Effect of Bad Actors in a Geographical Disease Model

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Abstract

Modeling and understanding the spread of disease has been a topic of much focus for epidemiological researchers in recent years, due to the effects of the COVID-19 pandemic. High levels of global attention and an abundance of recently collected data has created an environment for epidemiological models to be highly detailed and impactful. Determining parameter importance based on this available data is an ongoing analysis, which aims to help policymakers understand where to focus their attention. Here, NetLogo is leveraged to explore the spread of disease in Kingston, Ontario, with the significance of all major parameters, including various parameters relating to geographical and population data, being identified via modern sensitivity analysis techniques. Namely, Sobol sensitivity analysis is used, which explores the contribution of each parameter to simulation outcome variance. Bad actors are also introduced who further propagate the spread of disease as a way of mimicking individuals who do not adhere to prescribed policies, and the importance of the proportion of these bad actors in a population is measured alongside that of the other parameters. Masking and vaccination are core elements of this disease model, drawing from the available real-world statistics, and these bad actors intentionally avoid this behaviour. Sobol' analysis found that bad actors have a large impact on the outcome of disease spread relative to other parameters, and as bad actors partake in less and less preventative measures, their impact becomes higher while the impact of other parameters becomes lower.

1 Introduction

Disease modeling is a domain that has been the subject of extensive research over time, and has seen a significant uptake due to the emergence of COVID-19. These models are broken up into two man categories: mathematical models and agent-based models. Mathematical models aim to set up a system of differential equations that can be evaluated to simulate disease dynamics. While these models are relatively simple to interpret, they are generally less realistic than agent-based models due to the possible complexity associated with agent behaviour and associated stochasticity. This stochasticity can be leveraged to gain a deep understanding of possible simulation outcomes based on the values of underlying parameters. In turn, this concept can be used to study the relative importance of certain parameters, whose effects are relatively unknown, through a technique called sensitivity analysis.

Having a clear understanding of the factors fueling these parameters that contribute to the spread of disease is crucial for making decisions related to its mitigation. The majority of the factors that are studied relate to fundamental behaviours associated with disease dynamics, or population statistics. For most purposes, these types of factors alone are sufficient - historically, we have been able to use them to predict disease spread with a high degree of accuracy. However, only using factors of this nature implies the assumption that everything else in the problem space is behaving as expected. In reality, this is almost never the case. There are always differences between generated models and the real world, though these differences often impact disease spread in minor ways compared to fundamental disease dynamics and population statistics and are thus, commonly ignored. While this oftentimes does not have large consequences, special exceptions can occasionally have significant impact.

When examining how disease spreads within a human population, one obvious factor that can determine spread patterns is the behaviour of people in the population. In most cases, modelers assume people to behave quite similarly, usually having their moment-to-moment decisions being broad-scale, and based off of known or estimated distributions. Modelers also assume the behaviour of human agents to be relatively uniform. While models making these assumptions have proven to be quite reliable, possible scenarios exist where large behavioural differences have the potential to change the course of a disease's life cycle

in a population. As an example, let us examine how COVID-19 affected people's behaviour. COVID-19 saw immense amounts of information being delivered to people worldwide, but along with that came large amounts of misinformation and skepticism. This led sizable portions of populations to have beliefs that were not in line with the general public or health officials aiming to reduce the disease's spread. Common behaviours associated with this group were to avoid masking and vaccinating. This behaviour undoubtedly enhances disease spread, but it is important to understand exactly how impactful this behaviour is in order for health officials (and the broader public) to determine how to distribute their attention.

This paper presents an agent-based model created in NetLogo that aims to leverage sensitivity analysis to understand the potential impact of these sorts of agents, called "bad actors" henceforth, in a geographical setting based on the city of Kingston, Ontario.

2 Background

2.1 Disease Modeling

Understanding SEIR modeling is essential in the field of epidemiology. The most basic form of the SEIR model is actually called the SIR model, where "S stands for "Susceptible", "I stands for" "Infectious", and "R stands" for "Recovered". Each of these represent a compartment (or class) where agents can occupy. Importantly, all agents must be in one of these compartments. In my model, I include the "Exposed" class, represented by "E" to create a model that is more representative of most diseases. Agents in the formulation of my model are also able to move from the Recovered class back into the Susceptible class, technically making this an SEIRS model. The way an agent may move between these four classes can be seen in 1, and the four classes can be thought of as the following:

- 1. Susceptible: Individuals who may contract the disease at any given point
- 2. Exposed: Individuals who have contracted the disease but are not symptomatic and are not able to spread the disease
- 3. Infectious: The individuals in this class may or may not be showing symptoms but are now able to spread the disease
- 4. Recovered: Here, individuals are fully recovered from the disease, meaning that they cannot spread it *and* they cannot contract it

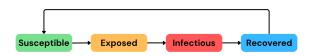


Figure 1: The classes that an individual moves through in an SEIRS model

Agents may move between these classes depending on whether or not they have contracted the disease. Length of time in each class can vary, and is usually dependent on severity of infection, genetics, and treatment status.

We must also define the R_0 value here, which represents the average number of susceptible individuals that an infectious individual will spread disease to. One formulation is to have the R_0 value be calculated as $R_0 = \beta/\gamma$, where β represents the number of individuals the infection is spread to per time step, and γ represents the rate of recovery. This equation can be used to elicit realistic disease mechanics, as will be shown in the Methodology section.

2.2 Sensitivity Analysis

Sensitivity analysis, in this context, is the process of determining the degree to which certain variables/parameters comprising a model affect the outcome of simulations. There are a variety of methods for completing this task, but for this paper, the Sobol' method is used. All sensitivity analysis methods involve identifying parameters of interest, changing them slightly, and observing the simulation outcome as a result. In contrast to local sensitivity analysis, the Sobol' method is a type of global sensitivity analysis, meaning that all parameters of interest are changed simultaneously from run to run. Through this, the total-order, first-order, and higher-order sensitivity indices may be retrieved. The first-order indices represent the fractional input of one parameter to model output - that is to say how much one parameter contributes to outcome variation independent of the other parameters. The higher-order indices (ranging from two to the number of parameters in the model) represent the interaction importance between two or more parameters. Finally, the totalorder indices represent the overall effects of one parameter on model output including interaction with all other parameters. The derivation of these values is drawn from a series of statistical calculations, and is out of scope for this paper. However, the process is outlined in more detail in a paper by Zhang et. al.[1].

3 Related Work

There exist a plethora of agent-based models relating to disease spread, most of which are open source and regularly updated [2, 3, 4, 5]. Many of these were created or adapted upon the emergence of COVID-19 [6]. Some of the more popular of these models, like OpenABM-Covid19 and Covasim [7, 8] have been used my public health officials worldwide, solidifying the idea that agent-based models can be used in very important ways. One major drawback of these models is that they are quite general, and do not incorporate region-specific geography and population statistics. One model, GSAM [9], takes this idea further and allows for billions of agents to act distinctly at the cost of specificity. These kinds of general models, while interesting and necessary for certain kinds of research, are not what is covered in this paper. It is also a fact that most influential models in recent years have not been created in NetLogo. While these models are successful, it is important to explore different software in order to reach wide audiences and keep software that specialize in different domains relevant.

However, there are some detailed models that have been created in NetLogo recently revolving around the topic of disease spread. One model leverages NetLogo 3D to model how disease spreads in a fish population [10]. This model describes the pathogen itself as an agent, as it has the ability to move in the water. While this sort of thing is not applicable to the model presented in this paper, it is a fascinating application that could readily be applied to traditional NetLogo in 2D. Another NetLogo model aims to explore disease spread in the Irish town of Schull [11]. This model sees agents adopt daily routines that are quite detailed, and explores regional statistics and geography unique to Schull. This is guite similar to the model presented in this paper, though agent movement and disease spread is captured when agents "bump into each other" on their predetermined paths in the Schull model.

4 Methodology

4.1 Modeling the Environment

Space Initialization Given that every disease model is constructed a little bit differently per the intention of the designer, I will go over how the model is created. This model exists in a 2-dimensional space, and the initialization of this geographical space (mimicking Kingston, Ontario) will be described in detail.

Each agent is assigned a home, job, and two stores. Depending on the age bracket of the agent,

their "job" is either a school, a traditional job, or a store. Agents move between their homes, jobs, and stores depending on the time of day and day of the week, and this whole process will be outlined.

Finally, I will discuss how the disease actually spreads in this environment. I will go into detail describing the calculations on each time step that govern this disease spread. I will also show some graphs illustrating how the SEIR classes behave over time on simulations.

Using the OSMnx package in Python [12], I was able to retrieve geographical information from Kingston, Ontario, in the following four exclusive categories:

- Residential: "House", "Apartment", "Dormitory", or "Residential", encapsulating the left-over residential buildings
- Work: "Office", "Commercial", "Industrial", "Retail", or "Warehouse"
- Commercial "Commercial" or "Retail"
- Education: "School", "College", or "University"

Ensuring that there were no overlapping coordinates (as overlapping coordinates cannot be easily represented in NetLogo), the buildings associated with each of the four categories were saved to shapefiles. From here, the shapefiles were read into NetLogo and the buildings appeared as colour-coded points (red for residential buildings, blue for commercial buildings, yellow for educational buildings, and green for workplaces).

Agent Initialization Agents are created with a set of attributes, and they have the ability to move between the defined locations. Note that they do not "walk" from location to location, but "teleport" instead, as disease transmission is rarely due to passing an infectious person on the street. Each agent is assigned an age bracket based on the age distribution of Kingston, Ontario [13]. The age brackets range from 0 to 8, where 0 represents 0-9, 1 represents 1-19, and so on until 8, which represents 80+. These age brackets determine their daily behaviours, and are used in the calculation of vaccination rates.

Agents are assigned homes, jobs, and stores that they move between on time steps. Each home contains 1-5 agents (and all agents have a home) per Kingston statistics [13], and they will always move to or from their home (i.e. they will never go from their job to a store).

For job assignment, agents that are in the "0" or "1" age brackets are made to be attending schools. The school that each agent in these age brackets attends is chosen to be the closest school to their home by Euclidian distance. Some agents in the "2" age bracket will attend post secondary school. These agents are placed in a reverse manner when compareed to the students in the "0" and "1" age brackets - they are assigned a post-secondary school, and then placed in a home that is the shortest distance from the school such that the home is not full already (each home has a pre-determined capacity). This will result in the majority of students being in dormitories, with some being placed in houses or apartments close to campuses. Some other agents in the "2" age bracket and all agents in the age brackets ranging from 3 to 6 are said to work during the weekday. Their work location is chosen randomly from all of the available options. Finally, the agents that are in the "7" or "8" age brackets go to a store during the weekday. The store they go to is chosen to be the closest store to their home by Euclidian distance.

Agents also all have stores associated with themselves. On *weekends*, agents go to two stores (for what can be thought of as Saturday and Sunday, though it is not represented as such) that are chosen to be the closest to agent homes.

Disease Dynamics Agents exist in on of the SEIR states, depending on their exposure to disease. At each location, agents have a probabilistic chance of contracting disease from other agents. The chance of *one* infectious agent passing disease to *one* susceptible agent is defined in this model to be equal to:

 $\frac{(R_0 / \text{(total time in infectious class)})}{\text{(number of susceptible agents at the location)}}$

Here, R_0 , referring to the basic reproductive ratio, is user-defined in NetLogo. This calculation "forces" each agent to spread disease to a number of agents approximately equal to R_0 , matching its definition. If this chance of infection is successful, the susceptible agent moves into the exposed class, where they are said to have the disease, but be incapable of spreading it. When they move into the infectious class, they are able to spread disease, and have a chance of isolating (where they cannot spread disease to anyone) per user specification. They then move into the recovered class, where the agent no longer carries the disease, and cannot contract it. Agents remain in the exposed, infectious, and recovered classes per the user's specification (time in classes is represented by a normal distribution, and users can edit the mean and standard deviation in each class).

Agents can also be masked and/or vaccinated, which is determined at the start of the simulation. Agents have a 70% chance of masking [14] and a chance of vaccination that is age-dependent [15]. This is determined on simulation setup, and the agents remain in these states for the whole simulation. Masking and vaccination works by multiplying the chance of infection defined earlier by a fixed value between 0 and 1. This fixed value can be changed by the user to simulate situations where the efficacy of masking and vaccinating varies.

Agents were also created to have a chance of being "bad actors", which is the main subject of study for this paper. If an agent is a bad actor, they will behave identically to other agents, but they have the capabilities of not masking, not vaccinating, and not isolating. How the bad actors behave is determined by the user, where their masking/vaccinating/isolating functionalities can be enabled or disabled.

Figure 2 is an example of how disease may spread under certain conditions. Here, the basic reproductive ratio is 3, the exposed time is Normal(9,2), the infectious time is Normal(16.4), the recovered time is Normal(14,2) (values similar to those of COVID-19 [16, 17, 18]), masking and vaccination strengths are 0.9 and 0.5 respectively, agents isolate at a rate of 0.3. These dynamics are in line with what is expected, and an equilibrium is reached, showing the validity of this model. We can introduce bad actors who do not mask, vaccinate, or isolate at a rate of 0.2, which alters the dynamics as shown in figure 3. The introduction of bad actors consistently pushes the equilibrium value of susceptible agents down (and other classes up), and the magnitude of this effect will be studied via sensitivity analysis.

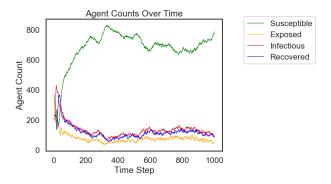


Figure 2: Sample run of a simulation with no bad actors

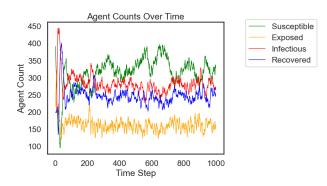


Figure 3: Sample run of a simulation with bad actors included, who do not mask, vaccinate, or isolate

4.2 Sensitivity Analysis

In order to perform sensitivity analysis on the model, I needed to extract information from NetLogo into Python, where the bulk of analysis would be performed. This process was done by using the Pynetlogo package [19], which enables the execution of NetLogo models directly from Python via a link (called a NetLogo Link in the Pynetlogo Documentation). Simulations can be run in bulk through this, and the desired results can be retrieved and recorded for each run.

Using the SALib Python package [20], a number of parameter configurations (768 here, chosen such that analysis could be performed in a timely, yet effective, manner. The options above and below this are 1536 and 384 respectively), tailored for Sobol' analysis, were generated via the "sobolsample" function using parameter names and bounds as inputs. The parameters examined here were isolation rate, bad actor proportion, basic reproductive ratio, vaccine infection factor, and mask infection factor. These are variable parameters in the model that can highly impact the outcome of a simulation, and so the intensity at which they contributed compared to each other seemed to be an interesting exploration. The parameter bounds were 0 to 0.8, 0 to 1, 3 to 7, 0.3 to 0.5, and 0.7 to 0.95 respectively, which are ranges that are both plausible and elicited simulation outcomes that had a large variation (i.e. ranges in which the disease was not simply eradicated or entirely saturated within the population). Sensitivity analysis can be performed in a variety of ways, with some focusing on epidemic peaks, and others focusing on broader trends. For this work, I elected to examine approximate equilibrium values for the number of infectious agents. So, for each simulation I retrieved the average of the values from time step 250 to 500, which is where simulations tended to always hover around

equilibrium.

This sample/simulation process was performed five times for configurations where bad actors acted the same as regular agents (i.e. where there are no bad actors), where they *only* mask, where they *only* vaccinate, where they *only* isolate, and where they do none of the three. In each case, the impact of all tested parameters was retrieved via Sobol' analysis, and the relationships between the number of infectious agents and each parameter could be loosely inferred.

5 Evaluation

When running all 768 simulations, examining the count of agents in each class can be quite informative, especially when varying single settings at a time. Figure 4 shows how the distribution of infectious agents (of all 768 simulations) varied when there were no bad actors, when bad actors only vaccinated, and when bad actors did not vaccinate, mask, or isolate. We can see a clear shift towards there being more infectious agents at equilibrium when bad actors engage in less and less preventative measures. This alone is indication that the prevalence of bad actors has a large impact on the behaviour of this model, but it is not enough to determine how the model actually behaves as the proportion of bad actors increases relative to other parameters.

Figure 5 shows how the number of infectious agents changes across each parameter as their values change. The top left cluster of four shows the configuration where bad actors are indistinguishable from regular agents, the top right cluster shows the configuration where bad actors only vaccinate, and the bottom cluster shows the configuration where bad actors do not mask, vaccinate, or isolate. Looking at the "bad-actor-proportion" graphs specifically for the top right and bottom clusters (as bad actors have no effect in the top left cluster), we can see a clear trend, where as the proportion of bad actors increase, so do the number of infectious agents at equilibrium. This relationship is stronger when bad actors take no preventative measures, and appears to be somewhat logarithmic in nature. We also see that the other parameters, while still maintaining the fact that they affect the number of infectious agents at equilibrium, appear to have the intensity of their effects dampened somewhat as bad actors engage in less and less preventative measures. A plausible reason for this would be that as more agents engage in reckless behaviour, the disease reaches saturation (i.e. a point where about as many agents are infectious as possible given

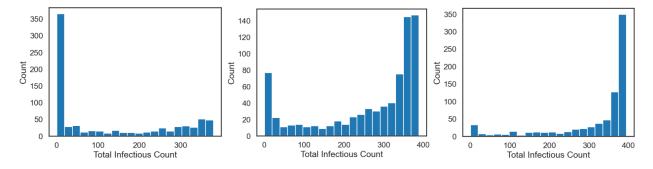


Figure 4: Histograms showing the distribution of infectious agents at equilibrium. The left graph shows the configuration where bad actors are indistinguishable from normal agents, the middle graph shows the configuration where bad actors only vaccinate, and the right graph shows the configuration where bad actors do not mask, vaccinate, or isolate.

the population configuration, and any only marginal increases are possible with parameter value modifications) faster and faster, and so the upper bounds of the parameter ranges matter less and less.

For Sobol' analysis, a series of graphs were generated showing the first-order and total-order sensitivity indices. Figure 6 shows the sensitivity indices for all five parameters that were varied, across configurations where bad actors are indistinguishable from regular agents, where bad actors only vaccinate, and where bad actors do not mask, vaccinate, or isolate, from left to right. Note that the y-axis values are dimensionless and without units, only existing to compare parameter significance.

When bad actors take no actions, their significance drops to essentially 0 - this is what was expected. In the same graph, we can also see that the rate of isolation and the value of the basic reproductive ratio appear to have the most significant impact on infectious agent count at equilibrium when varied. This means that small perturbations of the two will result in large changes relative to changes of the vaccine and mask strength across their ranges. The isolation rate having such an impact does make sense - an agent isolating essentially means that they are removed from the model and cannot spread to other agents. In this way, the value of the basic reproductive ratio has no bearing on them. The basic reproductive ratio being important also makes sense, since it is the driving force for infection survival in a population. However, the vaccine and and mask strength being so insignificant across their ranges is surprising - this behaviour could be due to the fact that the majority of simulations with this configuration resulted in susceptible agents being very high (close to or equal to 1000), and thus, increasing the effect of minor preventative measures when compared to the isolation rate would not have much of an impact.

In the middle graph, where bad actors only vaccinate, we can see that the significance of their proportion is very important. This graph also sees the effect of the isolation rate being less significant, while the vaccine and mask strength actually appear to become slightly more significant. This could be due to how the bad actors interact with masks and vaccines. Since their behaviours are quite linked, having the mask and vaccine values be different would have a large impact on how the simulation plays out.

In the rightmost graph, where bad actors do not mask, vaccinate, or isolate, we see that their importance becomes even more pronounced when compared to the other parameters, following with what was expected. The proportion of bad actors becomes highly important as they elect to engage in no preventative measures, essentially making the preventative measures non-existent as the proportion of bad actors tends to 1. We also see in this graph that the firstorder and total-order sensitivity indices appear to become more similar. This result is somewhat unexpected, since as the proportion of bad actors (whose existence is heavily related to masking, vaccinating, and isolating) increases, one would expect that the interaction effect shown by the total-order indices would be more exaggerated.

6 Discussion

Limitations One major limitation is the lack of data available on people who refuse to mask, vaccinate, or isolate. Ideally, one would determine the parameter ranges for a specific disease, and perform sensitivity analysis on them for their respective ranges. This would include bad actor proportion as a parameter, but the data on that would be essentially non-existent, making the process difficult to approach.

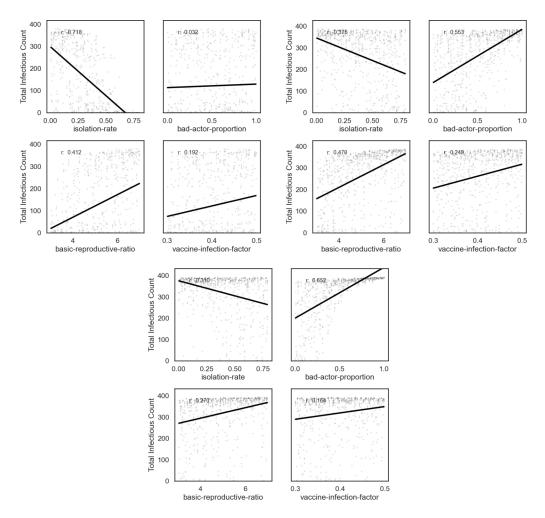


Figure 5: Graphs indicating infectious agent count as parameter values are varied. The top left cluster of four shows the configuration where bad actors are indistinguishable from regular agents, the top right cluster shows the configuration where bad actors only refuse to vaccinate, and the bottom cluster shows the configuration where bad actors do not mask, vaccinate, or isolate.

Another limitation here was computational power. The error bars on the sensitivity indices were quite large, and this is likely due to the limited number of simulation runs that could be performed (though they would not entirely disappear due to the stochastic nature of the model). The sensitivity analysis becomes increasingly accurate as more simulations are run, and so the data may have presented somewhat differently in this case. Computational power also limited the size of the worlds being used in each simulation. For all simulations run, only 1000 agents were used - this is far less than the population of Kingston, and would affect the disease dynamics and the outcome of the sensitivity analysis.

Another limitation is in the capabilities of geographical representation in NetLogo. In a setup where agents are moving around some 2D space,

there cannot be two locations on the same patch. This becomes problematic when attempting to map a city into 2D space, because there are many instances where either buildings are very close together, and would get lumped into the same NetLogo patch, or when a residential space is directly on top of a commercial space. This happens regularly with apartment buildings being located on top of stores. In this way, it was impossible to directly capture the geography of Kingston, and some buildings needed to be removed.

Future Work This work can be progressed in several directions. One possible improvement would be to have the representation of Kingston be more accurate and create more complex agent behaviour as a result. For example, Kingston has many recreational

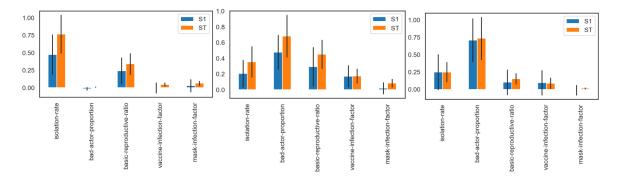


Figure 6: Sensitivity indices for different configurations. The left graph shows the configuration where bad actors are indistinguishable from normal agents, the middle graph shows the configuration where bad actors only vaccinate, and the right graph shows the configuration where bad actors do not mask, vaccinate, or isolate.

areas that were not incorporated in this model, and so future work could see the inclusion of these recreational spaces and have agents hold events at them. This would potentially change the prevalence of disease in a community, and would make the geography of Kingston be more important relative to disease spread.

Another line of work could see sensitivity analysis techniques used in different ways. There are a variety of methods that see use, and they all perform slightly different functions. Some analyse simulation behaviour over time, while others only look at individual points (like Sobol' analysis). Examining the peak number of infectious agents could prove insightful, and looking at how student populations are affected by parameter configurations could also provide interesting results. It is often the case that we aim to prioritize vulnerable groups, and so performing sensitivity analysis with these groups in mind could be important - if it turns out that bad actors disproportionately affect vulnerable groups, then that would increase the importance of the matter even further.

This process could also be generalized to other agent-based modeling platforms. While NetLogo is arguably the most accessible tool, it lacks in customizability, and as such, certain functions are dif-

ficult or impossible to implement in NetLogo. Many agent-based modeling tools are now available in languages like Python, where the developer has much more control of exactly what is going on "behind the scenes".

7 Conclusion

This paper analyses the effects of "bad actors" on the spread of disease via a geographical agent-based disease model of Kingston, Ontario implemented in NetLogo. By performing Sobol' sensitivity analysis after variation of several parameters under different bad actor functionalities, the relative importance of each parameter to the number of infectious agents at equilibrium could be inferred. The results in this paper show that these bad actors who refuse to engage in preventative measures have a very high impact on how the disease spread within a community, increasing the number of infectious agents at equilibrium significantly even at small proportions. As they increasingly partake in less and less preventative measures, their effect becomes more pronounced, and the significance of the other parameters relating to disease spread shrink as a result.

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