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SIR Models: Differential Equations that Support the Common Good

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Abstract: This article surveys how SIR models have been extended beyond investigations of biologically infectious diseases to other topics that contribute to social inequality and environmental concerns. We present models that have been used to study sustainable agriculture, drug and alcohol use, the spread of violent ideologies on the internet, criminal activity, and health issues such as bulimia and obesity.

1 Introduction

In 2015, UNESCO argued that "education and knowledge are common goods and represent a collective societal endeavor in a complex world based on respect for life and human dignity, equal rights, social justice, cultural diversity, international solidarity and shared responsibility for a sustainable future." [24] This article surveys how SIR models have been extended beyond investigations of biologically infectious diseases to other topics that contribute to social inequality. The goal of this paper is twofold. First, we show how the SIR model provides one lens to investigate a number of topics that broadly fall in the field of social justice. We present models that have been used to study sustainable agriculture, drug and alcohol use, the spread of violent ideologies on the internet, criminal activity, and health issues such as bulimia and obesity. Second, as an entry in a special issue dedicated to education and knowledge regarding differential equations and social justice, this paper also contributes to the common good as proposed by UNESCO.

The topics discussed here are by no means comprehensive but instead are intended to present some ways that SIR models give insight to policy makers and advocates who are interested in fostering equity. In each section, we describe the construction and conclusions of one model and then point the reader to papers investigating similar topics. The models presented get more complicated as the paper progresses.

The paper is organized as follows. We introduce the basic SIR system of differential equations described by Kermack-McKendrick in Section 2 and provide an example of how the model was used to describe the spread of smallpox on Easter Island in 1863. The basic model assumes a closed population and does not account for birth or immigration into the population. In Section 3, we present how the SIR system can be adapted to

study the spread of ideas. We focus on an application investigating the dissemination of violent topics on a terrorist web forum. This model is slightly modified from the original Kermack-McKendrick model in that it incorporates logistic growth of the population.

In Section 4, we present a model of disease transmission in plants. Clearly, this application is closely related to disease transmission in humans, but sustainable agriculture fits well with the ideals of fostering equity that are addressed in this special issue of the journal. In this model, the rate at which plants are infected can change over time. Section 5 presents a SIR model of heroin use in Ireland. This model incorporates different death rates for the susceptible population and the drug using population as well as the possibility that drug users can stop using drugs and not re-enter the susceptible population.

Section 6 describes a system in which a person's decision to start criminal activity is modeled as a socially contagious disease. This model splits the population into four groups, instead of the three groups of Kermack-McKendrick's model. Finally, Section 7 of the paper describes a model investigating whether obesity rates in the United States will plateau by using a model that splits the population into five different subgroups.

2 Kermack-McKendrick's SIR Model

Kermack-McKendrick [14] introduced the SIR system of differential equations to model the spread of infectious diseases. They applied their analysis to plague deaths in Bombay in 1905-1906. In their study, they let $S(t)$, $I(t)$, and $R(t)$ respectively be the proportion of the population susceptible, infectious, and recovered at time t. (By "recovered," we simply mean that the individual has been removed from the infectious population by recovery or death.) The SIR system is defined by the equations

$$\begin{aligned} \frac{dS}{dt} &= -bSI, \\ \frac{dI}{dt} &= bSI - kI, \\ \frac{dR}{dt} &= kI. \end{aligned} \tag{2.1}$$

The number b represents the rate at which the disease is transmitted when an infected individual interacts with susceptible individuals. The first differential equation in Equation 2.1 describes how individuals move from the susceptible group to the infected group. The constant k represents the proportion of the infected group that will recover each day, which is the reciprocal of the infectious period of the disease. So the second equation in Equation 2.1 describes how the infected group increases as susceptible people become sick but decreases as infected people recover. Finally, the recovered group grows as people move from the infected group to the recovered group.

As an example, the SIR model was used in [15] to model the spread of smallpox on Easter Island in 1863. At that time, there were approximately 2700 susceptible people living on the island when between 1 and 15 people infected with smallpox arrived. Using historical data for smallpox transmissibility and contagion length, estimates of $0.14 \leq b \leq 0.85$, $1/25 \leq k \leq 1/20$, and $1/2700 \leq I(0) \leq 15/2700$ were used to simulate the epidemic. In all

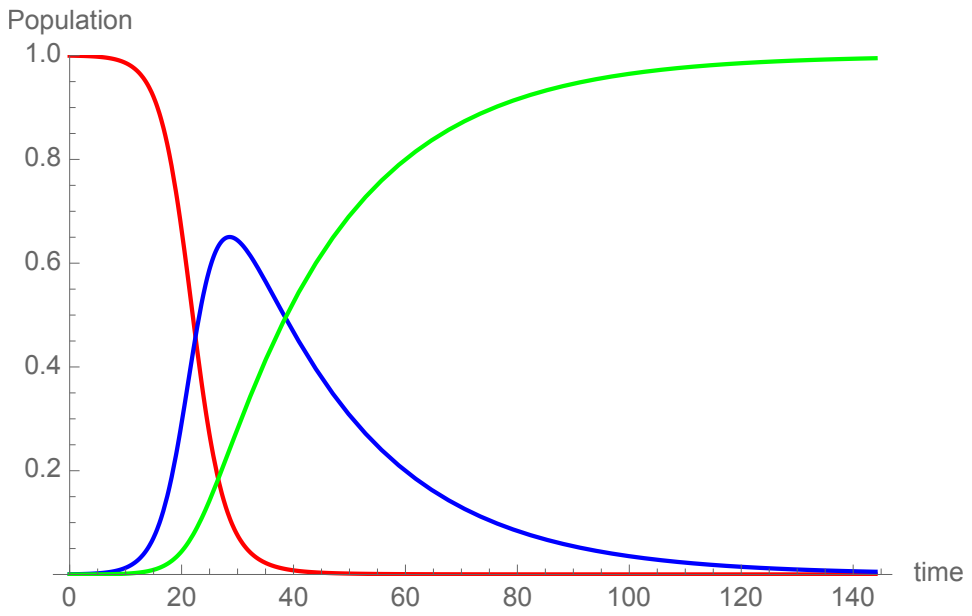

Figure 1: Simulating smallpox on Easter Island with the parameters $b = 0.4$, $k = 1/23$, and $I(0) = 1/2700$

simulations using parameters in these range, more than 90% of the islanders contracted smallpox. A simulation with $b = 0.4$, $k = 1/23$, and $I(0) = 1/2700$ is shown in Figure 1. In the figure, the red curve represents the susceptible population $S(t)$, the blue curve represents the infectious population $I(t)$, and the green curve represents the recovered population $R(t)$.

3 Intellectual ideas

The use of SIR models to understand the transmission of ideas dates back at least to 1964 when Goffman and Newill wrote a paper in Nature that explored modeling the development of ideas within a population [10]. In their model, Goffman and Newill described personal communication or publication as possible routes for the spread of infectious material. A more modern mechanism of transmission of ideas is through social media. In this section, we describe work by Woo, Son, and Chen [27] who used the SIR model to investigate the spread of violent topics on a major Jihadi web forum. Their model is very close to the system described in Section 2 except that they incorporated a variable population into their model.

In this model, $S(t)$ denotes the number of future authors, or the group of users who have interest in a topic and might read or comment on posts. The model assumes logistic population growth of the population with rate μ and carrying capacity K. Some susceptible users move in to the group of current authors $I(t)$ by writing or commenting on a thread after they have read previous posts about a topic. The constant b represents the degree of infectiousness of a topic. Authors move into the group of past authors $R(t)$ at a rate of k when their posts lose the power to infect others. The total number of authors at time t is

$N = S + I + R$. The model is given by

$$\begin{aligned} \frac{dS}{dt} &= \mu\left(1 - \frac{N}{K}\right)N - bSI, \\ \frac{dI}{dt} &= bSI - kI, \\ \frac{dR}{dt} &= kI. \end{aligned} \tag{3.1}$$

The authors collected data from a Dark Web Forum called Ummah from 2002–2010 which had over one million posts. They studied seven key topics discussed in the forum: *suicide bomb, anti-American, George Bush, wear hijab, honor killing, Bin Laden*, and *nuclear weapon*. They evaluated how well their model fit the data collected and found good results, with R-squared values ranging from 0.57 to 0.80. Of the seven topics, *anti-American* exhibited the highest infection rate, recovery rate, and growth rate, where *wear hijab* had the lowest infection rate. The authors concluded that the SIR model reasonably describes how violent topics spread on web forums.

Beyond the topics of violent ideologies, social media can also be used to spread information relevant to other social justice issues. For example, Woo, Ha, and Chen [28] modified the model given in Equation 3.1 to study the key topics of *healthcare and insurance, minimum wage and pay, stock price*, and *low price* on a Walmart discussion board. They adjusted the model by incorporating the possibility that an outside event, perhaps an important news story, could accelerate the infection rate. This paper also gave a helpful literature review of a number of studies of online information diffusion over discussion boards, computer viruses, blog connections, and other social media.

Beyond transmission by social media, Gurley and Johnson [12] used a more complicated model to investigate how sub-fields of economic theory grow or contract by exploring peer-reviewed journal publications. Similarly, Bettencourt et. al. [2] used a SIR model to investigate the spread of Feynman diagrams through the theoretical physics communities via personal communication in the period immediately after World War II.

4 Sustainable agriculture

Attention to sustainable agriculture is critical to humanity and the environment. There is a rich body of research using differential equations in this field; see [6, 8, 16, 20] as examples. Here, we describe work done by Gilligan, Gubbins, and Simons [9] in which they used a SIR system to model stem canker disease in tomatoes. This disease is caused by a fungus that attacks germinating tubers at the base of the stem, potentially destroying the stem.

Gilligan, Gubbins, and Simons set up their model by dividing the population into three categories. The population of susceptible stems is represented by $S(t)$ and the population of infected stems is $I(t)$. We use $R(t)$ to denote stems that are removed or dead. The study modeled how different levels of infection influenced the production of susceptible tissue,

called the *host response to infection load*. Their model was

$$\begin{aligned} \frac{dS}{dt} &= m(K-N) - b(t)S - f(I,S), \\ \frac{dI}{dt} &= b(t)S - kI, \\ \frac{dR}{dt} &= kI. \end{aligned} \tag{4.1}$$

Define $N = S + I + R$ to be the total number of stems. In this model, m represents the per capita rate of production of susceptible stems, k is the per capita death rate of infected stems, and K is the carrying capacity, or maximum number of stems per plant. The function $b(t)$ represents the force of infection, which involves the rate of infection of susceptible stems by the soil based fungus, the density of the fungus in the soil, and the level of host susceptibility. The function $b(t)$ can change with respect to time as the fungus becomes less infective and the host plant develops resistance.

The authors are primarily interested in determining an appropriate model for different host responses to the presence of infected stems. They consider seven different functions $f(I,S)$ that represent different responses. One of the possible functions is the null response, $f(I,S) = 0$. Another possible function is $f(I,S) = \alpha I$, where the reduction in the production of new stems changes in direct proportion to the amount of infected stems on the plant, where α is the rate of inhibition of production of susceptible stems due to infection. The other five functions $f(I,S)$ range from stronger reductions in the stem production with the presence of small amounts of fungus, to the possibility that the fungus significantly inhibits the production of more stems.

The authors fit their models to data collected Simons and Gilligan [21, 22] as well as Hide and Read [13] and Cother and Cullis [5]. They found that there is no evidence to reject the simplest SIR model where $f(I,S) = \alpha I$. In this case, disease can be controlled by reducing the initial inoculate density.

For an interesting discussion of a number of different models in sustainable agriculture, readers might look to Gilligan's paper *Sustainable agriculture and plant diseases: An epidemiological perspective* [8]. Gilligan describes a number of different SIR modifications that can be used to study agriculture on different scales, ranging from studies that focus on what happens within the plant to studies on national and continental scales.

5 Addiction

Alcoholism and drug use can be modeled as infectious diseases. In 2007, White and Comiskey [26] studied heroin use in Ireland in the hopes of gaining insight into how individuals progress through drug-using stages. White and Comiskey set up their model as follows.

The number of susceptible users, ranging from age 15–64, is given by $S(t)$. We use $I(t)$ to represent initial drug users or relapsed drug users who are not in treatment. The number of drug users in treatment is represented by $R(t)$. The number Λ represents a population growth rate resulting from individuals turning 15 during the study period and

thus entering the susceptible population, and N is the total size of the population under study. The constant b represents the probability of becoming a drug user, and k is the proportion of drug users who enter treatment. Finally, β is the probability that a drug user in treatment relapses to untreated drug usage.

In this model, individuals are removed from the population under study in a number of different ways. The natural (non-drug related) death rate is μ. The constant δ_1 represents a removal rate due to drug-related deaths of users not in treatment as well as a spontaneous recovery rate of individuals not in treatment who stop using drugs and are no longer susceptible. Similarly, the constant δ_2 represents a removal rate due to drug-related deaths of users in treatment as well as a spontaneous recovery rate of individuals in treatment who stop using drugs and are no longer susceptible.

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \frac{bSI}{N} - \mu S, \\
\frac{dI}{dt} &= \frac{bSI}{N} - kI + \frac{\beta IR}{N} - (\mu + \delta_1)I, \\
\frac{dR}{dt} &= kI - \frac{\beta IR}{N} - (\mu + \delta_2)R.
\end{aligned} \tag{5.1}$$

The authors use the model to investigate the *basic reproduction number* R_0, the threshold value representing how many secondary infections result from the introduction of one infected individual into a population. In their model, $R_0 = b/(k + \mu + \delta_1)$. They perform a sensitivity analysis and determine that, to control the spread of habitual drug use, preventing initial drug use is more effective than increasing the number of individuals in treatment programs. Results such as these offer suggestions to policy makers about where to focus limited resources.

Njagarah and Nyabadza performed similar work analyzing drug epidemics in which they also included functions representing the roles of drug lords in the process [18]. Benedict [1] and Walters, Straughan, and Kendal [25] use SIR models to perform similar analyses of the spread of alcoholism and binge drinking.

6 Crime

A person's decision to start criminal activity can be modeled as a socially contagious disease. Mushayabasa [17] used a four function model to investigate the effects of unemployment on property crime under the assumption that criminality is a socially contagious process.

In Mushayabasa's model, the population of 15–64 year olds is divided into four subgroups: $S(t)$ is the number of unemployed individuals at time t, $W(t)$ is the group of employed individuals, $I(t)$ is the group of unemployed and undetected criminals, and $R(t)$ is the group of detected and incarcerated criminals. Note that this model assumes that an individual or either employed or a criminal, but not both at the same time. The model is given by the following system of differential equations.

$$
\begin{aligned}
\frac{dS}{dt} &= \mu - bSI - (\mu + \alpha)S + p\upsilon R, \\
\frac{dW}{dt} &= \alpha S - (1-q)bIW - \mu W, \\
\frac{dI}{dt} &= bI(S + (1-q)W) - (\mu + g + d)I + (1-p)\upsilon R, \\
\frac{dR}{dt} &= kI - (\mu + \upsilon)R.
\end{aligned} \tag{6.1}
$$

The parameter μ denotes both a constant growth rate as people age into the population as well as a natural death rate which is consistent among the different subgroups. Criminals are assumed to have an additional death rate d due to their criminal activities. Again, b represents the infection rate when individuals in the two susceptible groups S and W interact with individuals in the group I. We use α to denote the difference between employment rate and retrenchment rate. The term $1-q$ represents the assumption that employed individuals are less likely to be drawn into criminal activity. Criminals are detected and incarcerated at a rate of g. Criminals are released from incarceration at a rate υ, and a proportion p of them enter the susceptible classes while the remainder $1-p$ become criminals again. Finally, the model allows for no movement of undetected criminals into the employment group by assuming that fear of detection would prevent this move.

The author investigates the basic reproductive number,

$$
R_0 = \frac{b(\mu + (1-q)\alpha)}{(\alpha + \mu)(\mu + g + d)} + \frac{(1-p)g\upsilon}{(\mu + \upsilon)((\mu + g + d)}
$$

The basic reproduction number accounts for how new criminals are generated. The first term of R_0 measures the effects of having susceptible members interact with criminals. The second term measures the effects of relapse. Mushabaya's analysis determines that if at most 20% of released criminals relapse into criminal behavior and at most 10% of employed individuals are influenced into property crime, then property crime will die out. The author continues his analysis by modifying the model in Equation 6.1 by adding functions representing government or police policies intended to detect or incarcerate criminals, improve employment rates, or improve rehabilitation systems and seeing how that affects the long term outcome.

Sooknana, Bhatt and Comissiong performed a similar study of the spread of gang membership in Trinidad and Tobago [4]. In addition, Ormerod, Mounfield, and Smith used an SIR model to investigate burglary and violent crime in the United Kingdom [19].

7 Eating Behaviors

The SIR model can be used to model the spread of eating behaviors leading to health issues by incorporating biological, behavioral, and social factors into the model. Obesity is not an illness in itself (it is possible to be both obese and healthy), but obesity is correlated with higher risks of cancer, heart disease, disability, and mortality [7].

In 2014, Thomas et. al. [23] constructed a model to investigate whether obesity rates in the United States will plateau or continue to rise. The authors were especially interested in observing the impact of the birth rate on obesity prevalence as well as the effects of a person being raised in an obesogenic environment. The model described in this section is the most complicated in the paper, as it divides the population into six different groups, which the authors immediately simplify into a system of five equations. The susceptible group $S(t)$ are individuals consistently classified with body mass index (BMI) less than 25 at year t. The exposed group $E(t)$ describes individuals consistently classified with BMI less than 25 that have been effectively exposed by year t but are not yet overweight. Individuals with higher BMIs were split into three groups: $I_1(t)$, individuals classified as overweight ($25 \leq BMI < 30$) at year t, $I_2(t)$, individuals classified as obese ($30 \leq BMI < 40$), and $I_3(t)$, individuals classified as extremely obese ($40 \leq BMI$). The function $R(t)$ represents individuals who have reduced back to normal weight at year t, so they are susceptible but predisposed to weight regain. Again, we use $N = R + S + E + I_1 + I_2 + I_3$ to denote the entire population. Solving for R, we obtain $R = N - S - E - I_1 - I_2 - I_3$, so the authors don't use a sixth equation to describe R in the model.

The model has many parameters necessary to describe the relationships between each of the populations. The constant p represents the probability of being born in an obesogenic environment, and the birthrate and uniform death rate is μ. So the first term $(1-p)\mu N$ in the first equation in Equation 7.1 represents the proportion of births to individuals who have BMI less than 25. People in the overweight and obese classes provide social influence, or infectiousness, to susceptible individuals represented by the constants k_1 and k_2. Individuals in each class can spontaneously gain weight at rates α for the susceptible class, a for the exposed class, a_1 for overweight class, and a_2 for the obese class. The rate of weight loss is represented by β_3 moving from the extremely obese group to the obese group and β_2 for moving from the obese group to the overweight group. Overweight individuals recover at a rate ρ_1. Finally, ρ_R represents the fraction of the recovered population that move to the exposed group.

$$\begin{aligned}
\frac{dS}{dt} &= (1-p)(\mu N) - \mu S - \frac{k_1 I_1 S}{N} - \frac{k_2 I_2 S}{N} - \alpha S, \\
\frac{dE}{dt} &= p(\mu N) - \mu E - aE + \frac{k_1 I_1 S}{N} + \frac{k_2 I_2 S}{N} + \rho_R(N - S - E - I_1 - I_2 - I_3) + \alpha S, \\
\frac{dI_1}{dt} &= -\mu I_1 + aE - a_1 I_1 - \rho_1 I_1 + \beta_2 I_2, \\
\frac{dI_2}{dt} &= -\mu I_2 + a_1 I_1 - a_2 I_2 - \beta_2 I_2 + \beta_3 I_3, \\
\frac{dI_3}{dt} &= -\mu I_3 + a_2 I_2 - \beta_3 I_3
\end{aligned} \tag{7.1}$$

Thomas et. al. use data from the US and the UK to develop values for the given parameters. For example, $p = .55$ in the US using data from 1988–1998, and $p = .3$ in the UK. For the US, if birth and death rates remain constant, the model predicts that obesity rates will plateau in 2030 at 28 % overweight, 32 % obese, and 9 % extremely obese.

Gonzales et. al. perform a similar study focusing on the role of college peer pressure on bulimic students who are not anorexic [11]. Ciarcià et. al. [3] study the effects of peers, media, and education on the dynamics of anorexic and bulimic populations.

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