

## Interaction Based Model for Identifying Asymptomatic Carriers of an Infectious Disease

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### Abstract

*Epidemiological models are commonly used to predict and describe the spread of a viral outbreak among a population. Many such models use differential equations and transition rates to predict the growth dynamics of the infectious and exposed groups with a greater population. These methods do not distinguish between infectious individuals. In this paper, we propose a new model that includes asymptomatic carriers while holding constant many of the transition rates and assumptions of the classical models. Seeking to replicate realistic outbreak scenarios, we introduce a way to estimate the reproduction number  $R_0$  of epidemics and apply these estimations to our model. Our results replicate those described in similar research papers, showing that a small proportion of asymptomatic carriers can be responsible for a majority of the transmissions. We propose possible extensions to our model, underlining the impactful applications it may have on healthcare management and public safety policymaking.*

### 1. Introduction

Many epidemiological tools have been developed to help predict the spread of viral outbreaks. The dynamics of infection speed and its effect on populations can be described by many mathematical and quantitative tools. A popular application of such models is to predict the future number of cases, which can be used to plan for utilization and upcoming workload that healthcare providers, clinics and hospitals will be presented with. However, while they offer good policy guidelines for healthcare professionals and epidemiologists, these methods do not actively identify growing areas of risk and members of the population that pose the greatest danger to others. It is of grave importance to know how fast a virus will spread, but it is of even greater importance to be able to identify individual spreaders and isolate them immediately.

Classical models such as the SIR model compartmentalise the entire population into a few well-defined groups. A system of differential equations

and transition rates between these groups predicts the evolution in size of each group. These univariate temporal evolutions are the basis for prediction and policy making regarding the best case load that regional healthcare systems should prepare for.

The purpose of this paper is to develop a methodology that maintains many of these caveats, and can describe the evolution of sub-populations using similar differential equations and transition rates. However, the methodology would also allow for these groups to be more granular, and describe each of their respective members individually, presenting a finer description than a singular size measurement that evolves over time. Additionally, the methodology would account for the fact that the infectious group is not homogeneous in its nature, and that it is rather comprised of two potential spread vectors: one symptomatic and short-lived, while the other is undetected asymptomatic which is never isolated and can therefore expose others over a more prolonged period. By treating everyone in the population as a granular entity with its own unique evolution path and outcomes, the proposed model would give public healthcare professionals better quantitative forecasts of the population sub-groups which are likely to pose high risk for susceptible individuals.

A model with these characteristics could be used not only as a predictive tool, but also as a decision-making method that allows for more active management of an epidemic. By allowing each individual member of the population to follow a unique sequence of states, from exposure to recovery, the new methodology would have strong impact and application not only in prediction and forecasting, but as a contact-tracing tool on its own merit. This gives decision and policy makers the ability to not only predict future trends in outbreak numbers, but also to identify areas within the population that are at greater risk, and address them directly to reduce the load on the local healthcare systems along with the breadth of the overall viral spread.

## 2. Background

In epidemiology, the SEIR model has been used to predict the flow of population between four groups:

1. Susceptible ( $S$ )
2. Exposed ( $E$ )
3. Infectious ( $I$ )
4. Recovered/Removed ( $R$ )

The proportional sizes of these four states evolves in accordance with these four differential equations:

$$\begin{aligned}\frac{dS}{dt} &= \mu - \beta SI - \mu S \\ \frac{dE}{dt} &= \beta SI - (\mu + \alpha)E \\ \frac{dI}{dt} &= \alpha E - (\mu + \gamma)I \\ S + E + I + R &= 1\end{aligned}$$

where  $\mu$  is the equal rate of deaths and births in the population,  $\beta$  is the rate at which susceptible-infected contact results in new exposure,  $\alpha$  is the rate at which exposed individuals become infected, and  $\gamma$  is the rate at which infected individuals recover or are removed from the population. After recovery, individuals are immune. The path of an individual through these four states looks like this:

$$S \rightarrow E \rightarrow I \rightarrow R$$

Extensions to this formulation include specifications for isolating, quarantining, and even asymptomatic carriers [1,2]. Some work has been done on practical ways to mitigate asymptomatic carriers' infectiousness [4,5], but these methods utilize methodologies such as social distancing, and nasal swabbing, that are not tailored to the problem of asymptomatic carriers.

Some contact-tracing methods do account for the possibility of non-symptomatic individuals [3]. These models provide mathematical alternatives to describing spread dynamics of outbreaks and treat individual members of the population as unique. However, these contact-tracing models only produce insightful answers regarding the growth rate and criticality of the outbreak, only modelling the population size and not treating each individual member of the population as a unique potential spreader.

## 3. Problem Statement

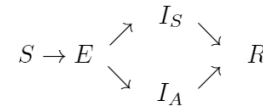
An extension to the SEIR model can account for the phenomenon of asymptomatic carriers. After a

latent period of  $1/\alpha$  (henceforth referred to as the incubation period), an exposed individual can exhibit symptoms that make them easy to identify and isolate, thus stopping the spread of the disease. Alternatively, the exposed individual can show no symptoms after incubation, and do not self-isolate or become hospitalized as they continue to infect others.

Since asymptomatic and susceptible individuals display the same set of characteristics, there is no deterministic way of telling them apart. Yet models similar to SEIR fail to account for this discrepancy by not distinguishing between the symptomatic and asymptomatic. Since only the former is detectable and presents the greatest load onto the healthcare system, it may be most useful to forecast and predict the amount of symptomatic individuals in a population. However, as asymptomatic individuals may be similarly infectious, it could be the case that a hefty portion of transmissions and symptomatic infections is a result of asymptomatic exposure, as shown in [8]. For this reason, a tool which accounts for the asymptomatic may be more useful to accurately predict and forecast the resources required to manage an outbreak. And in contrast to previously implemented methods, the tool must be centered around the individual, rather than the population.

## 4. Proposed Solution

We first propose a model that allows for the degeneracy in the infectious population. We assume that asymptomatic and symptomatic individuals are characterized by different lengths of infectiousness and different probabilities of exposing others, and other dissimilarities. Therefore, our model paints the spread as follows:



where  $I_A$  and  $I_S$  represent the undetected asymptomatic and symptomatic members of the infected population, respectively. Exposure remains identical for all individuals, while infectiousness can be symptomatic or asymptomatic. Recovery or removal is also identical for all individuals.

The new model can be described by the following set of differential equations:

$$\begin{aligned}\frac{dS}{dt} &= \mu - \beta S(I_A + I_S) - \mu S \\ \frac{dE}{dt} &= \beta S(I_A + I_S) - (\mu + \alpha)E \\ \frac{dI_S}{dt} &= \alpha p E - (\mu + \gamma_S)I_S\end{aligned}$$

$$\frac{d}{dt}I_A = \alpha(1 - p)E - (\mu + \gamma_A)I_A$$

$$S + E + I_A + I_S + R = 1$$

Here, the infectious population  $I$  is split into the two degenerate infectious groups, one representing the symptomatic cases  $I_S$  and one representing the asymptomatic cases  $I_A$ . The rate at which exposed individuals become infectious is also split in this matter, it is represented by  $p$ , a probability that the exposed individual will be symptomatic and infectious. With probability  $1-p$ , the exposed will become asymptomatic and infectious. The rates at which the symptomatic and asymptomatic infectious groups recover,  $\gamma_S$  and  $\gamma_A$  respectively, are also modified to characterize the asymptomatic and symptomatic cases individually. Realistically,  $\gamma_A$  would be slower than  $\gamma_S$  as asymptomatic cases remain within the population for a longer time without isolating, as symptomatic cases are easily identified and removed from the population.

As noted before, solutions to these equations do not help to isolate the asymptomatic carriers among the population and do not treat each individual as a unique member of the population. Therefore, we impose additional assumptions and pursue a simulation-based approach to finding non-symptomatic infectious individuals within this augmented system.

#### 4.1. Formal Modification to SEIR

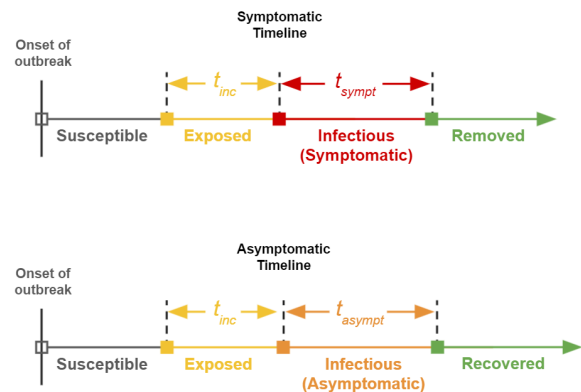
We assume that the total size of the population, comprising the five subgroups proposed above, is equal to  $N$ . Additionally, the following assumptions about the system are made:

1. Births and deaths are negligible. This would be equivalent to setting  $\mu = 0$  in the original SEIR model.
2. Contact occurs in discrete times, not continuously.
3. At every time interval  $t$ , the susceptible ( $S$ ), exposed ( $E$ ), symptomatic ( $I_S$ ) and asymptomatic ( $I_A$ ) populations are spread among  $n_{cells}(t)$  infection cells. Individuals can only come into contact with others who are in the same infection cells as them. Additionally, all contact between individuals is known and stored as historical data.
4. When a susceptible individual interacts with an infected individual, the susceptible person becomes exposed with probability  $p_{expose}$  or remains susceptible with probability  $1 - p_{expose}$ . This exposure parameter is similar in nature to in the SEIR model.

5. When a susceptible individual is exposed, an incubation period of  $t_{inc}$  discrete time steps begins. During this period, an exposed individual cannot transmit the disease to others. After this incubation period, two possible outcomes can occur:
  - a. With probability  $p_{sympts}$ , the exposed individual begins to show symptoms and is isolated after  $t_{sympt}$  periods of infectiousness. Isolation comprises complete removal from the system and no further interactions with others. However, during the  $t_{sympt}$  infectiousness period, the individual can expose others to the virus. We note that all isolated symptomatic individuals are known, identifiable and countable.
  - b. Alternatively, with probability  $p_{asympt} = 1 - p_{sympts}$ , once incubation is complete, the individual might not show symptoms. During  $t_{sympt}$  periods after incubation, the individual can expose other entities it interacts with to the virus. After this period, the asymptomatic carrier recovers and cannot infect others or get re-infected. We reiterate that this assumption prohibits asymptomatic carriers from becoming symptomatic. This makes it impossible to tell a susceptible individual from an active asymptomatic or a previously asymptomatic individual.

Figure 1 shows these two possible timelines of an infected case.

6. In the initial state of the system there is only one infectious asymptomatic carrier, with all other individuals being susceptible.

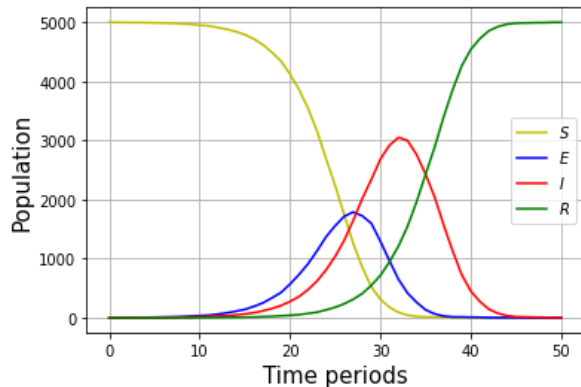


**Figure 1.** Symptomatic vs. asymptomatic timelines. Both progress to be exposed after

being susceptible, with incubation lasting  $t_{inc}$  time periods. Symptomatic cases are then infectious for  $t_{sympt}$  time periods, while asymptomatic cases are infectious for  $t_{asympt}$  time periods. Here,  $t_{asympt} > t_{sympt}$ . Both cases are removed from the system when no longer infectious.

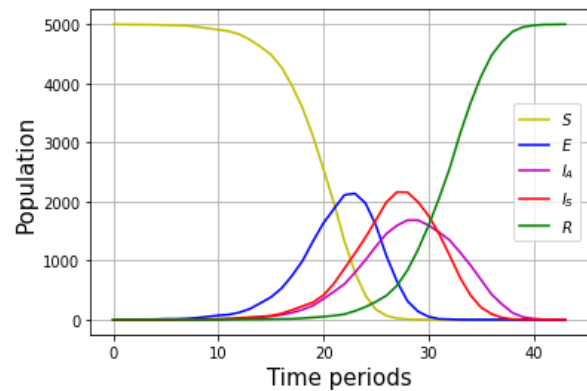
#### 4.2. Validation of Correctness

If  $p_{asympt} = 0$ , we expect the system to resemble a basic SEIR model with no distinction between asymptomatic and symptomatic infected. Using a simulation of  $N = 5,000$ ,  $n_{cells} = 2,500$  and  $p_{expose} = 0.33$ , a simulation of the proposed system was run with  $p_{asympt} = 0$ . We set  $t_{inc} = t_{sympt} = 4$  as the incubation and infectiousness periods. We plot the population sizes after 50 time periods in figure 2. The new proposed system shows similar behavior to a solution of the SEIR model [7] even though the dynamics of exposure are completely different. The gradual decrease in the yellow curve representing the susceptible  $S$  population is identical to the shrinkage of  $S$  in the SEIR model. The gradual increase in the green curve representing the recovered or removed  $R$  population is also identical to the growth of the  $R$  population in the classical epidemiological models. Both these populations reach a stable equilibrium level by the end of the outbreak, which occurs when no members of the population are left to be exposed or no new infections have occurred. The blue curve, representing the exposed  $E$  population, grows and shrinks as seen in classic SEIR model predictions, and a similar observation can be made for the red curve representing the infectious population  $I$ .



**Figure 2.** Validation of our proposed model. The population sizes after 50 time periods resemble analytical results to the SEIR model seen in earlier research [7]. ( $N = 5,000$ ,  $n_{cells} = 2,500$ ,  $p_{expose} = 0.33$  and  $t_{inc} = t_{sympt} = 4$ ,  $p_{asympt} = 0$ ).

With our model validated, we now pivot to the case where  $p_{asympt} > 0$ , and where asymptomatic carriers can exist in the system. As an example, we repeat the same simulation for  $N = 5,000$ ,  $n_{cells} = 2,500$  and  $p_{expose} = 0.55$ ,  $p_{sympt} = 0.6$ ,  $t_{inc} = t_{sympt} = 4$  and  $t_{asympt} = 10$ . The results can be seen in figure 3. A similar dynamic between the population subgroups can be observed. The group of asymptomatic individuals, represented by the purple line, grows and shrinks in near resonance with the symptomatic infectious group. However, since asymptomatic carriers remain in the system longer, the peak size and equilibrium state for  $I_A$  occur after those of  $I_S$ . The results of our validation show that even though our proposed model treats each individual as an independent and identifiable entity, the infection dynamic of the entire population can still be measured, tracked and analyzed in an epidemiological manner.



**Figure 3.** An example of the system's evolution when including asymptomatic carriers. The purple plot-line shows how the asymptomatic group grows in a similar pattern to the symptomatic one. However, the asymptomatic subgroup lingers in the system and expedites the rate at which the general population is exposed. ( $N = 5,000$ ,  $n_{cells} = 2,500$  and  $p_{expose} = 0.55$ ,  $p_{sympt} = 0.6$ ,  $t_{inc} = t_{sympt} = 4$  and  $t_{asympt} = 10$ ).

**4.2.1 Validating the transmission from asymptomatic individuals.** Recent advances in epidemiology have introduced models that account for infectious individuals who do not develop symptoms, in an effort to estimate the portion of the population that may be transmitting a disease while asymptomatic. In [8], the peak of infectiousness and the relative infectiousness of asymptomatic individuals were varied, and the resulting proportion of the transmission from asymptomatic carriers is observed. Though our model differs from that used in the paper, we repeat the analysis and observe whether the assertions hold under our assumptions and methods.

For  $N = 1,000$ ,  $t_{sympt} = t_{inc} = 5$ , and  $t_{asympt} = 10$ , we ran 100 simulations for varied  $p_{sympt}$  and  $p_{asympt}$  ratios. We calculated the mean proportion of infectious individuals who never show symptoms ( $P_{ns}$ ) and the mean proportion of exposures that come from asymptomatic individuals ( $T_{ns}$ ) for each variation. For example, when  $p_{asympt} = 0.4$ , the mean proportion of transmissions coming from asymptomatic carriers was approximately 50%. This result is in tune with the proportion found in [8], in spite of the fact that our model uses deterministic lengths of infectiousness and a completely different infection dynamic. Other scenarios are shown in table 1, all of which point to a relationship between  $P_{ns}$  and  $T_{ns}$  that fits the following form:

$$K = \frac{T_{ns}(1 - P_{ns})}{P_{ns}(1 - T_{ns})}$$

For the scenarios used in our validation,  $K \approx 1.5$  holds for each of the  $p_{sympt}$  values. The equation above is another form of a relationship used in [8] to model the proportion of transmissions caused by asymptomatic individuals. This relationship holds under the model described in [8], providing further validation of our proposed model.

**Table 1**

$p_{asympt}$	$P_{ns}$	$T_{ns}$
0.4	$0.396 \pm 0.04$	$0.505 \pm 0.03$
0.5	$0.50 \pm 0.02$	$0.593 \pm 0.03$
0.6	$0.595 \pm 0.06$	$0.69 \pm 0.03$

**Table 1.** The impact of asymptomatic carriers on infections and transmissions ( $N = 1,000$ ,  $t_{sympt} = t_{inc} = 5$ , and  $t_{asympt} = 10$ , for 100 simulations)

The results shown in [8] are in accordance with the infection dynamics of our model, and prove that even a small proportion of asymptomatic infection can result in a majority of transmission being due to non-symptomatic individuals. We go forward from this result to further focus on how asymptomatic individuals interact with others in the population. First, we tune the parameters to match the infection rates of a realistic viral outbreak.

## 5. Tuning Model Parameters to Attain a Realistic Scenario

We increase the number of individuals in the population to  $N = 10,000$  and simulate the system for 50 time periods under different settings. For each setting, we investigate how the parameters affect the system dynamics. Specifically, we focus on the following aspects of the system:

1. Peak level of infectious individuals.
2. Number of susceptible individuals over time.

The parameters  $n_{cells}$  and  $p_{expose}$  were varied to create different settings. Figure 4 shows the number of infectious individuals over time. The results are quite intuitive: the larger the peak, the earlier it occurs. The right subplot in figure 4 displays how the number of susceptible individuals decreases over time under each set of parameters. An intuitive relationship between the two parameters and the population sizes can be derived from the figure: A greater number of cells and a greater probability of exposure both increase and push forward the peak of infectious cases, as well as speed the rate at which the number of susceptible individuals declines.

In figure 4 we see that different dynamics can be achieved using the model parameters, but it is still unclear how the model can be tuned to replicate realistic outbreak settings. For this purpose, we use a single epidemiological measurement to characterize our simulated outbreak. The  $R_0$  factor, representing the basic reproduction number, is equivalent to the number of susceptible persons a virus-carrying individual infects while he or she is infected. For a transmission via respiratory droplets, this measure is roughly 2.0. In [6], this quantity is defined as follows

$$R_0 = \int_0^{\infty} \lambda(a) da$$

where  $\lambda(a)$  is the transmission intensity function, representing the number of infections an infectious person transmits  $a$  days after becoming infectious.

In reality, this function depends on underlying parameters that can only be approximated. Additionally,  $\lambda(a)$  may vary from one individual to another, resulting in different  $R_0$  values for each infected individual. We also intend to tune our model to have realistic  $R_0$  not just at its onset or any other singular point in time, and so a better reference would be the time evolution of the reproduction number. Therefore, a different calculation is required. We consider two methods for computing the temporal evolution of  $R_0$ :

1. Compute the true value: Count the number of exposures each infectious individual causes, and compute the average number of persons exposed per infectious individual, arriving at the true value of  $R_0$ .
2. Predict using state variables: Approximate  $R_0$  by assuming that at any time  $t$ , the ratio of the exposed population's size to that of the infected population, letting  $R_0 \approx E(t)/I(t) = E(t)/(I_A(t)+I_S(t))$ .

The first method is nearly impossible to implement in the real world using traditional contact tracing tools. Large outbreaks may happen too fast and spread too quickly to permit accurate measurement of the number of exposures each infection causes, let alone the number of infections. However, the second method can be imitated using approximation of the exposed  $E$  and infectious  $I$  population sizes, which are possible to estimate using the classic SEIR model. In figure 5 we display the effect of changing  $p_{expose}$  and  $n_{cells}$  on  $R_0$ , and use both of the methods above to display the reproduction number. We consider two scenarios and show that an appropriate  $R_0 \approx 2.0$  can be achieved for a sustained period of time when  $p_{expose} = 0.33$  and  $N/n_{cells} = 1.6$ . Another interesting insight is that the approximation  $R_0 \approx E/I$  is quite accurate, especially for higher values of  $p_{expose}$ .

Using the same sets of parameters as before, we computed the mean percent error (MPE) between the true  $R_0$  and the approximation ratio  $E/I$ , whenever they are not equal to zero, for the four scenarios. Table 2 gives our results for simulations with  $N = 10,000$  individuals, and clearly shows that the estimation is equally accurate regardless of the density of the population. Specifically, whether there are on average 5 individuals in an interaction cell at any time ( $n_{cells} = 2,000$ ) or 1.6 individuals per cell ( $n_{cells} = 6,250$ ), the MPE between the true  $R_0$  and the  $E/I$  ratio will be relatively similar. However, the error in both cases is greater when  $p_{expose}$  is smaller. That is, when the probability of exposure at any interaction is smaller, our simulation shows that the MPE will be greater.

**Table 2**

$n_{cells} = 6,250$		$n_{cells} = 2,000$	
$p_{expose}$	MPE	$p_{expose}$	MPE
0.55	7.4%	0.55	7.8%
0.33	10.2%	0.33	9.1%

**Table 2.** Different sets of parameters are used to calculate  $R_0$  and the  $E/I$  ratio at every time period

of a simulation. The mean % error (MPE) between the two measures is shown for each parameter combination. ( $N = 10,000$ ,  $p_{asympt} = 0.4$ ,  $t_{inc} = t_{sympt} = 5$  and  $t_{asympt} = 10$  for all simulations, ending after 50 time periods).

## 6. Future Work and Extensions

Our model has the potential to help in addressing real public health problems. For example, contact tracing as a solution to viral outbreaks can be improved by preemptively taking into account the possibility of asymptomatic transmission. Our model simplifies the interaction dynamics for a population using the interaction cell formulation, which makes it possible to track interaction history and quantify the likelihood that any particular infection was caused by an asymptomatic carrier.

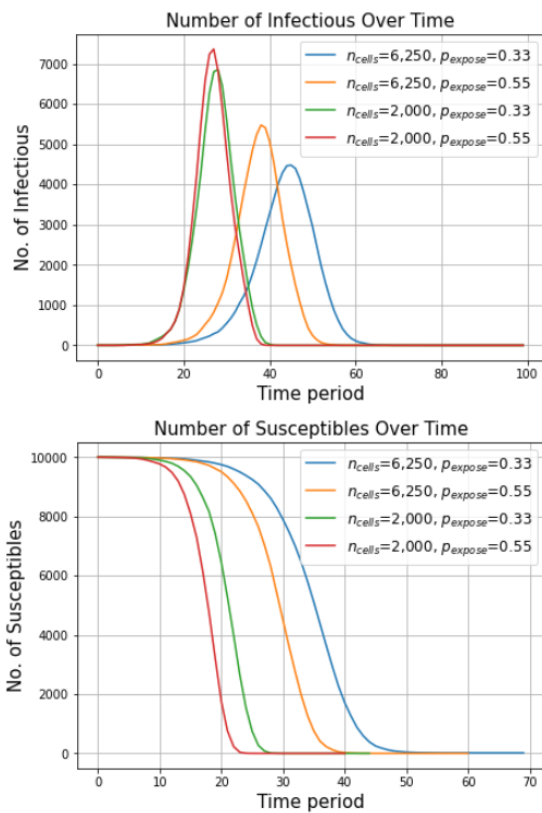
Additionally, using interaction data and known infectious cases, our model can be used as a training-ground for more advanced statistical methods. For example, a machine-learning classifier could be used to identify asymptomatic carriers in the system and recommend they be isolated. Because each individual interaction that occurs in the system is quantified, historical data could be used by this classifier to surmise whether an individual has been causing infections asymptotically, and thus significantly impacting epidemiological and public-health initiatives during an outbreak.

## 7. Conclusions

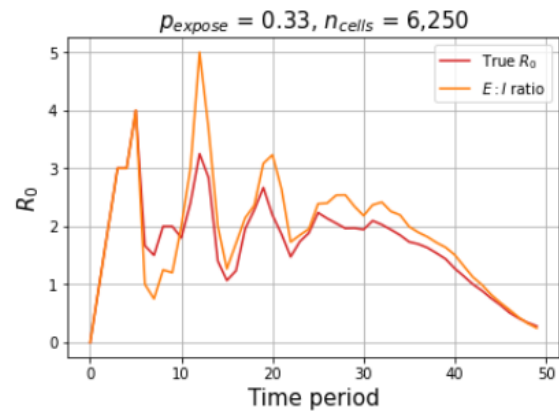
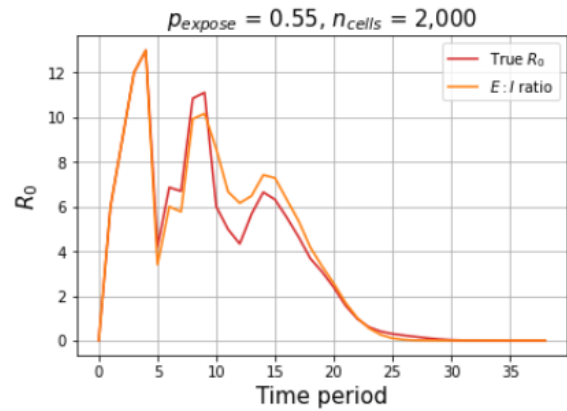
Our model extends the basic SEIR methodologies by allowing for asymptomatic carriers to exist in the system, and giving these carriers their own unique characteristics that differentiate them from infected individuals who will eventually become symptomatic. In tuning the model, we presented a method of estimating the reproduction number  $R_0$  and showed that it can be accurately estimated by the ratio of exposed  $E$  to infectious  $I$ . The discretization of the infection dynamic using infection cells allows for a direct analysis of the effect social isolation and distancing might have on transmissions. Similar relationships between model parameters and resulting infection rates can be studied and predicted by the model as well. We also replicated the results of a recent research work regarding asymptomatic carriers' existence in an epidemic. Our study showed that even a small proportion of undetected asymptotically infectious can be responsible for a significant portion of the overall transmissions. The model's validity is amplified by the fact that it allows the tracking of individual

interactions to be used for contact-tracing applications. Currently contact tracings systems do not seem to have a proven way for identifying asymptomatic carriers within the community. The robustness of our model may make it of value in a variety of use cases involving asymptomatic carriers during outbreaks of infectious diseases.

The model can be used as a basis for much more powerful healthcare applications. By analyzing the granular history of known infections, a statistical tool can directly classify likely asymptomatic carriers. These tools can actively inform policy makers on what the true rates of infection are among the population, preventing scenarios of misjudgments and improper resource allocation.



**Figure 4.** The effect of model parameters on the system. The top figure shows the evolutions of the infectious population  $I$  in the model. Greater  $n_{cell}$  and  $p_{expose}$  values result in a sharper and earlier peak in  $I$ . On the bottom, the susceptible population's size  $S$  over time is plotted for the different parameter combinations. Greater  $n_{cell}$  and  $p_{expose}$  values show a faster decline in  $S$ .



**Figure 5.**  $R_0$  over time under different  $p_{expose}$  and  $n_{cell}$ . The red curve displays  $R_0$  (the average number of exposures caused by an infectious individual). The orange curves show the ratio between the exposed  $E$  and the infectious  $I$  populations. In the bottom figure,  $R_0$  remains near a realistic value 2.0. ( $N = 10,000$  and  $p_{asympt} = 0.4$ ,  $t_{inc} = t_{sympt} = 5$ , and  $t_{asympt} = 10$ ).

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