural birth rate. As the susceptible population grows without infection, the chances for a random contact with an infectous individual either from the initial population or migrated from another population increases. Provided that the susceptible population has grown enough before encountering the infectous individ- ual, there is a chance (predicted by the log base 10 theory) of a random epidemic.

The log base 10 theory is an idea, discussed in the book "Deep Simplicity" by John Griffin, that attempts to explain the correlation between the magnitude and frequency of random or chaotic events. As the Magnitude of a choatic event like extinction, earthquakes, winning money, and epidemics increase the frequency of the event occuring at that magnitude falls by a power of ten. The frequency to magnitude ratio has been shown to follow close to the progression of log base 10. An easy experiment that is used to support the chaos theory is the frozen potatoe experiment. Once a frozen potatoe is shot at high velocity at a wall it explodes. The fragments of the potatoe come in various sizes that seem to follow the log base ten theory, one will find that the number of very small pieces are in the order of millions while the number of chuncks 1000 times bigger are in the order of hundreds, and one rarely finds one or two really big clumps of potatoe left.

These random epidemics would result in ambiguous spikes of various magnitudes in the infectous symptomatic population. This sort of chaos is masked by any model which maps the trends of the infection as fractions of the total population, since ideally any increase in the susceptible population would follow the same tread as the initial suscpetible population did. The actual population model of the ow of Mononucleousis is actually much more complex due to its non-linear nature, however both of the general models of the disease presented in this report do an accurate job of predicting the ow of the disease.

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