Research Article

The effect of infectiousness, duration of sickness, and chance of recovery on a population: a simulation study

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Abstract. Mathematical modeling has become a mainstream approach in scientific research when it is not feasible to design adequate experimentation. Agent based modeling provides rapid assessments of possible scenarios, especially in epidemiological studies where the goal is to carry out quick and effective preventive steps when an infection breaks out. In this paper, the relationships between infectiousness, chance of recovery, duration of a micro-parasitic disease, and the population dynamics of the host via parameter sweeps in a simple model system were analyzed. A disease with a very low infection rate is unable to spread in the population, unless the population density is high and the duration of the sickness is long. In cases of low recovery rates the number of sick and immune individuals showed a maximum value at 30% infectiousness. As the size of the population decreases, the population density decreases, and therefore, the transmission rate decreases as well. This effect leads to a new equilibrium population size, where the number of births and deaths will be balanced, and a large proportion of the population has acquired immunity. Based on these simulation models, the dynamic properties of populations contribute to the resilience of illness due to infections from micro-parasitic diseases.

Introduction

Worldwide about 1,415 human pathogens are known, of which 53% are micro-parasites (Cleaveland et al., 2001). These micro-parasites show a complex dynamic with their hosts. An infection begins when a virus or bacterium enters the host and replicates. The immune system attempts to stop the infection, and, if this fails, the host becomes sick and is then able to infect other individuals with the pathogen. Three main properties that control the transmission and life cycle of the virus are infectiousness, duration of the disease, and the chance of recovery of the host. These properties are important because they affect the dynamics of the infectious disease. Each of these properties, in combination with the others, lead to either a continuation or an extermination of the disease within a population. The interaction of the parameters (infectiousness, duration of the disease, and the chance of recovery) will predict how the disease may affect populations, and knowing this, preventive steps can be developed that hinder and stop the infection in the population.

Mathematical modeling has become a mainstream approach in scientific research when it is

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not feasible to design adequate experimentation. Mathematical modeling of infectious disease dates back to the 1920's when Kermack and McKendrick (1927) developed the SIR (Susceptible, Infectious, Recovered) model. The SIR model is a lumped, nonlinear, differential equation model in which all members of the population are in one of three stages of the disease, namely Susceptible (S), Infected (I), or Recovered (R). Infected individuals can transmit the disease to susceptible individuals before they recover (or die) and stop infecting others.

Since 1998, the use of models in scientific literature has increased more than four fold (Keeling and Rohani, 2007). The differential equation models with their aggregated formulation make a number of simplifying assumptions (homogeneity of the population, perfect mixing within each compartment, and the flow between states are expected values). Proponents of agent-based modeling argue that heterogeneity in networks of social contacts and in individual attributes, such as contact rates and infectivity along with the inherently stochastic nature of contacts among individuals, can make significant differences in the evolution of an epidemic (Cummings et al. 2004).

Modeling offers rapid assessments of possible scenarios. This is especially important when timely decision is required. Agent-based models are especially useful and versatile in providing a wide range of possible outcomes and implementing new rules rapidly (Hazir and Sterman, 2008). Models also can be refined after an outbreak comparing the prediction of the model and the collected data. The non-linear nature of the pathogen dynamics cannot be predicted by simple decision trees or step-by-step logical derivations because the complex relationship of parameters and the stochasticity of the processes involved. Models can be a powerful tool in this approach, allowing us to optimize the use of limited resources or simply to target control measures more efficiently (Keeling and Rohani, 2007). Insights gained from such simple models are often robust and generic, and therefore can be applied for developing an intuition for understanding the complexities of infection and disease dynamics.

In this paper, the relationships between infectiousness, chance of recovery, duration of the micro-parasitic disease and the population dynamic of the host were analyzed. Using a modified version of a simple agent based model developed by Wilensky (1998), a parameter sweep procedure was used to predict how population size and epidemic dynamics are affected by these parameters, especially: 1, how the successful invasion of the virus depends on the infectiousness and duration of the sickness and 2, how population size is affected by the recovery rate.

Material and Methods

A modified version of Wilensky's (1998) program called Virus was used in a NetLogo 3.13 simulation environment. The size of the habitat is a 37 by 37 grid in 1089 patches where the individuals move randomly and interact. Carrying capacity is set to 750 individuals which has an effect on the rate of reproduction since mating can only occur when the population size is less than the carrying capacity. When the population size is smaller than K, the reproduction rate is 0.074/year. Initial population size is 150 individuals, of which 10 are infected. Individuals are scheduled to die at 27 years, but they can die earlier due to the disease. Susceptible individuals can be infected; infected individuals can die in sickness or recover, where recovered individuals become immune and healthy again. In this model these recovered individuals will not lose their immunity.

The infectiousness, duration, and chance of recovery properties are used as main parameters in this study. The infectiousness concerns the ease with which a microparasite can infect its host. It gives the probability of transmission between infected and susceptible individuals who are on the same patch. The chance of recovery describes the likelihood that the infection will end in recovery. The duration determines the percent of the average life-span (1500 weeks) that an infected person goes through before the infection ends in either death or recovery. A duration of zero in the program produces a very short (2 weeks) infection period instead of a zero infection due to the order of commands that are handled in the simulation.

The duration of simulation was 60 years and the maximum and minimum values of the last complete fluctuation were recorded and averaged for all three subpopulations. Each parameter combination was repeated five times, and the mean and standard deviations were calculated from the runs. Instead of a full parameter sweep, our study was designed to sample some interesting regimes. For example, to evaluate the effect of infectiousness on the population, the chance of recovery set at 50 percent, duration at 20 weeks, and the degree of infectiousness was altered in increments of 10 percent.

Results

In the initial population density used in these simulations, 10 of the 150 individuals started as infected. In cases with low infectiousness rates (<8%), the disease quickly became extinct (Figure 1). With an infectious rate between 8 and 10 percent, the infection remained longer in the population but rarely survived until the end of the simulation. Due to the sensitivity to the random events, the longevity of the infection in the population varied within this range (Figure 1). However, when the infectiousness obtained 20%, the virus remained in the population the entire time. When the duration of the sickness was extended, the virus was able to survive in the population due to a smaller infectious rate, resulting in an S-shaped curve (Figures 2 and 3).

The infection resulted in a change of the population size and the mix of different population categories. As the infectiousness increases the number of healthy individual decreases and due to the infection-induced deaths the total population size decreases as well (Figures 4–6). Even in cases of aggressive infections (Infectiousness 99%, recovery rate 20%) the population will survive with an average of 52 healthy, 46 immune and 127 sick individuals. In cases of low recovery rates the population size of the sick (350.2 ± 9.74) and the immune (136.7 ± 6.00) individuals show a maximum value between 20 and 30 percent infectiousness. Larger infectiousness decrease the population size more dramatically due to a larger number of deaths, with each population category becoming smaller (Figure 4).

If the recovery rate was larger (50% or more) then the population size remained close to the carrying capacity, because the infection generated plus the age related deaths combined are smaller than the number of potential offspring. The size of the different population categories do not show a maximum value as the infectiousness increases, but a sigmoid curve can be observed (Figures 5 and 6). Beyond 30% of infectiousness the number of sick individuals did not increase considerably. However, the number of healthy individuals decreased as a function of the infectiousness, while the number of immune individuals increased (Figure 5). This trend was even more prominent in case of larger recovery rate

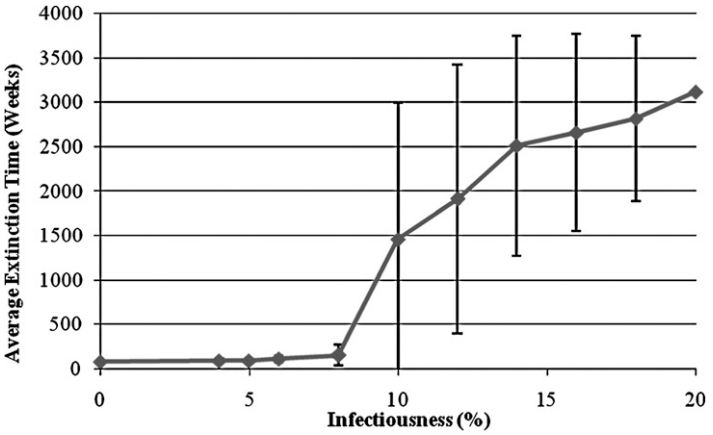

Figure 1. Average extinction time as a function of infectiousness (Duration of disease is 20 weeks). Mean values and standard deviations of 5 trials.

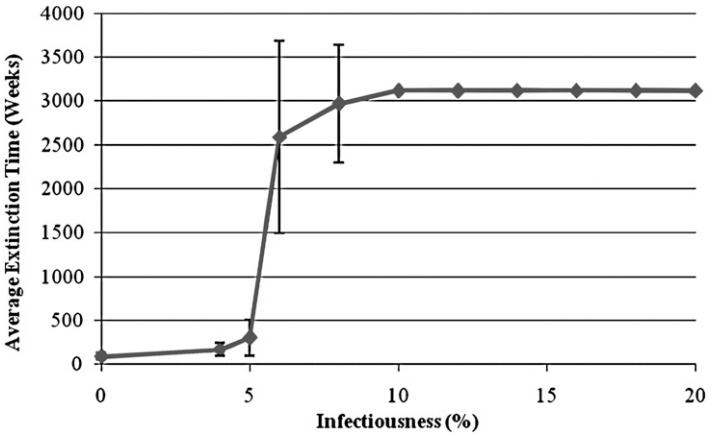

Figure 2. Average extinction time as a function of infectiousness (Duration of disease is 40 weeks). Mean values and standard deviations of 5 trials.

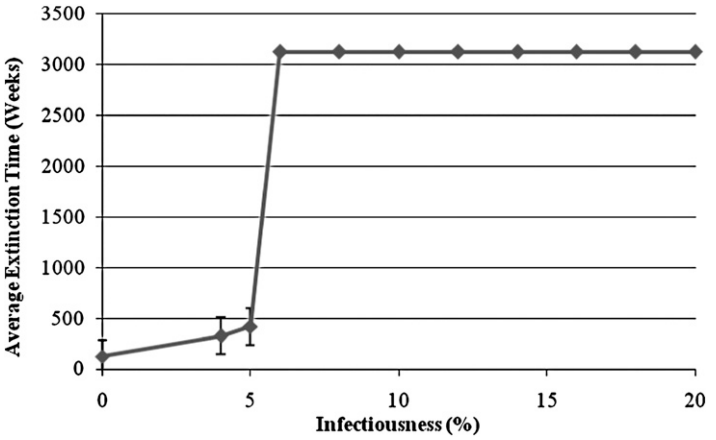

Figure 3. Average extinction time as a function of infectiousness (Duration of disease is 60 weeks). Mean values and standard deviations of 5 trials.

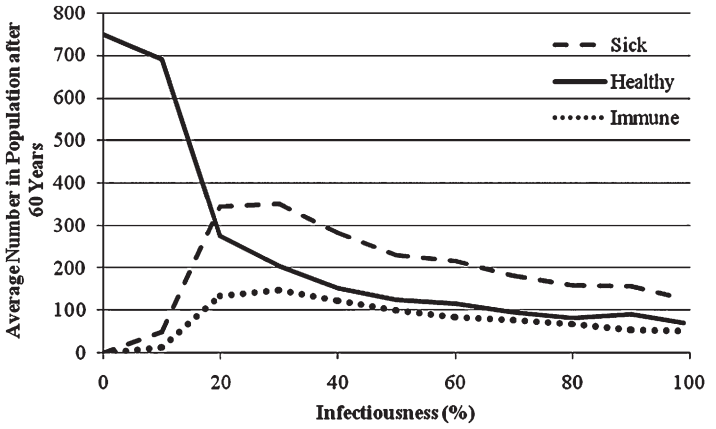

Figure 4. Average numbers (5 trials) of different population categories (sick, healthy and immune) as a function of infectiousness after 60 years from the initial infection if the chance of recovery is 20 %.

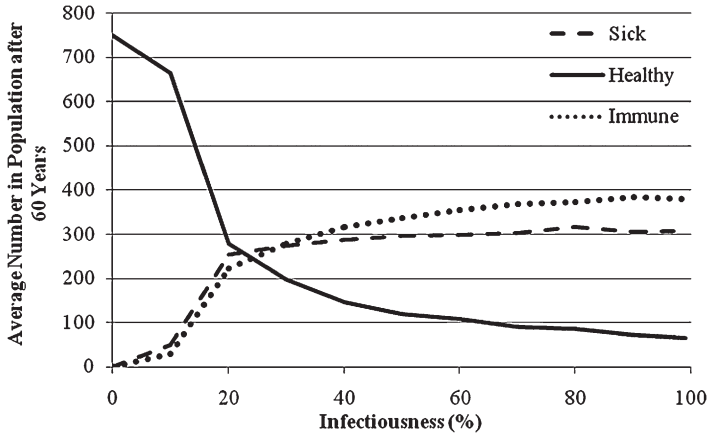

Figure 5. Average numbers (5 trials) of different population categories (sick, healthy and immune) as a function of infectiousness after 60 years from the initial infection if the chance of recovery is 50 %.

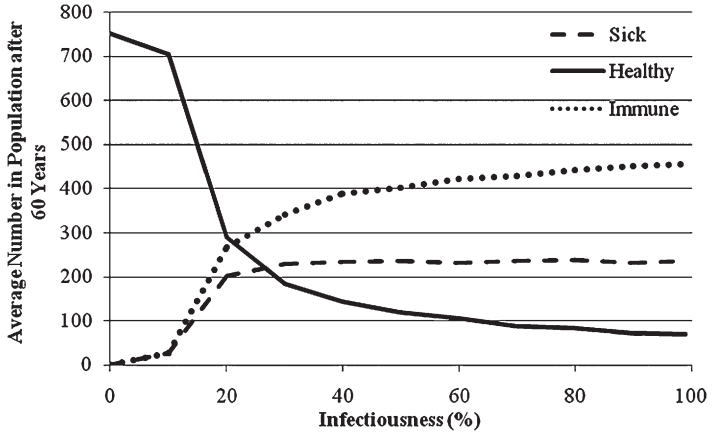

Figure 6. Average numbers (5 trials) of different population categories (sick, healthy and immune) as a function of infectiousness after 60 years from the initial infection if the chance of recovery is 80 %.

(80%) where the immune individuals comprised approximately 66% of the population in the case of high infectiousness (Figure 6).

Discussion

During the 2001 foot-and-mouth epidemic in the United Kingdom, three distinct models were used. However, due to the robustness of the problem, all three models provided similar prediction: A large-scale epidemic was predicted and additional, locally targeted culling would reduce the overall loss of livestock by dramatically reducing the number of cases (Keeling, 2005). The simple model used in this study has many general assumptions, but it provides robust general predictions. These types of generic models have an important role in understanding how an infectious disease spreads and how various complexities affect population dynamics (Keeling and Rohani, 2007). Our goal was to explore the relationship among the key parameters via simulations. Our aim was not a specific study of the epidemic of a given disease, but to explore general interdependencies of main parameters of the microparasitic disease to the population size. Models in general have two distinct roles, understanding and prediction (Keeling and Rohani, 2007). For example the failure to accurately predict epidemic in a particular area can

act as a diagnostic warning and in this case the models can be used as statistical tools (Stollenwerk and Jansen, 2003).

It appears that the infectious rate is not the only important factor. Diseases with low infectious rates are unable to spread in the population, except when population density is high and the duration of sickness is long. In the parameter ranges examined, the larger infectious rate (above 30%) did not increase the number of sick individuals. The long term response of the population was a relative increase of the immune population. This and the increased number of births kept the size of the sick population moderate even in case of high infectious rates. In case of low recovery rate the sick an immune population size showed a maximum value around 30% of infectiousness. Low recovery rate means that most sick individuals die and will not become immune. This has serious consequences on the number of births and there will be a breaking point when the number of deaths is larger than the number of births. However these populations will not become extinct, but they will survive below the carrying capacity of the population. As the size of the population decreases, the population density decreases and therefore the transmission decreases as well. This will result in a new equilibrium size, where the number of births and deaths will be balanced.

Our studies suggest that efficient control of the disease on the long term is an inherent property of the populations. Low density populations are resilient against mildly infective agents. High density populations increase the number of births and the percentage of immunes in the population as a countermeasure of the disease. Moreover as the population decreases in size and density, the number of contacts decreases as well and this in turn decreases the number of new infections. These long term population level responses suggest that in short term active control decreasing the population density and/or contacts paired with the increase of the number of immune individuals are effective way to combat the epidemic. It seems that the effective countermeasures of infection (vaccination, culling, decreasing number of contact) humans discovered were part of the natural population responses to these infective agents.

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