Combining Behavioral Choice with a Branching Process Model of Disease

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2021 Sept DRAFT

Abstract

Whether a disease outbreak dies out early or expands into a full blown-epidemic depends not only on the average spread of disease, but also on the variation between individuals in how likely they are to spread the disease to others. The source of this variation is, in part, due to the fact that different people have differing levels of contact with others. In a behavioral model in which people choose the level of contact they have with others in response to new of disease outbreak, highly connected people respond qualitatively different from people with few social connections. When transmissibility is high, highly connected people can become fatalistic.

1 Introduction

People differ in how much they desire connections with other people. This paper explores the effect this heterogeneity in social affinity has on the outbreak of disease, and on how people choose to respond to such an outbreak.

It's important to understand this variation because disease is spread through contact with other people. And because there is variance in the level of social contact people choose, so too is there variance in how prone people are to catch disease and spread it to others.

The variance of individual infectiousness has important implications for the spread of disease, especially in the earliest stages of an outbreak. Average statistics like the basic reproductive number, R_0, only tell us about the *expected* spread of a disease. When the distribution of individual infectiousness is more variable, a disease is more likely to quickly go extinct, but also tends to spread more explosively in those cases where it does turn into a full-blown epidemic (Lloyd-Smith et al. 2005).

As an example, imagine two different epidemic scenarios where $R_0 = 2$. This means that early on, when someone gets sick, they are expected to spread their disease to two other people on average.

Imagine first that the reason this happens is that every person always spreads the disease to *exactly* two other people: Alice is patient zero. She gets sick and infects Bob and Carlos, Bob infects Elijah and Francine, Carlos infects Grace and Hunter, etc. In this case, there's no variance between people in how much they spread the disease to others. The number of infections exactly doubles with each 'generation' of spread, and the disease grows exponentially out of control.

Now imagine a second scenario where there are two different types of people, who behave differently after getting sick. The first type of people are shut-ins, who stay home and spreads the disease to zero other people. The second type are superspreaders, who go clubbing and spread the disease to 200 people. If 99% of people who get sick are the type who stay at home, while 1% are the type to go clubbing, then each sick person will, on average and early on, infect 2 other people. This means that $R_0 = 2$, just as it does in the first scenario. But unlike in the first scenario, there is uncertainty in how the disease will spread. If Alice is patient zero, and she stays out home, then the disease outbreak will immediately go extinct and nobody else will get sick after Alice. If Alice is patient zero, and goes clubbing then the disease outbreak will very quickly spread out of control to hundreds of people.

Real-world diseases don't tend to exhibit such starkly contrasting distributions in their *individual reproductive numbers*[1], but the outbreak of respiratory pathogens - like the SARS family of coronaviruses - has been characterized by superspreading events not too dissimilar to the second imagined scenario above. In the 2002-2004 SARS outbreak, approximately 80% of infections were caused by only about 20% of infected individuals (Lloyd-Smith et al. 2005) and the COVID-19 epidemic (caused by a SARS variant) has exhibited similarly skewed patterns of spread (Endo et al. 2020). Korea's patient 31 is an illustrative example of this phenomenon. South Korea initially managed to contain the spread of COVID-19. But patient 31, after checking into the hospital with COVID-19-like symptoms, subsequently left the hospital to get food at a buffet and attend a crowded church service, singlehandedly infecting hundreds of people (Hernandez, Scarr, and Sharma 2020).

This paper explores the hypothesis that the heterogeneity in the spread of respiratory disease is driven by heterogeneity in social affinity, by which I mean the extent to which people desire in-person interactions with others. In this paper, I construct a simple equilibrium model of disease spread, in which people choose social activity levels in response to the severity of an epidemic, and the severity of the epidemic is determined by the distribution of social activity levels

[1] In (Lloyd-Smith et al. 2005), the spread of disease is modelled by assuming that the number of people each infected person infects is determined by a person-specific poisson process, and refers to the mean of a person's process as that person's "individual reproductive number". Among the models they try, they find that the spread of SARS is best characterized by gamma-distributed individual reproductive numbers, which results in the overall distribution of offspring infections being negative binomial. This negative binomial "offspring distribution" is then the basis of the model used in (Endo et al. 2020) to estimate overdispersion in the outbreak of COVID-19.

that people choose.

The two main influences on my model are as follows.

The first is the behavioral model from (Kremer 1996). In this model, people choose their desired rate of new sexual partners in response to the prevalence of the disease among potential partners. However, the spread of HIV is qualitatively different from the spread of a respiratory infection, and so the model from this paper isn't directly applicable to understanding a disease like COVID-19. But some of my conclusions, such as the fact that high-activity people might become 'fatalistic' and respond to intervention in counterintuitive ways, are similar to the results from this paper. In particular, the exogenous rate at which people die is a key parameter in Kremer's model, and that paper is able to construct a steady-state equilibrium, in which people become newly infected at the same rate that people die. An acute respiratory epidemic, by contrast, has a large portion of the population quickly becoming sick, and then dying or recovering in a relatively short time span. The long-run steady state of such an epidemic, barring the ability of the reinfect at high enough rates to achieve fixation in the population, is one in which the disease eventually dies out after infection some portion of the population. Rather than modelling a disease which attains a steady-state equilibrium in its prevalence at some specific point in time, the model I use looks at the entire epidemic as a single event, based on the statistical properties of a network of individuals.

The second influence is (Newman 2002), which characterizes the fate of a disease in terms of the properties of an underlying network of people through which the disease is presumed to spread. In contrast to a standard compartmental epidemiological model, in which a system of differential equations describe the rate of change in prevalence at time t as a function of the numbers of infected and susceptible people at time t, this approach explicitly models discrete *generations* of a disease, and lacks an explicit representation of time itself. This approach is better able to model the effects of social heterogeneity on disease outbreak, and has been used to quantitatively analyze the effects of policy interventions (Meyers et al. 2005)(Bansal, Pourbohloul, and Meyers 2006), but in the context of an exogenous social graph. I partially endogenize the structure of the social network.

In Section 2, I describe a model which incorporates behavioral choice into the social-network based model of disease from (Newman 2002). In Section 3, I describe some of the properties of this model. In Section 4, I describe some illustrative example equilibria.

2 Model

In brief, this model will require two conditions for equilibrium to hold. Firstly, each person is responding to the spread of disease in a way that is best for them And secondly, the spread of disease follows a pattern consistent with the

responses that people choose.

In addition to parameters describing people's preferences, equilibrium will also depend on a parameter T, representing how transmissive the disease is. Given people's preferences and T, equilibrium will be characterized by endogenous parameters $\{N_i\}$ and Ψ. $\{N_i\}$ represents the decisions people make about social activity levels, while Ψ is a parameter describing the "edge risk", or the risk that one of a person's neighbors gets them sick. All of these parameters will be described in more detail in the following subsections.

In section 2.1, I describe the social network. Section 2.2 describes how disease spreads through this network, given $\{N_i\}$. Section 2.3 describes how the edge risk Ψ is determined, and 2.4 describes how likely an individual is to get sick as a function of Ψ. Section 2.5 describes how $\{N_i\}$ is chosen. And section 2.6 defines equilibrium in this environment.

2.1 The Social Network

There are infinitely many people, differentiated by type $i \in \{1, 2, 3, ..., I\}$. The relative population of each type i is $\alpha_i > 0$, such that $\sum_i \alpha_i = 1$. These types correspond to different levels of *social affinity*, meaning that different types desire different levels of social activity.

Each person has only a single choice to make: the level of social activity they desire. The *level of social activity* chosen by each person of type i is denoted by N_i. This activity level can be any non-negative real number: $N_i \in [0, +\infty)$.

The spread of disease occurs through an infinite *social network*, in which individual people are the *vertices*, and the physical social connections between them are the *edges*. The degree of each vertex must be an integer value, but the level of social activity need not be. If a person has social activity level N_i, then their number of edges in the social graph is determined by a Poisson process with mean ${N_i}^2$, such that the probability of a type i vertex having exactly k edges is

$$\frac{N_i^k e^{-N_i}}{k!} \tag{1}$$

This also means that the chance a randomly selected vertex has degree k is given by

$$p_k \equiv \sum_i \alpha_i \frac{N_i^k e^{-N_i}}{k!} \tag{2}$$

[2]It is common in models of disease to assume that each individual spreads disease to others via a Poisson process. See for example, the discussion in (Lloyd-Smith et al. 2005). In addition to make the math simpler, this assumption that the numbers of physical contacts are generated via a poisson process about N_i will also ensure that each individual spreads disease Poisson-wise, with mean $N_i T$.

While the degree of each vertex is influenced by the decisions about social activity level, the edges are otherwise determined randomly[3], and people don't know the social activity nor type of their neighbors.

2.2 The Spread of Disease

In this model, disease expands through the social network by spreading from person to person through the edges of the network. Each edge, which represents the presence of a physical social connection between two people, is marked as 'opened' with independent probability T, or 'closed' with probability $1 - T$. The parameter T depends on the disease in question, and represents the chance that this disease will spread along a connection if one of the two connected people gets sick.

T is an exogenous parameter, representing the *transmissivity* of the disease. The *transmissible social network* is the original *social graph*, but each edge has been removed with iid probability $1 - T$. And it is this *transmissible social network* along which the disease can spread. If an edge is "open to the spread of disease", it means that this dice role has kept that edge in the transmissible social network.

A spontaneous outbreak starts by introducing the disease to one person, Patient 0, chosen at random. Then for each neighbor who shares an edge with Patient 0, there is an independent chance T that that neighbor also gets sick. It's possible that none of their neighbors get sick, (if Patient 0 has k neighbors, then this happens with probability $(1 - T)^k$), in which case the outbreak *dies out* before turning into an epidemic. If some of Patient 0's neighbors do get sick, then each of *those* people can transmit the disease to *their* neighbors, also with probability T for each edge.

The disease can't spread back to someone who has already become sick, like Patient zero. Otherwise, this process continues either forever or until the outbreak dies out. If the process continues forever, then infinitely many people will get sick, and the outbreak has becomes an *epidemic*.

An equivalent way to think about this process is that the edges of the social graph are preemptively removed, each with probability T, and the set of edges that remain give us a subset of the original social network, called the *transmissible social network*. After introducing a disease to a random person, the set of people who get sick is simply the set of people in Patient 0's connected component in the transmissible social network.

Given this setup, (Newman 2002) provides the following results about the spread of a disease.

[3]See the appendix for more details about how the social network is characterized.

2.2.1 Critical Transmissibility Threshold

Firstly, given the distribution of degrees in the social network, there is a *critical transmissibility threshold* T_c, such that an epidemic occurs with zero probability whenever $T \leq T_c$. If p_k is the probability that a randomly selected person has exactly k neighbors, then

$$T_c = \frac{\sum_k p_k k}{\sum_k p_k k(k-1)} \tag{3}$$

Intuitively, this is simply the inverse of the edge-weighted average of the number of physical contacts people have. If the disease travels along a random edge to Bob, who has degree k, then there are $(k-1)$ additional edges along which Bob can spread the disease to others. (Exclude the connection that the disease travelled along to *get to* Bob.) If $t < \frac{1}{k-1}$, then Bob will infect fewer than one person on average.

The additional factor k in the denominator of T_c comes from the fact that a person with many connections along which they can transmit the disease also has many connections along which they can contract the disease. So weight $(k-1)$ by the number of edges people have to get $\frac{\sum_k p_k k(k-1)}{\sum_k p_k k}$. If T is less than the reciprocal of this quantity then each newly infected person will infect fewer than one people on average, and the disease will almost surely die out.

In fact, the familiar basic reproductive rate, meaning the average number of people each sick person transmits to, is simply $R_0 = \frac{T}{T_c}$.

In the environment of my model, there are multiple types and the distribution of degree with each type is poisson with mean N_i. So $p_k = \sum_i \alpha_i \frac{N_i^k e^{-N_i}}{k!}$, and the generic formula from (Newman 2002) becomes:

$$T_c(\{N_i\}) \equiv \frac{\sum_k \sum_i [\alpha_i \frac{N_i^k e^{-N_i}}{k!} k]}{\sum_k \sum_i [\alpha_i \frac{N_i^k e^{-N_i}}{k!}(k-1)k]} = \frac{\sum_i \alpha_i N_i}{\sum_i \alpha_i N_i^2} \tag{4}$$

In particular, consider the case where there is only one type $i = 1$. Then the degree distribution of the social network is Poisson with mean N_1, and the critical transmissibility threshold is

$$T_c(\{N_i\}) = \frac{N_1}{N_1^2} = \frac{N_1}{N_1^2} = \frac{1}{N_1} \tag{5}$$

In other words, when there isn't variation between the types of people, the critical threshold for epidemics is simply that T is greater than the reciprocal of the social activity level.

2.2.2 Epidemic Probability

Thirdly, when $T > T_c$, an epidemic is possible, but not necessarily guaranteed. The probability that an random outbreak *does* turn into an epidemic is given by

$$R_\infty \equiv 1 - \sum_k [(1 - [1 - \upsilon]T)^k p_k] \tag{6}$$

where υ is the edge-weighted chance that a neighbor remains uninfected[4], and is the solution $\upsilon \in (0, 1)$ to

$$\upsilon = \frac{\sum_k [(1 - (1 - \upsilon)T)^k k p_k]}{\sum_k k p_k} \tag{7}$$

R_∞ is also the expected portion of people who do get eventually sick from the epidemic, which is one way to make sense of the equation above. In this model of disease, these two quantities – the probability an epidemic occurs and the fraction of people who get infected – are the same because they both describe the relative size of a connected component of the transmissible social network.

In the environment of my model, with $p_k = \sum_i \alpha_i \frac{N_i^k e^{-N_i}}{k!}$, the formula for R_∞ becomes

$$R_\infty \equiv 1 - \sum_k \sum_i [\alpha_i \frac{N_i^k e^{-N_i}}{k!} (1 - [1 - \upsilon]T)^k] = 1 - \sum_i \left[\alpha_i e^{-(1-\upsilon)TN_i}\right] \tag{8}$$

And υ is defined as the solution to

$$\upsilon = \frac{\sum_k \sum_i [k \alpha_i \frac{N_i^k e^{-N_i}}{k!} (1 - [1 - \upsilon]T)^k]}{\sum_k \sum_i [\alpha_i \frac{N_i^k e^{-N_i}}{k!} k]} = \frac{\sum_i [\alpha_i N_i e^{-(1-\upsilon)TN_i}]}{\sum_i [\alpha_i N_i]} \tag{9}$$

When there is only a single type, the formula for υ simplifies to

$$\upsilon = \frac{N_1 e^{-(1-\upsilon)TN_1}}{N_1} = e^{-(1-\upsilon)TN_1} \tag{10}$$

[4]More technically, υ is the chance that the end of a randomly chosen edge would remain uninfected from a random outbreak if that randomly chosen edge were removed from the graph.

2.3 The Risk of Disease from a Neighbor

Now imagine you are a person in this network, and you have a neighbor named Ann. You want to know how likely Ann is to transmit the disease to you. Two things must be true for this to happen. Firstly, the edge between you and Ann must be open to the spread of disease, which happens with probability T. And secondly, Ann herself must get sick from one of her neighbors for her to pass the disease onto you. If $T < T_c$, then Ann almost surely won't get sick, and so the chance she infects you is also zero. If $T > T_c$, then the chance she gets sick from one of her other neighbors is $(1 - \upsilon)$, and the chance she gets you sick is $(1 - \upsilon)T$.

Let Ψ denote the *risk of disease from a neighbor*, or "Edge Risk" for short, where

$$\Psi \equiv \begin{cases} 0 & \text{if } T \leq T_c \\ (1-\upsilon)T & \text{if } T > T_c \end{cases} \tag{11}$$

And let $\Psi^*(\{N_i\})$ denote the value of Ψ, taken as a function of social activity levels. Recall that T_c is a function of $\{N_i\}$ and write:

$$\Psi^*(\{N_i\}, T) \equiv \begin{cases} 0 & \text{if } T \leq T_c(\{N_i\}) \\ \text{the solution } \Psi \in (0,1) \text{ to } \Psi = T\frac{\sum_i A_i N_i (1-e^{-\Psi N_i})}{\sum_i A_i N_i} & \text{if } T > T_c(\{N_i\}) \end{cases} \tag{12}$$

Note that $\Psi^*(\{N_i\}, T)$ is continuous on $\{N_i\} \in \mathbb{R}^I_{\geq 0}$, (but isn't differentiable on that entire domain).

Also note that if there is only a singular type, then the formula which determines Ψ when $T > T_c(\{N_i\})$ becomes[5]

$$\Psi = T\frac{N_1(1-e^{-\Psi N_1})}{N_1} = T \cdot (1 - e^{-\Psi N_1}) \tag{13}$$

which is simply the transmissibility T multiplied by the probability that a given person gets sick.

2.4 The Individual's Risk.

Again imagine that you are a person in this environment, and you take $\Psi \in [0,1]$ as given[6]. Ψ is the chance that a neighbor gets sick and subsequently transmits

[5] In the singular-type case, this formula for Ψ can be explicitly solved to give $\Psi = \frac{W_0(-TN_1e^{-TN_1})}{N_1} + T$, where W_0 is the principle branch of the Lambert product log. Knowing this isn't particularly enlightening, however.

[6] Each person in this model is an infinitesimally small portion of the entire infinite social graph, and thus is assumed not be able to individually affect the overall pattern of disease outbreak.

their disease to you.

If you have precisely k neighbors, then the chance you *don't* get sick from a random outbreak is $(1-\Psi)^k$, and the chance that you do get sick is $1-(1-\Psi)^k$.

But in this model, people don't directly choose k. They choose N_i, which generates k from a Poisson process with mean N_i.

So if you choose a level of social activity N_i, then the chance you get sick is

$$P(N_i;\Psi) = \sum_k \left[(1-[1-\Psi]^k) \frac{N_i^k e^{-N_i}}{k!} \right] = 1 - e^{-\Psi N_i} \tag{14}$$

This is the expected value of $1-(1-\Psi)^k$, averaged out over the Poisson degree distribution which is generated by your choice of N_i.

2.5 The Individual's Decision

Each person takes Ψ as given and uses it to make their decisions about social activity level.

The payoff for a person of type i is:

$$U_i(N_i;\Psi) = u_i(N_i) - \delta_i P(N_i;\Psi) \tag{15}$$

Where $u_i(N_i)$ is the utility gained from social activity level[7], independent of the severity of the outbreak, $P(N_i;\Psi)$ is the probability that a person with social activity level N_i will get sick at some point during the outbreak, and δ_i is the disutility from getting sick[8].

The utility maximization problem for a person of type i is to choose $N_i \in \mathbb{R}_+$ such that N_i solves

$$\max_{N_i \geq 0} \left[u_i(N_i) - \delta_i P(N_i;\Psi) \right] \tag{16}$$

As discussed above, in this environment, $P(N_i,\Psi) = 1 - e^{-\Psi N_i}$

For convenience, I want to choose a u_i such that the total utility $U_i(N_i;\Psi)$ is continuous and concave down, and such that $N_i^*(\Psi)$, the person's optimal choice of N_i, is a continuous and bounded function of Ψ over $\Psi \in [0,1]$.

If δ_i is normalized to 1 for all i, then the following function has these desired properties:

[7] Note that this is set up so that the person is getting utility from their social activity level, which is the mean of the number of actual social connections they will have. As written, they are not getting utility from the physical social connections themselves.

[8] Equivalently, δ_i is the utility of remaining healthy, which is lost with probability $P(N_i;\Psi)$.

$$u_i(N) = \ln(N/\theta_i) - \frac{N}{\theta_i} \tag{17}$$

In the function above, θ_i can be thought of as the *social affinity* of a person of type i.
In the absence of an epidemic, when $\Psi = 0$, a person of type i desires a level of social activity $N_i^*(0) = \theta_i$.

2.6 Equilibrium Definition

Similarly to the equilibrium in (Kremer 1996), equilibrium in this model is defined by a behavioral condition (bc) and an epidemiological condition (ec). The behavioral condition is that taking Ψ as given, each individual chooses the level of social activity best for them. The epidemiological condition is that these decisions lead to an outbreak that generates Ψ.

To put it more explicitly:

Definition 1. Given exogenous T, $\{\alpha_i\}$, an equilibrium in this model consists of $\Psi, \{N_i\}$ such that

$$\Psi = \Psi^*(\{N_i\}, T) \tag{ec}$$

$$N_i = \arg\max_{N_i \geq 0} U_i(N_i; \Psi) \tag{bc}$$

3 Equilibrium Properties

3.1 Existence of Equilibrium

Equilibrium occurs when the epidemic that people are reacting to is consistent with the epidemic that their reactions create. As long as their reactions are "smooth" across different values of Ψ, there will be at least one value of Ψ that is part of an equilibrium.

Proposition 1. *If for each i, the optimal response $N_i^*(\Psi)$ is a continuous non-negative function on $\Psi \in [0, 1]$, then an equilibrium exists.*

Proof. By assumption, $\{N_i^*(\cdot)\}$ is a continuous function from $[0, 1]$ into $\mathbb{R}^I_{\geq 0}$, and $\Psi^*(\cdot, T)$ is a continuous function from $\mathbb{R}^I_{\geq 0}$ into $[0, 1]$. So $\Psi^*(N_i^*(\cdot), T)$ is continuous function from $[0, 1]$ into $[0, 1]$. And so there is at least one fixed point $\bar{\Psi} \in [0, 1]$ such that $\Psi^*(N_i^*(\bar{\Psi}), T) = \bar{\Psi}$. This value of $\Psi = \bar{\Psi}$, together with the set $\{N_i^*(\bar{\Psi})\}$ form an equilibrium for $T, \{\alpha_i\}$. □

Given the edge risk, Ψ, people's social activity levels in equilibrium will be $\mathbb{N}_i^*(\Psi)$. And given people's activity levels, $\{N_i\}$, the resulting equilibrium edge risk will be $\Psi^*(\{N_i\}, T)$. Ψ can be thought of a measure of the severity of an epidemic. If $\Psi^*(\{N_i^*(\Psi)\}, T) > \Psi$, then Ψ is too low to give an equilibrium; people's lax responses to such a mild epidemic would lead to a more severe epidemic. Likewise, if $\Psi^*(\{N_i^*(\Psi)\}, T) < \Psi$, then people's responses to Ψ would result in a an epidemic with lower severity.

3.2 The Trivial Non-Epidemic Equilibrium

When there is zero risk of catching a disease from a neighbor, people can choose their social activity levels without considering their risk of exposure to disease. That is, $P(N_i; 0) = 1 - e^0 = 0$, and so $U(N_i; 0) = u_i(N_i)$. The set $\{N_i^*(0)\}$ then represents the 'as normal' levels of social activity, which people would choose if not for the risk of disease. And the value $\Psi^*(\{N_i^*(0)\}, T)$ describes the pattern of disease spread that would result from people ignoring the disease.

In particular, with the utility function from Equation (17), this means that each type i chooses $N_i = \frac{\theta_i}{c}$.

If these baseline social activity levels are moderate enough to lead to $\Psi^*(\{N_i^*(0)\}, T) = 0$, then there is an equilibrium where $\Psi = 0$ and epidemic never happens.

> **Proposition 2.** *If $T \leq T_c(\{N_i^*(0)\})$, then there is an equilibrium where $\Psi = 0$ and $\{N_i\} = \{N_i^*(0)\}$.*

Proof. By definition, when $T \leq T_c$, $\Psi^* = 0$. The result follows. □

Essentially, if epidemic can always be avoided without any change in behavior, then the equilibrium is the one in which there is never an epidemic.

3.3 Individual Fatalism

Note that while the chance of getting sick $P(N_i, \Psi)$ is an increasing function of both edge risk Ψ and social activity level N_i, the marginal disease risk may actually *decrease* as Ψ goes up. That is, the following term can be either positive of negative:

$$\frac{\partial}{\partial \Psi} \frac{\partial}{\partial N_i} [1 - e^{-\Psi N_i}] = (1 - \Psi N_i) e^{-\Psi N_i} \tag{18}$$

When this cross-derivative is negative, an increase in Ψ will decrease the marginal risk of additional social connections at a given value of N_i, thus potentially leading to a situation in which high-activity people become "fatalistic", meaning

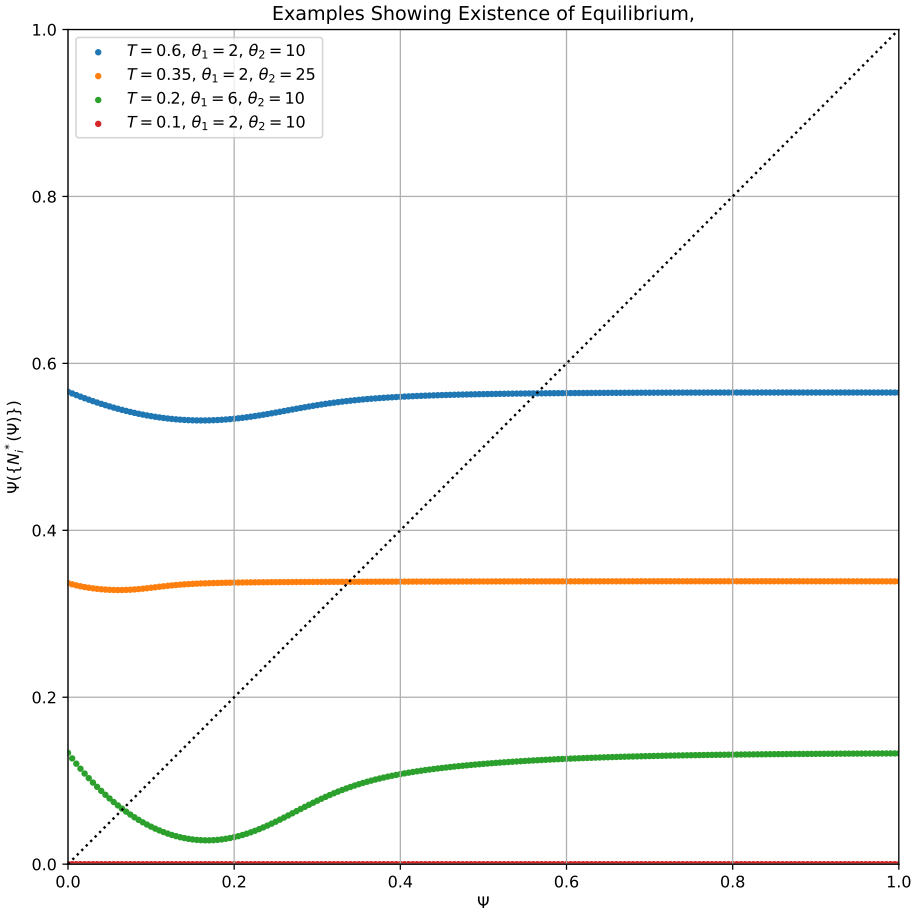

Figure 1: This figure shows the determination of several different equilibriums, each consisting of two types of people. In all three of these examples, $\alpha_1 = \alpha_2 = 0.5$. Each curve represents a different set of parameters T, θ_1, θ_2, and equilibrium Ψ occurs where a curve intersects the 45 degree line. The red line represents a trivial equilibrium where epidemic cannot occur. This happens because the value of T is below the critical threshold even for the highest desired level of social activity.

a situation wherein a higher risk of getting sick may counterintuitively lead to *higher* levels of social activity, N_i^*.

This "fatalism threshold" occurs at $N_i = \frac{1}{\Psi}$, which corresponds to a probability of getting infected of $p(N_i) = 1 - \frac{1}{e} \approx 63.2\%$. Figure 2 illustrates this threshold.

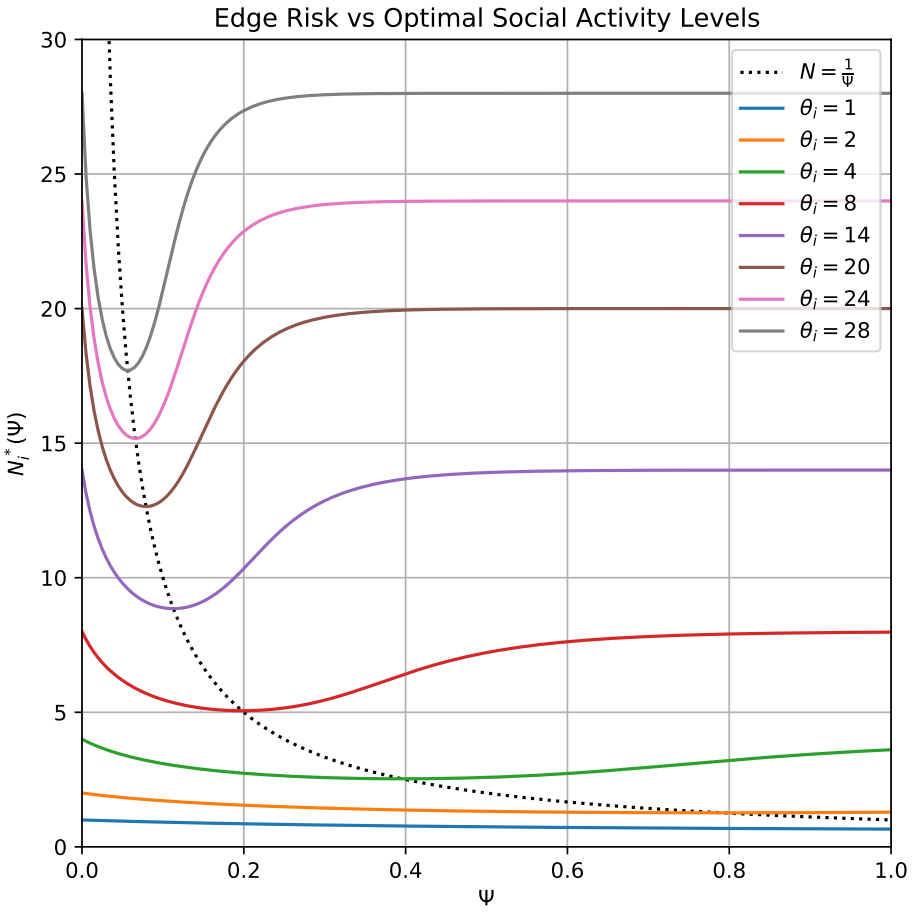

Figure 2: Optimal social activity level $N_i^*(\Psi)$ for various levels of social affinity θ_i when $U_i(N_i; \Psi) = \ln(N/\theta_i) - \frac{N}{\theta_i} - (1 - e^{-\Psi N_i})$. The dotted line marks the "fatalism threshold", past which the marginal risk of getting sick from additional social activity decreases when edge risk goes up.

3.4 Partial Equilibrium: When Can an Increase in Social Activity Decrease Edge Risk?

Suppose type j are highly introverted and have a very low social activity level. Holding the behavior of all the other types of people constant, what happens to

the edge risk $\Psi^*(\{N_i\})$ when N_j increases?

If $T < T_c$, then nothing happens at the margins. Epidemic will still be impossible.

When $T > T_c$ and epidemic is possible, then the marginal effect of type j's social activity is ambiguous. On the one hand, an increase in activity means that type js are more likely to get sick, and when type js do get sick, they will spread the disease to more people. On the other hand, type j might decrease Ψ by diluting the pool of neighbors, and reducing the likelihood others will match with even higher-activity people.

If the indirect effect from dilution outweighs the direct effect of increased disease exposure and spread for type j, then a marginal increase in N_j will decrease the value of $\Psi^*(\{N_i\})$, holding the value of all $\{N_{-j}\}$ constant. The following proposition provides a necessary and sufficient condition for when this occurs:

Proposition 3. *Assume $\{N_i\}$ is such that $T > T_c(\{N_i\})$. When this is the case,*

$$\frac{\partial \Psi(\{N_i\})}{\partial N_j} < 0$$

$$\Updownarrow$$

$$(1 - e^{-\Psi(\{N_i\})N_j}) < \frac{\Psi(\{N_i\})}{T}(1 - TN_j e^{-\Psi(\{N_i\})N_j})$$

Proof: See the appendix for proof.

The left-hand side of this equation is simply $P(N_j; \Psi)$, the probability that type j will get sick. On the right-hand side, the term $\frac{\Psi(\{N_i\})}{T}$ is equal to the likelihood that a random neighbor gets sick.

Note also that the term $\left(1 - TN_j e^{-\Psi(\{N_i\})N_j}\right)$ is less than 1 when TN_j is nonzero.

So this condition can also be interpreted as: For type j's social activity level to have positive externalities at the margin ($\frac{\partial \Psi(\{N_i\})}{\partial N_j} < 0$), it must not only be true that people of type j have lower social activity than the typical neighbor, but significantly less so.

The above necessary-and-sufficient condition leads to the following condition which is merely necessary, but more intuitively understandable: For an increase in the social activity level of type j to reduce the edge risk when all other $\{N_{-j}\}$ are held constant, it must be that the social activity of type j is no larger than the reciprocal of the transmissibility parameter T. Or to express the contrapositive of that condition:

Corollary 3.1. *If $N_j > \frac{1}{T}$ and $T > T_c(\{N_i\})$, then $\frac{\partial \Psi(\{N_i\})}{\partial N_j} > 0$.*

Proof. When $T > T_c$, $\upsilon \in (0,1)$ and $\Psi = (1-\upsilon)T \in (0,T)$, so $\frac{\Psi(\{N_i\})}{T} < 1$.

When $N_j > \frac{1}{T}$, $N_j T > 1$, and so $1 - TN_j e^{-\Psi(\{N_i\})N_j} < 1 - e^{-\Psi(\{N_i\})N_j}$.

Therefore, if $N_j > \frac{1}{T}$ and $T > T_c(\{N_i\})$, then

$$\frac{\Psi\left(\{N_i\}\right)}{T} \cdot \left(1 - TN_j e^{-\Psi(\{N_i\})N_j}\right) < 1 - e^{-\Psi(\{N_i\})N_j} \tag{19}$$

and so $\frac{\partial \Psi(\{N_i\})}{\partial N_j} > 0$, as per above. □

It's worth noting that expected number of secondary infections caused by an infected person of type j is $N_j T$. So one way to interpret the above result is that if a type of person already infects more than one person on average, then increasing the activity levels of that type of person can only worsen the epidemic.

Corollary 3.2. *If there is only a singular type and $T > T_c(\{N_i\})$, then $\frac{\partial \Psi(\{N_i\})}{\partial N_1} > 0$*

Proof. When there is only a singular type, the critical threshold is simply $T_c(\{N_i\}) = \frac{1}{N_1}$. So if $T > T_c(\{N_i\})$, then $T > \frac{1}{N}$, and so (because both T and N are non-negative) $N_1 > \frac{1}{T}$. Apply the previous theorem, and you get the result. □

Intuitively, this makes sense. The only way that additional activity can diminish the spread of disease is by displacing connections with even riskier people. With only one type of person, there is no way for this displacement to happen, and so additional social activity must make the epidemic worse, if it has any effect at all.

Corollary 3.3. *If $N_j > \frac{1}{\Psi(\{N_i\})}$ and $T > T_c(\{N_i\})$, then $\frac{\partial \Psi(\{N_i\})}{\partial N_1} > 0$*

Proof. When $T > T_c(\{N_i\})$, $0 < \Psi(\{N_i\}) < T$ and so $\frac{1}{\Psi(\{N_i\})} > \frac{1}{T}$.

If $N_j > \frac{1}{\Psi(\{N_i\})}$, then $N_j > \frac{1}{T}$, and the result follows.

□

The interpretation of this is that a fatalistic person cannot reduce the edge risk by increasing their social activity level. By the time that a person has a high enough risk of getting sick that they become fatalistic and start increasing their

social activity level in response to increased edge risk, it is too late for this increased social activity to do any good for the rest of society.

4 Example Equilibria

4.1 Example Equilibrium with a Singular Type

In this section, I examine an equilibrium in this environment when there is only one type of person, $j = 1$. This means that $\alpha_1 = 1$, and the degree distribution of the underlying social network is poisson with mean N_1.

In this example, the critical transmissibility threshold from Equations (3),(4) becomes

$$T_c = \frac{1}{N_1} \tag{20}$$

This means that if $T \leq \frac{1}{N_1}$, then an outbreak can never turn into an epidemic. And so the formula for edge risk from Equation 12 becomes

$$\Psi^*\left(N_1, T\right) \equiv \begin{cases} 0 & \text{if } T \leq \frac{1}{N_1} \\ \text{the solution } \Psi \in (0,1) \text{ to } \Psi = T \cdot (1 - e^{-\Psi N_1}) & \text{if } T > \frac{1}{N_1} \end{cases} \tag{21}$$

As an example, let $\theta_1 = 5$ in the utility function from Equation 17, and let the disutility from becoming sick be $\delta_1 = 1$. Then each person's utility maximization problem, taking Ψ as given, is:

$$N_1^*(\Psi) = \arg\max_{N_1 \geq 0} \left[\ln\left(\frac{N_1}{5}\right) - \frac{N_1}{5} - (1 - e^{-\Psi N_1}) \right] \tag{22}$$

Given exogenous transmissibility T, an equilibrium in this environment consists of any pair $(\bar{\Psi}, \bar{N_1})$ that satisfy $\bar{\Psi} = \Psi^*(\bar{N_1}, T)$ from Equation (21) and $\bar{N_1} = N_1^*(\bar{\Psi})$ from Equation (22).

Allowing T to vary from 0 to 1 yields different equilibrium outcomes, which are plotted in Figure 3.

4.1.1 The Trivial Non-epidemic Equilibrium.

In the absence of disease, a person with the utility function from Equation 17 will choose $N_i^* = \theta_i$. In this example, $\theta_i = 5$, meaning that each person will choose 5 units as their social activity level when social activity doesn't expose them to the risk of getting sick. In other words, in this example,

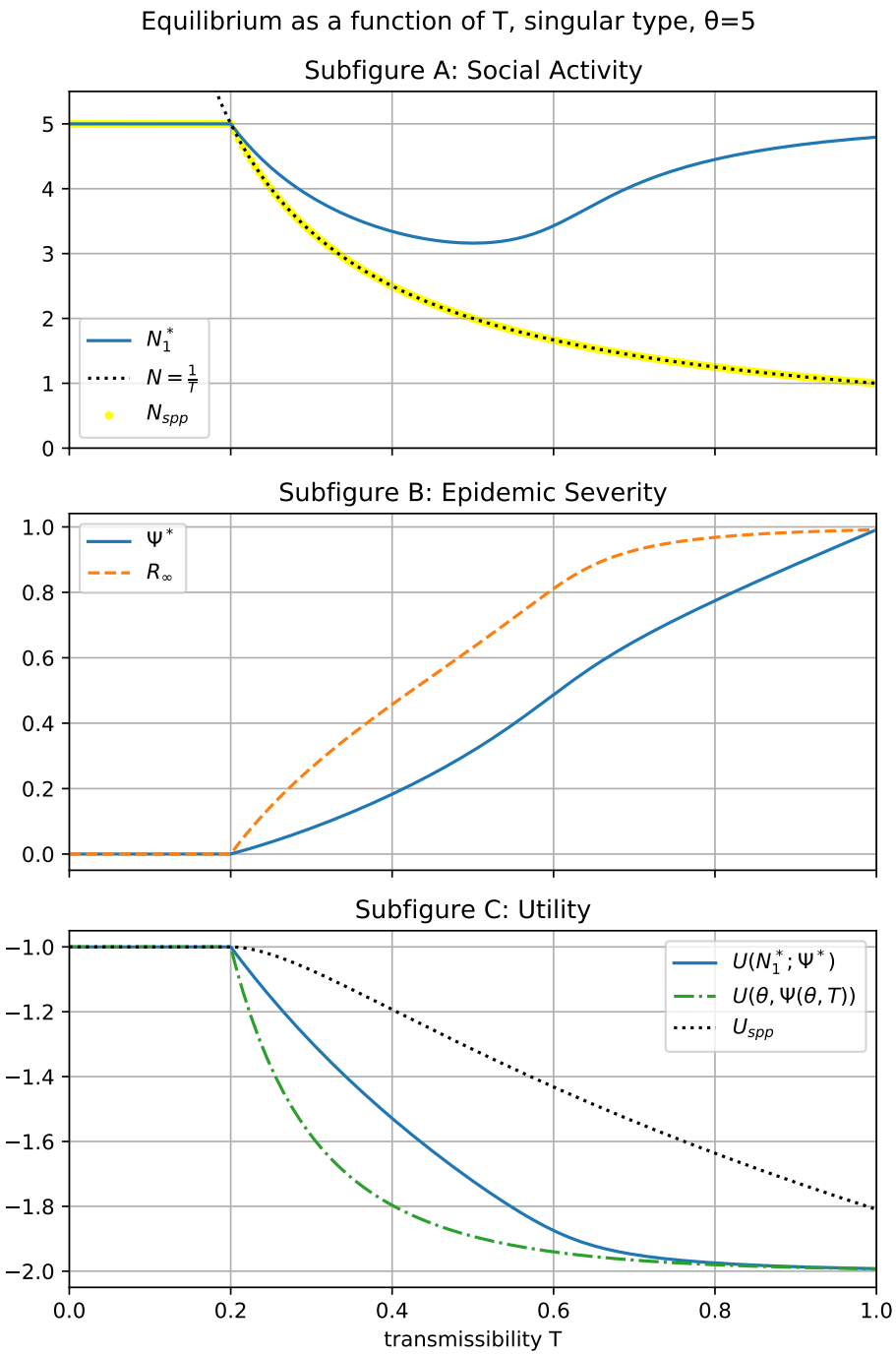

Figure 3: An example of equilibrium with only one type, at various levels of T.

$$N_1^*(0) = 5 \tag{23}$$

Then if $\Psi^*(5, T) = 0$, the pair $(\bar{\Psi}, \bar{N}_1) = (0, 5)$ will be an equilibrium. In this example, $\Psi^* = 0$ iff $T \leq \frac{1}{N_1}$, so if $T \leq \frac{1}{N_1^*(0)} = 0.2$, then the only equilibrium will be the one without disease.

This can be seen in Figure 3 as the flat region to the left in each graph.

4.1.2 Equilibria with Epidemics

In subfigure A of Figure 3, N_1^* is the equilibrium level of social activity for this example. The dotted line $N = \frac{1}{T}$ is the maximum level of social activity for which $T_c \leq T$, thus ensuring that a spontaneous outbreak never turns into an epidemic. The highlighted line N_{spp} is the non-equilibrium level of social activity which would maximize the welfare of each person. In this example, this socially optimal level of social activity is simply what the individuals would choose when that wouldn't cause an epidemic, and $1/T$ when it would.

Subfigure B depicts the equilibrium edge risk Ψ, along with the equilibrium chance that an epidemic occurs R_∞.

In subfigure C, $U(N_1^*; \Psi^*)$ is the equilibrium level of utility, the welfare of each person when they make their own decisions. Meanwhile, the dotted line U_{spp} is the maximal utility that each person could experience. In this example, whenever epidemic is possible in equilibrium, this dotted line also corresponds to the socially optimal level of utility; this is the highest possible welfare given transmissibility T. The gap between these two lines can be then be though of as the social costs that arise from the lack of coordination between people.

The green dashed line $U(\theta_1; \Psi^*(\theta_1, 0))$ depicts the utility that each person would have if they didn't adjust their behavior at all in response to disease. The gap between this line and equilibrium utility represents the gains in welfare from individuals trading social activity for a reduced risk of getting sick.

4.2 Example Equilibrium with Two Types

In this section I examine a slightly more complicated equilibrium in which there are two types of people: a "High-activity" type labelled H and a "Low-activity" type labelled L.

Taking Ψ as given, the utility maximization problem for each High-activity type person is:

$$N_H^*(\Psi) = \arg\max_{N_H \geq 0} \left[\ln\left(\frac{N_H}{20}\right) - \frac{N_H}{20} - (1 - e^{-\Psi N_H}) \right] \tag{24}$$

And likewise for each Low-activity person:

$$N_H^*(\Psi) = \arg\max_{N_L \geq 0} \left[\ln\left(\frac{N_L}{20}\right) - \frac{N_L}{20} - (1 - e^{-\Psi N_L}) \right] \tag{25}$$

The critical transmissibility threshold from Equation (4) in this example becomes:

$$T_c(N_H, N_L) = \frac{\frac{1}{2}N_H + \frac{1}{2}N_L}{\frac{1}{2}N_H^2 + \frac{1}{2}N_L^2} = \frac{N_H + N_L}{N_H^2 + N_L^2} \tag{26}$$

For the sake of the example, let $\alpha_H = \alpha_L = 0.5$, $\theta_H = 10$, and $\theta_L = 5$.

The solid lines in Figure 4 depicts how equilibrium in this example varies in response to transmissibility T. The dashed lines depict what the equilibrium outcomes would be if there were only a single type of person. The Low-activity type has the same utility function as in the previous example, and so the dashed lines for the Low-activity type depict the same equilibrium as in the previous example.

Note how the High-activity type begins to return to normal levels of social activity at a lower value of T than the Low-activity type. This is because the High-activity type's utility "bottoms out" as their probability of getting sick becomes very close to 1, and so they become fatalistic. Interestingly, the High-activity type people are the main driver of the severity of the epidemic, but because of the way utility is normalized, they also suffer from the largest drop in welfare.

Compared to what occurs in the singular-type equilibria, the equilibria with a mix of types increases both the value of T at which the High-activity type begins to reduce their social activity levels, and the value of T at which they become fatalistic. Conversely, the Low-activity types begin to reduce their activity and become fatalistic at lower values of T. This is because the behavioral response depends only on the value of Ψ, and the mixture of the two types moderates the equilibrium value of Ψ, as can be seen in Figure 4, subfigure B.

Looking at the equilibrium levels of utility in Figure 4, subfigure C, it appears that the presence of High-activity types lowers the utility of the Low-activity types, while the moderating influence of the Low-activity types increases the utility of the High-activity types by reducing the chance they get sick.

4.2.1 Can an Increase in Activity reduce Edge Risk?

In this example, with $\theta_H = 10$ and $\theta_L = 5$, the answer is no.

For an increase in the social activity level of some type to reduce the edge risk, it must be that the transmissibility T, together with the activity level of some type N_j and the edge risk $\Psi^*(T, \{N_i\})$ satisfy the condition described in Proposition 3.

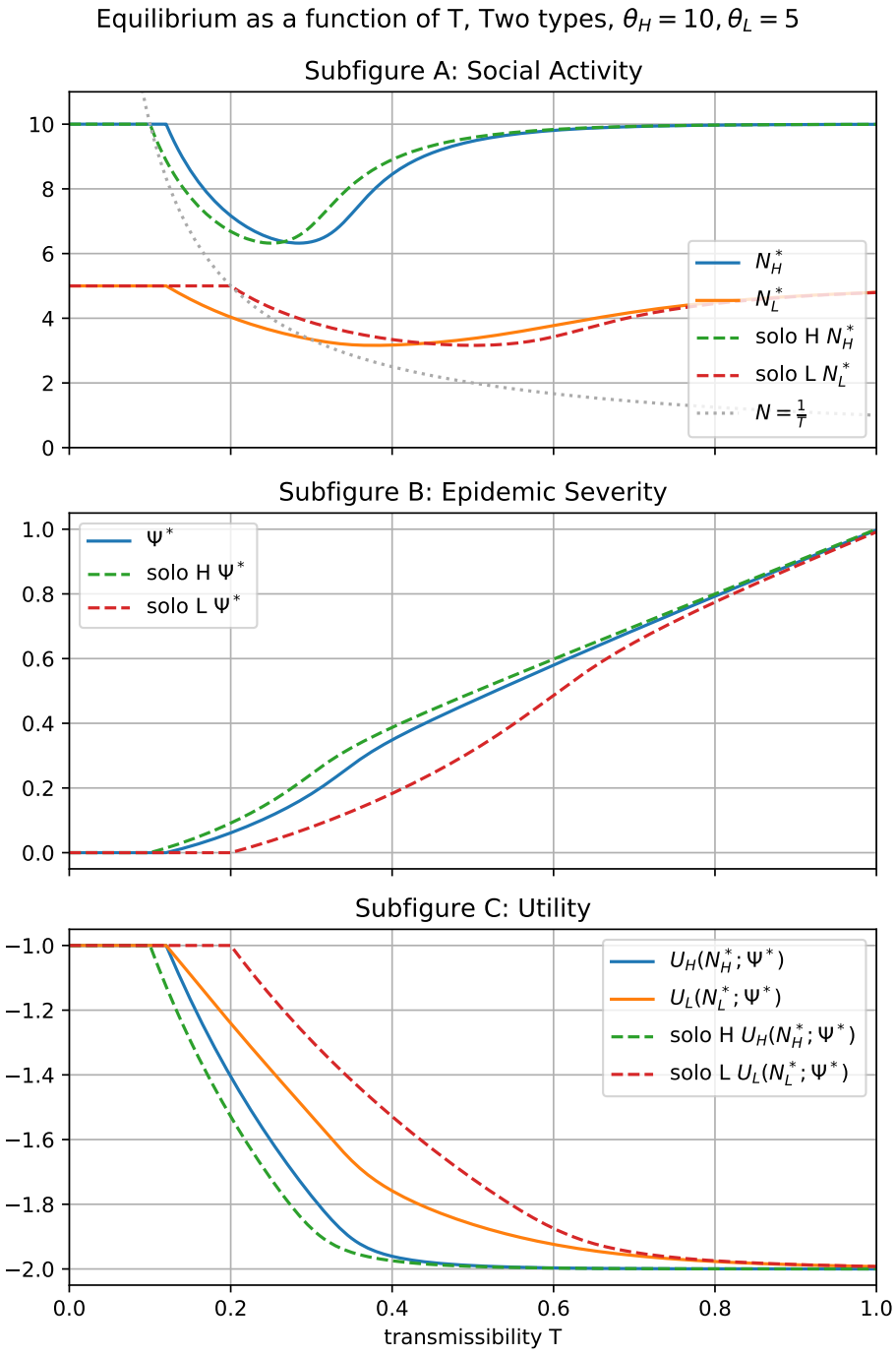

Figure 4: An example of equilibrium with two types, at various levels of T. The solid lines show the equilibrium outcome when each type comprises half of the population, while the dotted lines show what equilibrium would look like if there were only one type of person.

In the current example, the High-activity type always has above average levels of social activity in equilibrium. As such there is no level of transmissibility T in which the resulting equilibrium values of N_H and Ψ can satisfy the condition described in Proposition 3. In other words, making the High-activity type *even more* socially active would be expected to make the epidemic edge risk more severe.

And for this particular example, it also happens to be the case that there are no T for which the equilibrium activity level of the Low-activity type $\$N_L$ satisfies the condition described in Proposition 3 either.

On the other hand, if the utility parameter for the High-activity type were changed to $\theta_H = 20$, then would be ranges of T for which the equilibrium value of N_L would be suboptimally low. For these values of T, which have been highlighted in gray in Figure 5, the social activity of the Low-activity types has positive externalities at the margins.

5 Discussion

This paper has shown how a behavioral choice model can be combined with a branching process model of disease spread to construct an equilibrium model of an acute disease outbreak. When behavioral responses are sufficiently smooth, this model can be solved to find equilibrium outcomes.

Compared to the exogenously imposed levels of social activity which would maximize societal utility, the equilibrium levels of social activity are inefficiently low. This is because the negative externalities of social activity caused by disease spread are not fully internalized. There are situations in which very low-activity people have *inefficiently* low levels of equilibrium social activity, but the existence of such outcomes is circumstantial.

The equations for the spread of disease described in Section 2 capture the features of a static social graph, and my model supposes that each individual chooses an average level of social activity to engage in throughout the entire duration of the epidemic. However, in real life, people can change their behavior day to day in response to hearing news about the changing prevalence of the disease or the number of people dying. Models which take this into account, like (Farboodi, Jarosch, and Shimer 2021), predict that people engage in more activity when the risk from social activity is low, and less activity when the risk is high. This leads to the social risk stabilizing to some specific value, but through dynamic differences in behavior across time rather than static differences in behavior between different types of people. To the extent that frequent behavioral changes are significant, it may be that this percolation-based perspective is more useful in understanding the initial stages of public response, and more useful in understanding the outbreak of very rapidly spreading contagions than it is in thinking about persistent contagions.

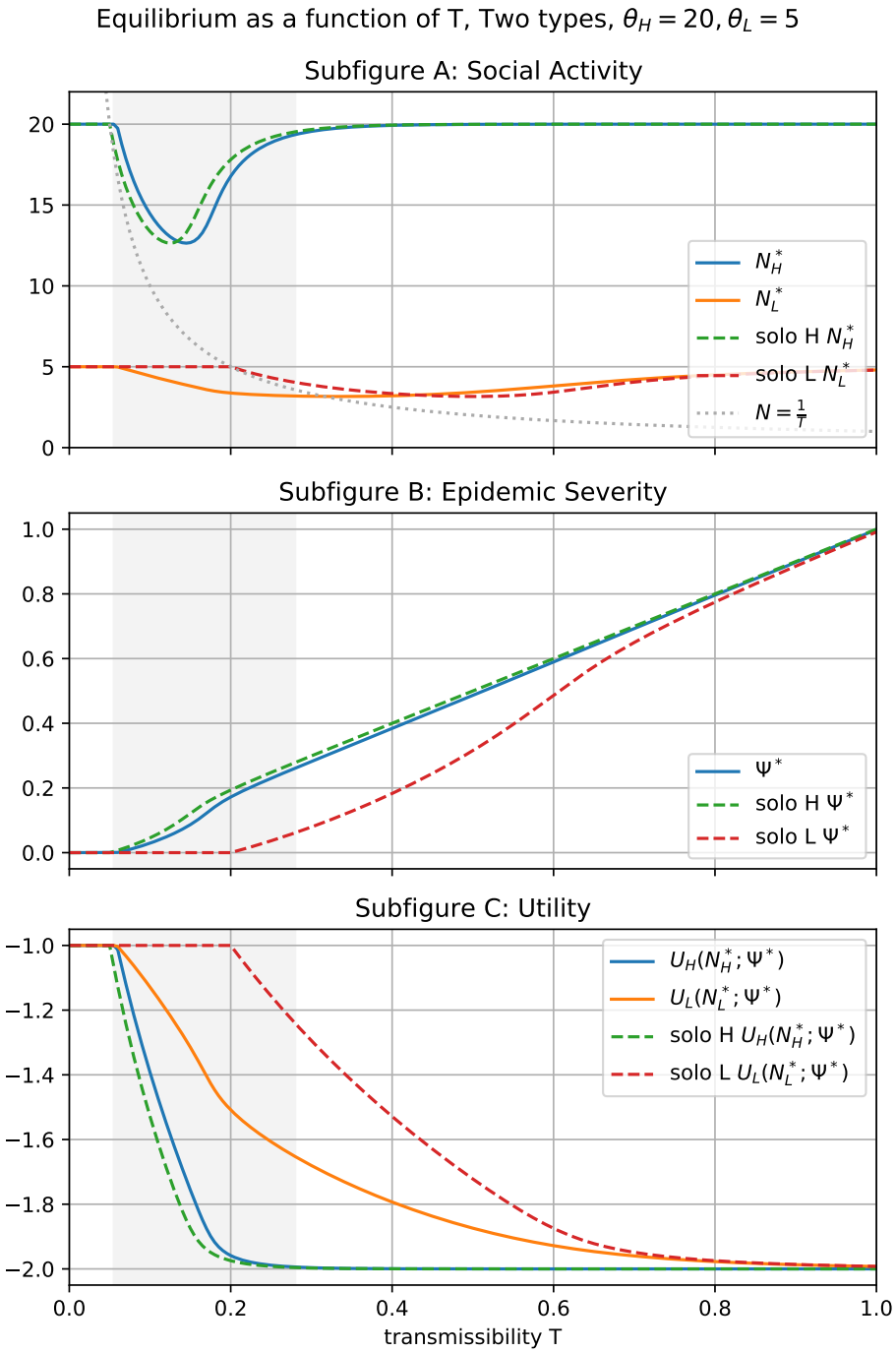

Figure 5: Another example of equilibrium with two types, at various levels of T. This Figure is similar to 4, except that $\theta_H = 20$. The region with a gray-shaded background indicates where the equilibrium value of N_L is inefficiently low.

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6 Appendix

6.1 Further Ideas for Extending this Research

6.1.1 Preferential Matching

In the model as presented in this paper, each person doesn't know the types of their neighbors. Social connections are random, and the probability that a given neighbor is type j is simply a function of the population and social activity level of type j.

However, it seems more intuitively correct to suppose that social connections are formed assortatively, meaning that people are more likely to have connections with others who are similar to them, including in terms of how much social activity they have.

The process of disease spread could be modified to allow for connections to be more common between people of the same type. Then the model could also be used to look at questions like whether it's better to encourage High-activity people to intermingle with Low-activity people or better to split the two groups, letting the disease spread through the High-activity people while the Low-activity people stay home.

To add this feature to the model, Psi would need to vary by type.

In the current model, the edge risk Psi is the risk of a given neighbor getting sick and transmitting the disease. The same Psi can be plugged into the utility function of different types of people because types vary only by the quantity of their neighbors and not by any quality of their neighbors. But if this were to be changed, then Ψ would need to be indexed by type as well. If type j are less likely to get sick than other types and type j has disproportionately many type j neighbors, then that means a type j person is at less risk from each of their neighbors than those other types. Compare that to the current version of the model, in which a Low-activity type is exposed to the same chance of getting sick from each neighbor and only has less chance of getting sick overall because they have fewer neighbors.

6.1.2 A Continuous Distribution Over Types

In estimating the pattern of COVID-19 spread, (Endo et al. 2020) fits data on the number of secondary infections caused by each case of COVID-19 to a negative-binomial distribution. Likewise, (Lloyd-Smith et al. 2005) finds that a negative-binomial distribution is a good fit for the spread of SARS-CoV-1.

A negative-binomial distribution can be described as a compound distribution wherein the mean of a poisson distribution is itself distributed according to a gamma distribution. If the distinct types in my model where written to instead be a continuum of types, with the parameter N continuously distributed according to some gamma distribution, the the distribution for the number of

neighbors would be negative-binomially distributed, as would the distribution of the number of offspring infections.

Such a change would mean that the model's parameters could be better calibrated to real-world epidemiological data.

6.1.3 Comparisons to SIR Model

It could be illustrative to set up a simple compartmental SIR model of disease spread, and compare the predictions made about the severity and extent of an epidemic to the predictions for a similarly parameterized disease in my model.

6.2 More Details about Branching Process model of disease spread

6.2.1 The Structure of the Social Network

Sections 2.1 and 2.2 describe the environment through which disease spreads in this model.

Section 2.1 describes how the distribution of degrees p_k across vertices in the social network is determined from the relative population sizes $\{\alpha_i\}$ and social activity levels $\{N_i\}$ of the different types i. The results from (Newman 2002) in 2.2 then describe the properties of a social network as a function of this degree distribution p_k.

In (Newman 2002), these properties are described as the average properties of an ensemble of graphs generated according to the following process: To generate a graph with V vertices, start by generating a degree sequence $\{k_1, k_2, k_3, ..., k_V\}$ by making V iid draws from the distribution p_k. Next, consider the set of all simple graphs of size V with this degree sequence, and select one of them uniformly at random. The results from section 2.2 can be thought of as being averaged over this graph generation process, with the results becoming exact as V becomes arbitrarily large.

6.2.2 Size of a Finite Outbreak

Secondly, when $T \leq T_c$, the mean number of people infected by a spontaneous outbreak is

$$\mu_s \equiv 1 + \frac{T \sum_k [p_k k]}{1 - T \frac{\sum_k p_k (k-1) k}{\sum_k p_k k}} = 1 + \frac{T \sum_k p_k k}{1 - \frac{T}{T_c}} \tag{27}$$

The 1 on the right-hand-side is the patient zero who spontaneously gets sick. $T \sum_k p_k k$ is the expected number of people whom patient zero infects. And $\frac{1}{1-T/T_c} = \frac{1}{1-R_0}$ is the expected number of infections resulting from each of those transmission events.

With the structure of different Poisson types, this becomes

$$\mu_s \equiv 1 + \frac{T \sum_k \sum_i [\alpha_i \frac{N_i^k e^{-N_i}}{k!} k]}{1 - \frac{T}{T_c(\{N_i\})}} = 1 + \frac{T \sum_i \alpha_i N_i}{1 - T \frac{\sum_i \alpha_i N_i^2}{\sum_i \alpha_i N_i}} \tag{28}$$

6.3 Partial Derivative of Ψ with respect to N_j.

When $T > T_c$, and epidemic is possible,

$$\Psi(\{N_i\}) = T - T \frac{\sum_i \alpha_i N_i e^{-\Psi(\{N_i\})N_i}}{\sum_i \alpha_i N_i}$$

Holding the social activity levels $\{N_i\}$ fixed, and taking the partial derivative with respect to some particular N_j,

$$\frac{\partial \Psi(\{N_i\})}{\partial N_j} = -T \frac{\partial}{\partial N_j} \left[\frac{\sum_i \alpha_i N_i e^{-\Psi(\{N_i\})N_i}}{\sum_i \alpha_i N_i} \right]$$

Take the partial derivatives of the numerator and denominator on the RHS:

$$\frac{\partial}{\partial N_j} \left[\sum_i \alpha_i N_i e^{-\Psi(\{N_i\})N_i} \right] = \sum_i \left[-\alpha_i N_i^2 e^{-\Psi(\{N_i\})N_i} \cdot \frac{\partial \Psi(\{N_i\})}{\partial N_j} \right] + \alpha_j e^{-\Psi(\{N_i\})N_j} \cdot [1 - \Psi(\{N_i\}) N_j]$$

$$\frac{\partial}{\partial N_j} \left[\sum_i \alpha_i N_i \right] = \alpha_j$$

Apply the quotient rule and rearrange:

$$\frac{\partial \Psi(\{N_i\})}{\partial N_j} = \frac{-T}{\left[\sum_i \alpha_i N_i\right]^2} \cdot \left(\left(\sum_i \left[-\alpha_i N_i^2 e^{-\Psi(\{N_i\})N_i} \cdot \frac{\partial \Psi(\{N_i\})}{\partial N_j} \right] + \alpha_j e^{-\Psi(\{N_i\})N_j} \cdot [1 - \Psi(\{N_i\}) N_j] \right) \cdot \sum_i \alpha_i N_i - \left[\sum_i \alpha_i N_i e^{-\Psi(\{N_i\})N_i} \right] \cdot \alpha_j \right)$$

$$\frac{\partial \Psi(\{N_i\})}{\partial N_j} \cdot \left[1 - T \frac{\sum_i \left[\alpha_i N_i^2 e^{-\Psi(\{N_i\})N_i} \right]}{\left[\sum_i \alpha_i N_i\right]} \right] = -T \alpha_j \frac{\left[e^{-\Psi(\{N_i\})N_j} \cdot [1 - \Psi(\{N_i\}) N_j] \right] \left[\sum_i \alpha_i N_i\right] - \left[\sum_i \alpha_i N_i e^{-\Psi(\{N_i\})N_i} \right]}{\left[\sum_i \alpha_i N_i\right]^2}$$

$$\frac{\partial \Psi\left(\{N_i\}\right)}{\partial N_j} = \frac{\left[T\alpha_j \frac{\left[\frac{\sum_i \alpha_i N_i e^{-\Psi(\{N_i\})N_i}}{\sum_i \alpha_i N_i}\right] - e^{-\Psi(\{N_i\})N_j \cdot [1-\Psi(\{N_i\})N_j]}}{\left[\sum_i \alpha_i N_i\right]}\right]}{\left[1 - T\frac{\sum_i \left[\alpha_i N_i^2 e^{-\Psi(\{N_i\})N_i}\right]}{\left[\sum_i \alpha_i N_i\right]}\right]}$$

And note that when $T > T_c$, $\Psi\left(\{N_i\}\right) = T - T\frac{\sum_i \alpha_i N_i e^{-\Psi(\{N_i\})N_i}}{\sum_i \alpha_i N_i}$, which can be rearranged to $\frac{\sum_i \alpha_i N_i e^{-\Psi(\{N_i\})N_i}}{\sum_i \alpha_i N_i} = 1 - \frac{\Psi(\{N_i\})}{T}$. Make this substitution to get:

$$\frac{\partial \Psi\left(\{N_i\}\right)}{\partial N_j} = \frac{\left[T\alpha_j \frac{\left[1 - \frac{\Psi(\{N_i\})}{T}\right] - e^{-\Psi(\{N_i\})N_j \cdot [1-\Psi(\{N_i\})N_j]}}{\left[\sum_i \alpha_i N_i\right]}\right]}{\left[1 - T\frac{\sum_i \left[\alpha_i N_i^2 e^{-\Psi(\{N_i\})N_i}\right]}{\left[\sum_i \alpha_i N_i\right]}\right]}$$

Lemma 4. *Whenever $T > T_c$, the denominator of the above partial derivative is positive.*

Proof. Consider the equation

$$0 = x - \frac{\sum_i \alpha_i N_i e^{(x-1)TN_i}}{\sum_i \alpha_i N_i}$$

When $T > T_c$, the only solutions to this equation are $x = 1$ and $x = \upsilon \in (0, 1)$. Because of the strict concavity of the second term, $x - \frac{\sum_i \alpha_i N_i e^{(x-1)TN_i}}{\sum_i \alpha_i N_i} > 0$ iff $x \in (\upsilon, 1)$.

Take the partial derivative of the RHS:

$$\frac{\partial}{\partial x}\left[x - \frac{\sum_i \alpha_i N_i e^{(x-1)TN_i}}{\sum_i \alpha_i N_i}\right] = 1 - T\frac{\sum_i \alpha_i N_i^2 e^{-(1-x)TN_i}}{\sum_i \alpha_i N_i}$$

Set this equal to zero, and solve for x to find the unique value x^* which maximizes $x - \frac{\sum_i \alpha_i N_i e^{(x-1)TN_i}}{\sum_i \alpha_i N_i}$. It must be that $x^* \in (\upsilon, 1)$.

Because $\upsilon < X^*$, it must also be true that $e^{-(1-\upsilon)TN_i} < e^{-(1-x^*)TN_i}$ for any $N_i > 0$ and $T > 0$. And so

$$1 - T\frac{\sum_i \alpha_i N_i^2 e^{-(1-\upsilon)TN_i}}{\sum_i \alpha_i N_i} > 1 - T\frac{\sum_i \alpha_i N_i^2 e^{-(1-x^*)TN_i}}{\sum_i \alpha_i N_i} = 0$$

Substitute $(1-\upsilon)T$ for Ψ in the above expression to get

$$1 - T\frac{\sum_i \alpha_i N_i^2 e^{-\Psi N_i}}{\sum_i \alpha_i N_i} > 0$$

□

Proposition 5. *Assume $\{N_i\}$ is such that $T > T_c$. When this is the case,*

$$\frac{\partial \Psi(\{N_i\})}{\partial N_j} < 0$$
$$\Updownarrow$$
$$(1 - e^{-\Psi(\{N_i\})N_j}) < \frac{\Psi(\{N_i\})}{T}(1 - TN_j e^{-\Psi(\{N_i\})N_j})$$

Proof.

$$\frac{\partial \Psi(\{N_i\})}{\partial N_j} = \frac{\left[T\alpha_j \frac{\left[1-\frac{\Psi(\{N_i\})}{T}\right] - e^{-\Psi(\{N_i\})N_j} \cdot [1-\Psi(\{N_i\})N_j]}{\left[\sum_i \alpha_i N_i\right]}\right]}{\left[1 - T\frac{\sum_i \left[\alpha_i N_i^2 e^{-\Psi(\{N_i\})N_i}\right]}{\left[\sum_i \alpha_i N_i\right]}\right]}$$

The denominator is positive whenever $T > T_c$, as per above.

And the term $\frac{T\alpha_j}{\sum_i \alpha_i N_i}$ is positive because $T > T_c > 0$, $N_j > \frac{1}{T} > 0$, and $\alpha_j > 0$ by assumption.

So whenever $T > T_c$,

$$\frac{\left(\frac{T\alpha_j}{\left[\sum_i \alpha_i N_i\right]}\right)}{\left(1 - T\frac{\sum_i \left[\alpha_i N_i^2 e^{-\Psi(\{N_i\})N_i}\right]}{\left[\sum_i \alpha_i N_i\right]}\right)} > 0$$

$$\frac{\partial \Psi(\{N_i\})}{\partial N_j} = \frac{\left(\frac{T\alpha_j}{\left[\sum_i \alpha_i N_i\right]}\right)}{\left(1 - T\frac{\sum_i \left[\alpha_i N_i^2 e^{-\Psi(\{N_i\})N_i}\right]}{\left[\sum_i \alpha_i N_i\right]}\right)} \cdot \left(1 - \frac{\Psi(\{N_i\})}{T} - e^{-\Psi(\{N_i\})N_j} \cdot [1 - \Psi(\{N_i\})N_j]\right)$$

And so whenever $T > T_c$,

$$\frac{\partial \Psi(\{N_i\})}{\partial N_j} < 0$$

$$\Updownarrow$$

$$\left(1 - \frac{\Psi(\{N_i\})}{T} - e^{-\Psi(\{N_i\})N_j} \cdot \left[1 - \Psi\left(\{N_i\}\right) N_j\right]\right) < 0$$

This last inequality can be rearranged to get the equivalent inequality that:

$$(1 - e^{-\Psi(\{N_i\})N_j}) < \frac{\Psi\left(\{N_i\}\right)}{T}(1 - TN_j e^{-\Psi(\{N_i\})N_j})$$

□

This is a condition which describes when an *increase* in activity by type i can counterintuitively lead to a *decrease* in edge risk.