

Diffusion on a Sorted Network

Robert Shimer*

University of Chicago

Liangjie Wu[†]

EIEF

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Abstract

We study the diffusion of a disease or of information in an endogenous network that consists of heterogeneous individuals. Individuals differ by the cost (value) of infection (information). Moreover, individuals realize an idiosyncratic match value before deciding whether to match. We characterize the equilibrium using a system of linear differential equations with time-varying coefficients. We show strategic complementarity in matching: when others are more likely to accept a match, the marginal utility from matching with others for each individual also increases. This strategic complementarity endogenously generates sorting by infection probability, return, and contact rates. This sorting endogenously slows down the transmission of an infectious disease and accelerates the diffusion of information. Decentralized equilibrium is inefficient due to the diffusion externality. Whether the externality is positive or negative depends on with whom individuals interact with.

Keywords: economic epidemiology, matching and sorting, random network, COVID-19, diffusion of information

*shimer@uchicago.edu

[†]liangjie.wu@eief.it

1 Introduction

The diffusion of disease or information transpires through interactions among individuals, with different returns and different frequencies of interaction with others. We study their optimal decisions regarding when and with whom to interact with, and the equilibrium impacts of their matching decisions.

We focus on the impact of sorting, whether people with similar characteristics tend to interact disproportionately among themselves, on the diffusion process. With a constant interaction pattern among types, a more sorted interaction pattern leads to higher peak reproduction number and lower long-run accumulated infection. Quantitatively, the effect of sorting is stronger when types differ more in how often they interact with others.

Our main contribution is a tractable equilibrium matching model with heterogeneous agents. We analytically show that positive sorting among individuals with similar returns due to infection is an equilibrium outcome, even with random meeting technology. Our second contribution is to show that in presence of heterogeneity and endogenous decisions of interaction, the diffusion externality can be either positive or negative. Increasing activity of low-cost types during a pandemic can be welfare enhancing, a prediction relies crucially on the existence of sorting.

In the model, forward-looking individuals differ by their returns due to infection. Individuals meet each other through a random meeting process, wherein the total number of meetings is the product of the innate contact rates of any two type of agents. Upon meeting one another, agents draw idiosyncratic preference shocks regarding matching with each other, and decide whether to form a match.

An infectious disease or information starts its diffusion at the beginning of the economy. Whenever an uninfected person meets an infected person, the infection is reproduced and diffused with some probability. The actual infection status is unknown to agents, but the agents form beliefs about their own and others' probability of infection. We study a perfect foresight equilibrium wherein all individuals behave optimally knowing the risk of infection forward.

The matching decision trades off the value from social interactions and the expected return of changing infection status. A costly disease prompts agents to forgo favorable matches with others, to avoid catching the virus; the presence of valuable information makes agents tolerate unfavorable matches with others, to increase their chances of becoming informed. We show that this dynamic decision can be reformulated as a problem of investment with heterogeneous adjustment costs. The optimal decision can be characterized by a single variable that summarizes how selective agents act in their meetings, which we define as caution.

Optimal caution is simply the discounted probability of staying susceptible looking forward multiplied with the discounted return from infection.

The equilibrium can be characterized by two sets of differential equations: (1) equations that describe the infection dynamics with endogenous diffusion rates (as is standard in the SIR models); (2) one equation that describes the caution dynamics. All equations are linear conditional on a path of diffusion rates, allowing for analytical solutions for a subset of endogenous variables.

Matching decisions are strategic complements. When others are more likely to accept matches, it become more valuable for individuals to increase their probability of accepting matches. This strategic complementarity leads to several properties of the decentralized equilibrium.

We provide analytical results regarding sorting in a SI model (one without recovery from infection). First, the equilibrium is rank-preserving in caution and infection probability. At any instant of time, agents with a higher cost from infection are always more cautious when matching with others and have a higher probability of staying susceptible. Second, the equilibrium matching pattern is endogenously sorted, although the meeting technology is random. We show that the sign of sorting depends on the sign of returns due to infection. In the disease case, equilibrium is positively sorted on characteristics. Agents who suffer more from disease are more likely to match with others who suffer more; Agents who meet others more frequently are more likely to match with others who meet more frequently; More importantly, agents who have a higher probability of infection are more likely to match with others who have higher probabilities of infection.

The assortative matching in equilibrium affects the speed of diffusion. We derive a decomposition of the effective reproduction number into: (1) baseline reproduction number, (2) changing stock of susceptible; (3) reduction in activity, and (4) endogenous sorting. When the matching network is positively sorted, the diffusion of infection is slowed down; When the matching network is negatively sorted, the diffusion of infection is accelerated. We illustrate this insight in the context of COVID-19. In the calibrated version of the model, we showed the positive sorting on infection probability can reduce the reproduction number of virus by 27% percent at its peak. This effect is sizable compared to the effect of reduction in activities (50%). Given two observed patterns regarding COVID-19: (1) the sizable activity reduction even without policy intervention and (2) the strong heterogeneity of death risks across different demographic groups, we argue that the sorting mechanism highlighted in this paper should also play an important role in predicting the transmission of the virus.

To understand the efficiency property of the decentralized equilibrium, we consider a social planner's problem. The planner instructs how individuals of heterogeneous types

should match with each other, subject to: (1) the lack of communication of preference shocks during the meeting stage; (2) the diffusion dynamics. The planner’s solution differs from the allocation from decentralized equilibrium in two aspects. First, the planner internalizes the matching externality due to an increasing return to scale in matching decisions, which is missing in individuals’ choices. This inefficiency is highlighted in [Diamond \(1982\)](#). Second, the planner internalizes the fact that the infected agents can further diffuse the infection to others, which is specific to the setting with a non-rival production of infection.

We highlight that the diffusion externality in the decentralized equilibrium can go either direction. In the infectious disease case, individuals might match too little or too much compared to the efficient allocation. From a socially efficient perspective, it might be beneficial to have types with lower loss due to the disease to interact more during the early stage of a pandemic. This increase in interactions lead to a higher and quicker peak of infection for low-cost types, which then protect the types with higher loss from catching the disease later. This type of allocation can never be implemented in a decentralized equilibrium, because the private incentive always leads to a reduction in activity. In our calibration to the COVID-19 pandemic, we indeed find the socially efficient allocation would increase young age groups’ interaction within themselves, while simultaneously reduces the interaction between young cohorts and old cohorts. This prediction relies crucially on the ability of reducing the interaction between old groups and young groups.

The paper is organized in the following order: [Section 2](#) sets up the diffusion environment with exogenous matching pattern among heterogeneous individuals, and decompose the reproduction number into various forces; [Section 3](#) describes and characterizes the equilibrium model that endogenize the matching pattern. [Section 4](#) provides the analytical results regarding equilibrium sorting; [Section 5](#) characterizes the social planner’s problem; [Section 6](#) summarizes the quantitative predictions of the model regarding the COVID-19 pandemic. We conclude in [section 7](#).

Related Literature. This paper is related to the literature that study epidemiology models with economic agents and the literature on information transmission in decentralized markets.

A large literature integrates economic theory and epidemiology. [Dow and Philipson \(1996\)](#) document the assortative matching in terms of HIV infection status, and argue that it is a force to slow down transmission. [Greenwood et al. \(2017\)](#) and [Greenwood et al. \(2019\)](#) study an equilibrium model of heterogeneous agents and apply it to the HIV pandemic in Malawi. In both papers, a force of endogenous sorting operates among agents with different preferences for risks, and it is a driver of aggregate infection dynamics. Inspired by the recent crisis of COVID-19, much on-going research has tried to integrate economic models with the

epidemiology models. For a summary, see the recent literature review [Brodeur et al. \(2020\)](#). Among them, [Acemoglu et al. \(2020\)](#) considers the optimal lock-down policy with exogenous contact matrices; [Brotherhood et al. \(2020\)](#) considers activity choices by different age groups with a random linear matching function; [Alfaro et al. \(2020\)](#) allows heterogeneous agents to make activity choices and have differential impacts on each other by integrating an exogenous homophily matrix. [Faia et al. \(2020\)](#) empirically documents the homophily in information acquisition of individuals during the pandemic and interpret this observation through a lens of a biased information acquisition model. We differ by considering a two-sided matching model where the matching network is endogenous.

Recent work by [Farboodi et al. \(2020\)](#) considers a similar environment to ours with homogeneous agents. [Lebeau \(2020\)](#) studies the interaction among ex ante homogeneous agents who make participation choice, instead of the probability of interaction in our paper. We view our paper as complementary to these works, because many equilibrium interactions featured by these papers are also present in ours. We contribute by addressing heterogeneity in similar strategic environment.

Our paper is also related to the study of information diffusion in decentralized markets. [Duffie and Manso \(2007\)](#) and [Duffie et al. \(2009\)](#) consider the diffusion of information through decentralized meetings. In these models, agents can make decision on the intensity of meetings, which generates faster speed of information acquisition. [Duffie et al. \(2014\)](#) is the most related to our paper. They consider an environment where agents have different contact rates and information quality. Through bilaterally trading with each other via a double auction, they fully incorporate each others' private information. Our paper differs in its focus on the extensive margin of information transmission. We allow agents to turn away meetings based on their perception of the trading partners and their own states. We believe our, which complements existing works, is more applicable to the study of disease diffusion or diffusion of viral information when the price mechanism is less feasible.

Lastly, this paper contributes to the literature on endogenous network formation. Assortative matching on numbers of contacts have received much attention from computer sciences, physics, and sociology ([Newman, 2002](#)). Several economic studies have attempted to understand this phenomena from an equilibrium perspective. [Currarini et al. \(2009\)](#) and [Cabrales et al. \(2011\)](#) consider sorting on social networks when there is complementarity in preferences. [Jackson and Rogers \(2007\)](#) show that sorting on contacts can occur due to differential time of entering. [Golub and Jackson \(2012\)](#) study the implications on social learning given an exogenous and sorted network. We provide an alternative theory of the sorting of an information network: heterogeneity in the reward or frequency of contacts generate sorting due to the interaction between caution and likelihood of infection.

2 Diffusion on an Exogenous Random Network

We start with a model of diffusion on an exogenous random network. This simple model prepares the notations for the diffusion block of the equilibrium model, and also provides insights regarding sorting and diffusion. Matching is more sorted if agents are more likely to interact with others with their own types. With a constant matching matrix that is more sorted, the peak reproduction number is higher and the accumulated infection is lower. Quantitatively, this effect is stronger in a network with large dispersion in how frequently individuals interact. In a general setting with time-varying matching pattern, we provide a decomposition of reproduction of infection into (a) basic reproduction number, (b) size of susceptible population, (c) reduction of activity, and (d) sorting.

2.1 Diffusion with Constant Matching Matrix

Environment.- Time is continuous. There is a unit measure of individuals, differ by their types $j \in [0, 1]$. The number of matches per instant of time between type j and type k is given by a contact function $m(j, k)$. Denote the total number of matches for type j as $\lambda_j = \int_k m(j, k)dk$. We normalize the mean number of matches across types to be 1. Different configuration of $m(j, k)$ allows the model to span between random matching and perfect sorting, and the in-between cases. For example, $m(j, k) = \lambda_j \lambda_k$ would lead to random matching, as all types have the same conditional distribution of interacting with others; $m(j, k) = \lambda_j \delta\{j = k\}$ ¹ would lead to perfect sorting, as all types only interact within themselves.

At $t = 0$, ϵ_j fraction of type j become infected. Diffusion happens through interactions. When an infected person meets an uninfected person, the uninfected becomes infected with probability β_0 . Meanwhile, an infected individual recovers with rate γ each instant of time. The following equation system in terms of infected probability $i(j, t)$ and susceptible probability $s(j, t)$ characterizes the diffusion process:

$$\begin{aligned}\dot{s}_j(t) &= -\beta_0 \int_{\mathcal{X}'} m(j, k) i_k(t) dk, \\ \dot{i}_j(t) &= \beta_0 \int_{\mathcal{X}'} m(j, k) i_k(t) dk - \gamma i_j(t).\end{aligned}$$

In order to characterize the equilibrium, we define a new variable accumulated hazard $z_j(t) = \beta \int_0^t \int_0^1 m(j, k) i_k(t) dk d\tau$. With this hazard rate, we can solve the susceptible and

¹Where δ is the Dirac Delta function.

infection probabilities in closed form:

$$s(j, t) = (1 - \epsilon_j)e^{-z_j(t)}.$$

In the appendix, we show the accumulative hazard can be characterized by the following non-linear integro-differential equation:

Lemma 1 *The accumulative hazard that is consistent with diffusion dynamics must solve:*

$$\dot{z}_j(t) = \beta\lambda - \beta \int_{\mathcal{X}'} m(j, k)(1 - \epsilon_j)e^{-z_k(t)} dk - \gamma z_j(t),$$

with boundary condition

$$z_j(0) = 0.$$

Figure 1 illustrates that sorting among heterogeneous agents can reduce the accumulated infection in the long run. When matching is perfectly sorted, individuals with high contact rates will only become infected from and infect others with equally high contact rates.

Parametric Example.- We use a flexible parametric example to illustrate the effect of sorting on diffusion. Suppose λ is distributed according to a log-normal distribution with mean 1 and varying variance. Suppose:

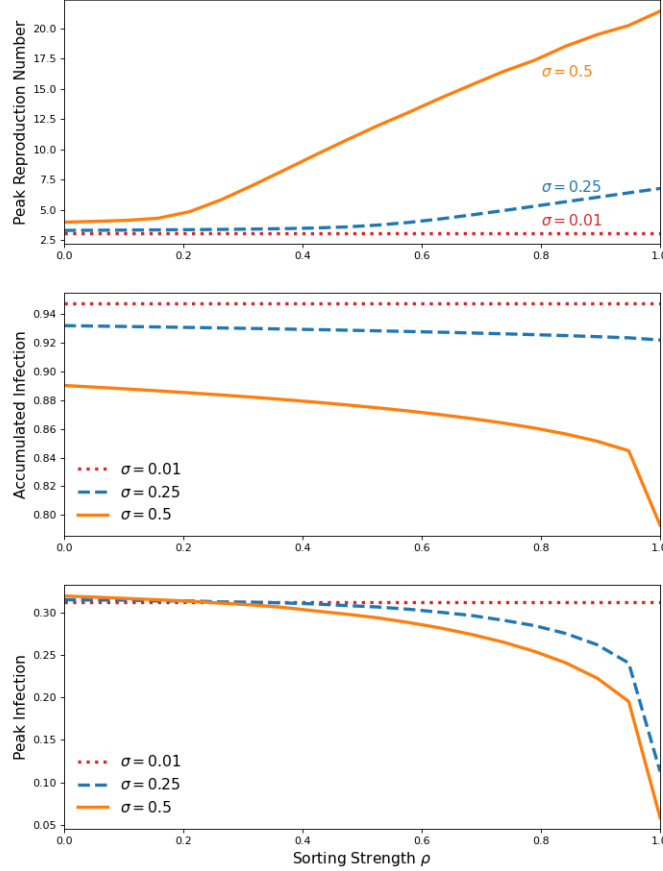
$$m(j, k) = \rho\lambda_j\delta\{\lambda_j = \lambda_k\} + (1 - \rho)\lambda_j\lambda_k.$$

We are interested in two aspects of the diffusion. First, how fast the infection is reproduced, which we measure by the peak reproduction number. Second, what is the total impact of the infection, which we measure by the accumulated population of infection. Figure 1 plots these objects for different variance in contact rates and different degree of sorting.

In the first panel, we plot the peak reproduction number. With more sorted matching (ρ increases), the peak reproduction number increases. This effect is stronger with larger dispersion in contact rates. This is due to two forces. First, the infection is reproduced faster within high-contact types with stronger sorting; Second, larger fraction of infected population is high-contact types, which further increases the reproduction number.

In the second panel, we plot the accumulated infection in the long run. With more sorted matching (ρ increases), the accumulated infection is lowered. This reduction is sizable when the dispersion in contact rates is high. Although the infection is reproduced faster at its peak, it affects less people. First, the high reproduction of infection is confined within a high-contact types. Second, the high-contact types are quickly infected and recover. They later become less likely to pass on the infection to others.

Figure 1: Diffusion Outcomes for Different Level of Dispersion and Sorting



Note: all lines based on $\lambda \sim \text{LogNormal}(-\frac{\sigma^2}{2}, \sigma)$, $\gamma = 1/15$, $\beta = 3.1\gamma$, $\epsilon_\lambda = \lambda \times 10^{-5}$.

In summary, sorting can lead to a reduction in the total impact of infection. The size of this reduction depends crucially on the dispersion of contact rates among individuals. This simple model leaves several questions unanswered. First, we are unable to address the impact of time-varying sorting pattern. Second, we cannot make meaningful statements on efficiency, because we have not specify how individuals differ in their returns due to infection, their decision making, and its link to sorting pattern. We now address the first issue with time-varying sorting in the next subsection, and address the second concern with an equilibrium model in section 3.

2.2 Sorting and Diffusion

In this section, we provide a formal definition of sorting: individuals with similar characteristics are more likely to interact with each other. We then provide a formula to decompose the reproduction of infection into (1) changing size of infected and susceptible population, (2) the composition shift of activity among heterogeneous agents, (3) general change of activity, and lastly (4) sorting among heterogeneous agents.

Environment.- We additionally assume the contact function $m(j, j', t)$ is time varying, which we will provide microfoundation through an equilibrium model. Otherwise, the setting is identical to before.

We are interested in the matching patterns among individuals. More specifically, are agents with similar characteristics tend to match with one another? This characteristic can include risk of infection, the innate contact rate, or loss due to infection. The following definition seeks to describe sorting in the most general way. For any heterogeneity X , we define the conditional distribution of x among a type j person as:

$$M_{X,t}(x|j) = \frac{\int_{x_k \leq x} m(j, k, t) dk}{\int_0^1 m(j, k', t) dk'}$$

Definition 1 (Sorting)

The matching pattern is positively sorted on X at time t if, for $x_j > x_{j'}$ implies:

$$M_{X,t}(y|j) \text{ F.O.S.D. } M_{X,t}(y|j')$$

It is negatively sorted if vice versa.

When there is positive sorting on X , individuals with a higher X tends to match more frequently with others who have a higher X . For example, if the equilibrium is positively sorted on infection probability, individuals who are more likely to be infected are more probable to meet others who are more likely to be infected.

The aggregate basic reproduction number at time t is the individual infection hazard adjusted by the share of each type among susceptible population, as well as the total number of infection in the economy:

$$R(t) = \beta_0 \int_k \int_j m(j, k, t) \frac{i_{j,t}}{I_t} \frac{s_{k,t}}{S_t} dj dk.$$

To interpret this number, we go back to the definition of the basic reproduction number as the expected number of new infections generated by each infected person. Suppose we

take two random person from the infected population and from the susceptible population. Their probability of being type x and x' are their population share within the two pools. They interaction with each other with frequency $m(x, x', t)$. The aggregate number of those meetings adjusted by β_0 would represent the basic reproduction number.

The following lemma provides a decomposition of the aggregate basic reproduction number, given any path of matching matrix $m(x, x', t)$. We define the total number of matches as $M(t) = \int \int m(j, k, t) dj dk$.

Lemma 2 (Decomposition of Reproduction Number)

Given any $m(x, x', t)$, $i(x, t)$, and $s(x, t)$, reproduction number can be written as:

$$R(t) = R_0 H(t) A(t) \alpha_I(t) \alpha_S(t) S(t)$$

where

(Sorting)

$$H(t) = 1 + \frac{\mathbf{COV}_t \left(i(x, t), s(x', t) \right)}{\mathbf{E}_t i(x, t) \times \mathbf{E}_t s(x', t)}$$

with both the covariance and expectation evaluated using distribution of (i_j, s_k) according to the matching pattern at t ;

(Aggregate Activity)

$$A(t) = \frac{M(t)}{M(0)};$$

(Infected Composition)

$$\alpha_I(t) = \frac{\mathbf{E}_t i(x, t)}{I(t)};$$

(Susceptible Composition)

$$\alpha_S(t) = \frac{\mathbf{E}_t s(x', t)}{S(t)}.$$

The focal object of this paper is $H(t)$, a term we define as the homophily index. It measures how sorted individuals are when they match with each other. When the infection probability and susceptible probability are positively correlated, the covariance is positive and $H(t)$ is larger than 1. When the infection probability and susceptible probability are negatively correlated, the covariance is negative and $H(t)$ is less than 1. Thus, sorting in the matching matrix provides an accelerator or brake to diffusion of the infection.

$A(t)$ measures how the aggregate number of meetings change compared to the onset of diffusion. A reduction in meetings will reduce the reproduction number, while an increase in meetings will increase the reproduction number. Lastly, α_I and α_S reflect the compositional

shifts within the infected and the susceptible population. When there are more individuals with high contact rates among infected population, α_I increases; When there are more individuals with low contact rates among susceptible population, α_S decreases.

2.3 Takeaways

Sorting affects the diffusion process. When individuals tend to interact more with others with similar contact rates, the diffusion of infection is faster yet confined to a smaller group of individuals. There are several important issues that we could not address using the exogenous contact matrix model. First, we could not use the model to make prediction about the actual course of diffusion without observing the time-varying pattern of interactions. Second, we could not use the model to address questions such as optimal allocation because the endogenous response of individuals to the diffusion process is not modeled. We thus investigate an equilibrium model where the matching matrix is endogenous.

3 Diffusion on an Equilibrium Network

In this section, we lay out the environment and characterize the equilibrium. To simplify terminology, we will refer to the state of being able to diffuse the disease or information as an infection, and we describe the loss (gain) due to the disease (information) as a return. A higher return means the individual either suffers less loss from disease, or gains a higher benefit from information.

Environment.- Time is continuous and potentially runs forever. It terminates with rate r , when the return due to infection is realized. There is a unit measure of individuals.

Individuals derive utility from interacting with one another. We break down this interaction process into two steps: meeting and matching. Meetings between any two types happen according to a random meeting function. At each instant of time, there is total λ_0 meetings in the economy, and every type meets other types according to their population share. The meeting network is not sorted, in the respect that all individuals have identical conditional distributions of contacting each type.

Upon meeting each other, individuals draw an idiosyncratic preference shock regarding matching with each other. Specifically, they draw ν from distribution $F(\nu)$. We assume $F(\nu)$ is a continuous distribution. Define $f(\nu)$ as its pdf, and $\alpha(\nu) = \frac{f(\nu)}{1-F(\nu)}$ as its hazard function. To simplify notation, we assume it is identical in the analytical descriptions, but allow $F(\nu)$ to be type-specific in the quantitative analysis.

We discuss separately the micro-foundations of the diffusion process in terms of disease

and in terms of information. Both lead to the same optimization representation of individuals.

Disease.- An infectious disease is discovered in period $t = 0$. At $t = 0$, ϵ fraction of individuals of each type is infected. Individuals do not know their own infection status or that of others, but they can form expectations of infection probability. The reproduction of the disease will happen only if one side of a match is infected while the other side is susceptible, with the baseline infection probability β_0 .

If a person of type j is infected, he experiences a terminal return ϕ_j when the economy terminates, which is translated to an expected flow return of $r\phi_j$. We offer several interpretations of this flow return due to infection. First, one can interpret this utility loss as the expected event of developing symptoms for an asymptomatic infected person. Second, one could interpret this loss as the cost of being in an intense care unit or of being forced to quarantine. As a result, it is both a primitive epidemiological parameter and a policy parameter. At any instant of time, infected individuals recover with rate γ .

Information.- There is an hidden aggregate event that will arrive with rate r . Consider this as an adoption of new technology or the initial offering of an asset. Suppose at $t = 0$, each individual observes a private signal.

When two agents meet, an information exchange happens with probability β_0 . We model information exchange as two agents simply merging their set of signals, after which each walks away with the same set of signals.

Signals can be potentially valuable. Specifically, ϵ fraction of the initial signals would yield reward. When the economy terminates, individuals who hold the reward-bearing signal would gain a return according to their types. At any instant of time, γ fraction of signals are lost.

We make a further assumption regarding the preference shock distribution: $0 < F(\frac{r\phi_j}{r+\gamma}) < 1$ for $\forall j$. This assumption ensures that there is always a positive possibility that agents in every type would want to form a match, because they might meet someone with a high match-specific value.

Preference Shock.- We first re-write the matching decision with preference shock as an concave utility maximization problem of choosing a matching probability. Suppose an individual sets threshold z for meetings: she accepts a match if $\nu > z$ and reject a match if $\nu < z$. Given this threshold, the probability of accepting match is $q = 1 - F(z)$. The expected value of preference shocks is $w = \int_z^\infty \nu dF(\nu)$. Using these two equations we define a convenient utility function from matches:

$$w(q) = \int_{F^{-1}(1-q)}^\infty \nu dF(\nu).$$

With this utility function, it is as if the individuals are choosing what fraction of meetings to consume, given a concave utility function $w(q)$ with

$$w'(q) = F^{-1}(1 - q) > 0,$$

$$w''(q) = -\frac{d}{dq}F^{-1}(1 - q) < 0.$$

Dynamic Decision.- We now formally describe type i 's problem. To clarify the notation, we use a capital letter for each of the aggregate variables, and the lower case of the same letter for an individual's choices. An individual of type j takes as given: (1) the probability that a type k individual accepts matches with her at any period, $Q_{k,j}(t)$, and the probability that a type k individual is infected, $I_k(t)$. She then chooses the type-specific matching probability $q_{j,k}(t)$ to maximize the expected utility:

$$\max_{q_{j,k}(t), s_j(t), i_j(t)} \int_0^t e^{-rt} \left(\lambda_0 \int_0^1 Q_{k,j}(t) w(q_{j,k}(t)) dk + r\phi_j i_j(t) \right) dt, \quad (1)$$

s.t.

$$\dot{s}_j(t) = -\beta_0 s_j(t) \lambda_0 \int_k Q_{k,j}(t) q_{j,k}(t) I_k(t) dk$$

$$\dot{i}_j(t) = \beta_0 s_j(t) \lambda_0 \int_k Q_{k,j}(t) q_{j,k}(t) I_k(t) dk - \gamma i_j(t)$$

The object function is the discounted value of the flow utility. The flow utility has two components. First, her meetings with type k would arrive with the exogenous rate $\lambda_j \lambda_k$. If type k accepts a match with probability $Q_{k,j}$ and if type j accepts a match with probability $q_{j,k}$, the expected utility is $Q_{k,j} w(q_{j,k})$. The utility from matches is the sum of utilities from all types. In addition, given her belief of own infection probability $i_j(t)$, the period return due to infection is $r\phi_j i_j(t)$.

How she decides to match with others also affects the evolution of her belief about infection. The inflow of infection probability is the total measure of meetings where she is uninfected (which happens with probability $s_j(t)$) while the others are infected (which happens with probability $I_k(t)$). For any instant of time t , the probability of infection also decreases due to the chance of recovery.

We look for an equilibrium with perfect foresight. Specifically, we require the matching decisions $q_{j,k}(t)$ to be individually optimal, given the aggregate infection probability of others and the strategy of matching with others.

Definition 2 (Perfect Foresight Equilibrium)

A *Perfect Foresight Equilibrium* is a combination of individual variables $\{q_{j,k}(t), s_j(t), i_j(t)\}$

and aggregate variables $\{Q_{j,k}(t), S_j(t), I_j(t)\}$:

1. (Individual Optimality) Given $\{Q_{j,k}(t), I_j(t)\}$, the individual variables $\{q_{j,k}(t), s_j(t), i_j(t)\}$ solve the optimization problem in equation (1);

3. (Perfect Foresight):

$$Q_{j,k}(t) = q_{j,k}(t),$$

$$I_j(t) = i_j(t),$$

$$S_j(t) = s_j(t).$$

3.1 Characterization

To characterize the optimization problem of individuals, we break the problem into two steps: whom to match with and when to match. This characterization allows us to simplify the problem and also offers economic insights about the interaction in equilibrium.

We start by writing out the Hamiltonian of the full problem as in equation (1):

$$\begin{aligned} \mathbf{H} = \max_{q_{j,k}} \lambda_0 \int_0^1 Q_{k,j}(t) w(q_{j,k}(t)) dk + r \phi_j i_j(t) \\ - (\mu_j^s - \mu_j^i) \beta_0 s_j(t) \lambda_0 \int_k Q_{k,j}(t) q_{j,k}(t) I_k(t) dk - \gamma \mu_j^i i_j \end{aligned} \quad (2)$$

In the appendix, we provide the formal derivation of the first order conditions. Here we provide an alternative interpretation of the optimal conditions, to facilitate the understanding of the economics. We note the Hamiltonian in equation (2) can be thought of as a two step maximization problem: (1) conditional on $\beta = \int_k \lambda_j \lambda_k Q_{k,j}(t) q_{j,k}(t) I_k(t) dk$, to choose the optimal $q_{j,k}$ that maximizes the total utility from matches; and (2) choose the optimal $\beta(t)$ that maximizes the Hamiltonian. The first step will be referred to as the *intratemporal problem*, and the latter step will be referred to as the *intertemporal problem*.

Intratemporal Problem.- We start by considering a static problem of type-specific matching decisions, as in equation (2):

$$W(\beta; j, t) = \max \lambda_0 \int_0^1 Q_{k,j}(t) w(q_k) dk, \quad (3)$$

s.t.

$$\beta = \beta_0 \lambda_0 \int_0^1 Q_{k,j}(t) q_k I_k(t) dk.$$

The individual chooses the optimal probability of accepting a meeting with type k , q_k , given others' strategy $Q_{k,t}(t)$ and infection risk $I_k(t)$, as well as an target infection rate β .

Conceptually, we ask: to maintain an infection rate of β , what is the optimal strategy of accepting meetings?

This static problem has a strictly concave objective function and a linear constraint. As a result, the first-order condition uniquely determines the optimal matching decisions:

$$w'(q_k) = F^{-1}(1 - q_k) = \beta_0 \kappa I_k.$$

The optimal matching decision equalizes the marginal utility from matching with type k to the marginal cost of getting infected, where κ is the marginal utility from increasing the infection rate target. This simple optimal decision rule hinges on two assumptions about the matching process. First, there is no transfer of utilities when pairs decide whether to form a match. Second, the disease only reproduces when both parties decide to match with each other. We now invert the $w'(q)$ function to obtain a closed-form solution for matching probabilities:

$$q_k = 1 - F(\beta_0 \kappa I_k).$$

The optimal matching threshold depends on three factors: (1) the baseline infection probability β_0 ; (2) the probability of type k being infected; and (3) the marginal utility of increasing infection target κ . Because both β_0 and i_k are positive, a higher κ means a higher threshold of matches and a lower probability of matches. Hereafter, we refer to κ_j as the caution of each individual: a higher caution means type j is more selective about meeting others.

The static problem in equation (3) greatly simplifies the characterization of equilibrium. Specifically, given any β , i_k , and $Q_{j,k}$, we find κ_j as solution to the following equation, and accordingly we find the optimal matching decision $q_{j,k}$:

$$\beta = \beta_0 \lambda_0 \int_0^1 Q_{k,j}(t) \left(1 - F(\beta_0 \kappa I_k) \right) I_k dk.$$

Before proceeding to the characterization of the fully dynamic problem, we derive two properties of $W(\beta; j, t)$ functions, which would be useful in characterizing equilibrium and its sorting pattern. First, the Lemma 1 shows that the $W(\beta; j, t)$ function is a single-peaked concave function, with Inada conditions on both $\beta \rightarrow 0$ and $\beta \rightarrow \bar{\beta} \equiv \beta_0 \int_0^1 \lambda_j \lambda_k Q_{k,j}(t) i_k dk$. Thus individuals will always choose an interior infection target in $(0, \bar{\beta})$:

Lemma 3 (Concavity of $W(\beta; j, t)$)

Given any equilibrium path, $W(\beta; j, t)$ is concave:

$$\kappa'(\beta; j, t) = W''(\beta; j, t) < 0.$$

Second, Lemma 2 shows that the W function has super-modularity in two pairs of variables: (1) the infection target β and other people's acceptance probability $Q_{k,j}$; and (2) the infection target β and contact rate λ_j .

Lemma 4 (Super-Modularity of $W(\beta; j, t)$)

Everything else equals, suppose $\lambda_j Q_{k,j} > \lambda_{j'} Q_{k,j'}$ for any k , then for any β ,

$$\kappa(\beta; j, t) > \kappa(\beta; j', t).$$

Lemma 2 implies that, when others are more likely to accept matches with type j , the marginal utility of any given β also increases, and she will be more incentivized to set a higher target for the infection rate. A similar statement holds for a person with higher contact rate λ_j . This comparative static result is the key to understanding the sorting pattern of equilibrium. Matches are strategic complements. When there is more inflow of possible meetings, turning others away would be more costly, because it is more likely that the focal individual is avoiding high-value matches. This means that in the equilibrium, individuals with a higher contact rate will set a higher infection target, so as individuals who are more preferable match partners to others.

Intertemporal problem.- In this subsection, we write type j 's dynamic matching decision using the static value from optimal matches $W(\beta; j, t)$:

$$\max_{\beta(t)} \int_0^t e^{-rt} \left(W(\beta(t); j, t) + r\phi_j i(t) \right) dt, \quad (4)$$

s.t.

$$\dot{s}(t) = -\beta(t)s_j(t),$$

$$\dot{i}(t) = \beta(t)s_j(t) - \gamma i(t).$$

Although $W(\beta; j, t)$ is still an endogenous function that depends on equilibrium outcomes, the decision of individuals is now a single variable dynamic programming problem. We characterize the solution by writing at the current-value Hamiltonian. To simplify notation, we omit the subscript for time and type when it is not necessary:

$$\mathbb{H}(s, i) = \max_{\beta} W(\beta) + r\phi i - \mu^s \beta s + \mu^i (\beta s - \gamma i).$$

The optimal decision must satisfy the first-order conditions. Specifically, the marginal utility from increasing the infection rate must equal the marginal cost of increasing the infection

probability:

$$\kappa(\beta) = W'(\beta) = (\mu^s - \mu^i)s. \quad (5)$$

Second, the marginal value of susceptible and infection probability must be consistent with the dynamic accounting equation at the optimum:

$$\dot{\mu}^s = r\mu^s + (\mu^s - \mu^i)\beta(t) \quad (6)$$

$$\dot{\mu}^i = (r + \gamma)\mu^i - r\phi \quad (7)$$

Finally, all the marginal values and state variables need to satisfy the transversality condition:

$$\lim_{t \rightarrow \infty} e^{-rt} \mu^s s = 0, \quad \lim_{t \rightarrow \infty} e^{-rt} \mu^i i = 0$$

Equation (6), together with the transversality condition, immediately imply that for any t , the marginal value of infection probability must stay constant, $\mu^i(t) = \frac{r\phi}{r+\gamma}$. Otherwise, the accounting equation implies that the marginal value will eventually grow faster than r in the limit.² This result has an intuitive interpretation: an asymptomatic infection costs exactly the discounted expected loss, because it does not shift the behaviors of individuals.

Furthermore, using the result in equation (4), we can now investigate how does caution evolves over time:

$$\dot{\kappa} = (\dot{\mu}^s - \dot{\mu}^i)s + \kappa \frac{\dot{s}}{s}$$

Because for any period $\dot{\mu}^i = 0$, the first part of the above equation must be $r\mu^s s + \kappa\beta$. Because the $\dot{s} = -\beta s$, the second part of the above equation must be $-\kappa\beta$. Thus, we reach a simple equation for caution $\dot{\kappa} = r\mu^s s$:

$$\dot{\kappa} = r\kappa + \frac{r^2\phi}{r + \gamma}s.$$

Conditional on any path of being susceptible $s(t)$, there is a unique solution to the equation for caution that satisfies the transversality condition:

$$\kappa(t) = -\frac{r^2\phi}{r + \gamma} \int_t^\infty e^{-r(\tau-t)} s(\tau) d\tau. \quad (8)$$

This closed-form solution further enforces our interpretation of $\kappa(t)$ as caution. Caution in any period t is simply the discounted probability forward from t adjusted by the discounted

²More specifically, we can solve for the closed form for $\lim_{t \rightarrow \infty} e^{-rt} \mu^i(t) i(t) = C(\epsilon + \int_1^\infty e^{\gamma\tau} s(\tau)\beta(\tau)d\tau)$. The only constant that makes sure this limit is zero would be $C = 0$, which implies $\mu^i(t) = \frac{r\phi}{r+\gamma}$.

loss due to infection. So $\kappa(t)$ is a measure of value at risk when an individual engages in risky interactions. Taking the limit $t \rightarrow \infty$ we derived the limiting behavior of $\kappa_j(t)$

$$\lim_{t \rightarrow \infty} \kappa(t) = -\frac{r^2 \phi}{r + \gamma} \lim_{t \rightarrow \infty} s(t).$$

One interesting feature we discovered from the equation (8) is that $|\kappa(t)|$ is an decreasing function in time. We refer to this property as *equilibrium fatigue*. In the case of disease, over time agents behave less cautious in their meetings and meet more often; in the case of information, over time agents are more selective and meet less often. This comes from the fact $s(t)$ is an decreasing function in time. As time progresses, as long as agents are still matching with others, they can only be less likely to stay susceptible. This makes the return from being cautious smaller.

Equilibrium.- Having characterized of individual optimization problem, we are now ready to characterize the full equilibrium. This only requires modifying the individual problem by requiring their conjecture of the action of others with the actual optimal choice of each type. The following lemma summarizes the equation system for equilibrium outcomes.

Lemma 5 (Equilibrium Equation)

The equilibrium is characterized by $\{\kappa_j, s_j, i_j\}$ solving
(Caution)

$$\dot{\kappa}_j = r\kappa_j + \frac{r^2 \phi_j}{r + \gamma} s_j;$$

(S-I-R)

$$\dot{s}_j = -\beta_j s_j;$$

$$\dot{i}_j = \beta_j s_j - \gamma i_j;$$

(Matching)

$$\beta_j = \beta_0 \lambda_0 \int_k \left(1 - F(\beta_0 \kappa_k i_j)\right) \left(1 - F(\beta_0 \kappa_j i_k)\right) i_k dk;$$

(Transversality)

$$\lim_{t \rightarrow \infty} \kappa_j(t) = -\frac{r^2 \phi}{r + \gamma} \lim_{t \rightarrow \infty} s_j(t).$$

The equilibrium is characterized by a three-equation linear o.d.e. system, in which the time-varying coefficients are determined by the equilibrium matching equation. The initial state variable is fixed. Thus, the only endogenous variable at the onset of the equilibrium is how cautious people are, $\kappa_j(0)$. Given any $\{\kappa_j(0)\}$, β_j are fully characterized by the matching equation, and, thus, the time drifts. This simple argument establishes that, given any initial caution $\{\kappa_j(0)\}$, the equilibrium is uniquely determined forward.

Not every initial condition is consistent with the transversality condition. Looking for an equilibrium is to solve a forward-shooting problem. We look for $\kappa_j(0)$ such that in the limit:

$$\lim_{t \rightarrow \infty} \kappa(t) = -\frac{r^2 \phi}{r + \gamma} \lim_{t \rightarrow \infty} s(t).$$

In practice, this shooting problem can be efficiently solved with an iteration algorithm.³

4 Equilibrium Sorting

In this section, we characterize the sorting pattern in the decentralized equilibrium. Analytically, we show that sorting on risks is a universal feature of any equilibrium when individuals differ in their loss due to infection or the innate contact rate in a case where the recovery from infection is shut down (SI model). The sorting pattern among heterogeneous individuals affects aggregate transmission dynamics. When the matching network has a positive sorting on risks, the reproduction of infection is slowed; When the matching network has a negative sorting on risks, the reproduction of infection is accelerated. Moreover, the strength of sorting is time-varying and depends on the aggregate prevalence of the pandemic.

Lemma 6 (Sufficient Condition for Sorting)

If the matching probability has log-supermodularity in (x_j, x_k) , then the equilibrium is positively sorted on x ; if the matching probability has log-submodularity in (x_j, x_k) , then the equilibrium is negatively sorted on x .

Log-supermodularity of probability density in terms of (x_j, x_k) is a sufficient condition for ranking of conditional CDFs in terms of stochastic dominance. Lemma 5 further narrow the discussion to matching probability because the contact rates are all multiplicative to the density. In other words: if the matching probability is a uniform distribution across (x_j, x_k) , then the conditional distribution is identical across all types.

The sufficient condition in Lemma 5 allows us to interpret the sorting pattern in terms of classical matching theory. We can write out the log-matching probability in equilibrium among any type j individual and type k pair:

$$\log q_{j,k} q_{k,j} = \log(1 - F(\beta_0 \kappa_j i_k)) + \log(1 - F(\beta_0 \kappa_k i_j)),$$

whether the equilibrium is positively sorted or negatively sorted depends on (a) the monotonicity of μ_j in x_j , (b) the monotonicity of i_k in x_k , and (c) the log-concavity of the survival

³However, the uniqueness of equilibrium is harder to establish due to the strategic complementarity nature of interaction. This is also featured in recent work by Lebeau (2020).

function $1 - F(\nu)$. Because the matching threshold is $\beta_0 \kappa_j i_k$, an individual who has a higher infection probability is less likely to be accepted for matches. Furthermore, due to the product form, a more cautious individual will amplify this differential. Two complexities remain: (1) how caution and infection probability are ranked among types and (2) how does the matching threshold is mapped into matching probabilities.

4.1 Analytical Result: Sorting in SI Model

In this section, we state our first result for the equilibrium matching pattern: the rank of caution and risk stays unchanged from the onset of the diffusion to the the limit.

Proposition 1 (Rank-Preserving in Infection)

If $\phi_1 > \phi_2$, for any $t > 0$:

$$i_1(t) > i_2(t) \quad \text{and} \quad s_1(t) < s_2(t).$$

Proof. See Appendix. ■

From a static decision perspective, the result of proposition 1 is intuitive. However, given the equilibrium interaction among individuals and infection dynamics, it is not immediately self-validating. More specifically, a type that has a lower infection probability is more preferable by others. Thus, it is more costly for them to turn away from meetings, due to the super-modularity of the utility from matching.

Proposition 1 shows that the the equilibrium effect could not dominate the direct impact of heterogeneity in loss and contact rate. We discuss here the economics behind the proof. Details aside, the main argument is a revealed preference argument. First, suppose there is a period during which the two types start with the same probability of infection and j' ends up with less likely to be infected. If other individuals behave optimally, they must accept matches with type j' with higher probability. This means the marginal utility of increasing infection target is higher for type j' , because of the super-modularity of $W(\beta; j, t)$ function. Thus, type 2 has higher return from infection because $\phi_{j'} < \phi_j$ and higher marginal utility from meeting others. Deviating from type j' 's strategy to type j 's strategy creates a strict improvement. This is a contradiction. What we ruled out so far is any crossing where type j' ends up with lower infection risk. So the whole equilibrium path must leads to that type j has higher infection probability.

Corollary 1 (Rank-Preserving in Caution)

If $0 > \phi_1 > \phi_2$, for any $t > 0$:

$$\kappa_1(t) < \kappa_2(t).$$

Corollary 1 is an immediately result that combines two facts: (1) the equilibrium is rank preserving in the probability of infection; and (2) caution is the discounted probability of staying susceptible adjusted by the expected loss due to infection. Take as an example the case of loss heterogeneity for an example. Individuals with a higher loss will have lower probability of being infected through out the equilibrium path. Because there is no recovery from infection, individuals with higher loss also has higher probability of staying susceptible through out the equilibrium path. This means they must also be more cautious.

Proposition 2 (Disease: Positive Sorting)

The equilibrium is positively sorted on infection or return, under the follow conditions:

- $\phi_j < 0$
- $\alpha'(\nu)\nu + \alpha(\nu) > 0$ for $\nu > 0$ (e.g. exponential, logit, normal, Pareto)

To understand the results in proposition 2, consider the cross partials of log matching probability with respect to x_j and x_k . Because the log-probability is additive, we focus on $q_{j,k}$:

$$\frac{\partial^2 \log q_{j,k}}{\partial x_j \partial x_k} = -\frac{d\kappa_j}{dx_j} \frac{di_k}{dx_k} \left(\alpha'(\nu)\nu + \alpha(\nu) \right) \Big|_{x=\mu_j i_k};$$

The endogenous object that affects the local sorting pattern between type j and type k is $\frac{d\kappa_j}{dx_j} \frac{di_k}{dx_k}$: how does caution and infection probability vary? Consider the case of sorting on infection probability. Here the local sorting pattern is solely determined by $\frac{d\kappa_j}{di_j}$. As shown in Section 3.1, the equilibrium in any SI environment must be rank preserving. For the case of infectious disease, types with higher infection probability at any $t > 0$ must also be less cautious about matching with others. As a result, $\frac{d\kappa_j}{di_j} < 0$. We further restrict attention to distributions with property $\alpha'(\nu)\nu + \alpha(\nu) > 0$. This condition states that the logarithm of the survival function $1 - F(\nu)$ cannot be too concave, which is satisfied by many familiar distributions (e.g. Exponential distribution, Normal distribution, or Logit Distribution). Under this restriction on the preference shock, it is immediately apparent that the equilibrium must be sorted on infection probability.

5 Efficiency

In this section, we consider the problem of a social planner whose goal is to maximize the discounted aggregate utility.

Setup.- The social planner chooses $q_{j,k}(t)$ to maximize the following problem:

$$\max_{q_{j,k}(t)} \int_0^\infty e^{-rt} \left\{ \lambda_0 \int_j \int_k q_{j,k}(t) w(q_{k,j}(t)) dk dj + \int_j r \phi_j i_j(t) dj \right\} dt,$$

s.t.

$$\begin{aligned} \dot{s}_j(t) &= -\beta_0 \lambda_0 \int_k q_{j,k}(t) q_{k,j}(t) i_k(t) dk s_j(t), \\ \dot{i}_j(t) &= \beta_0 \lambda_0 \int_k q_{j,k}(t) q_{k,j}(t) i_k(t) dk s_j(t) - \gamma i_j(t), \\ i_j(0) &= \epsilon. \end{aligned}$$

The social planner instructs individuals of type j to accept meetings with type k with probability $q_{j,k}$. Her objective function is the sum of the individual discounted utility from matches and from infection. We clarify the frictions faced by the planner. First, she faces the uncertainty about actual infection status of each individual, so has to for expectation about the infection status of each type. In fact, the law of motion for the planner's state variables is identical to the ones faced by individuals in the decentralized equilibrium. Second, the planner faces the same type of coordinating friction in each meeting. When two agents meet, there is no communication regarding their private value of matches. We keep this assumption in the planner's problem.

Characterization.- We first set up the Hamiltonian of the planner's problem. Denoting $\hat{\mu}_j^i$ and $\hat{\mu}_j^s(t)$ the shadow value of the infection probability and the shadow value of susceptible for type j for type j . The Hamiltonian of the planner's problem is:

$$\begin{aligned} H(\{\hat{\mu}_j^i, \hat{\mu}_j^s, i_j, s_j\}_j) &= \max_{q_{j,k}} \lambda_0 \int_j \int_k q_{j,k} w(q_{k,j}) dk dj + \int_j r \phi_j i_j dj \\ &\quad - \beta_0 \int_j (\hat{\mu}_j^s - \hat{\mu}_j^i) s_j \lambda_0 \int_k q_{j,k} q_{k,j} i_k dk dj - \gamma \int_j \hat{\mu}_j^i i_j dj \end{aligned} \quad (9)$$

We observe two features of how the matching probabilities enters planner's objective function: (1) they enter symmetrically in the return function. Interchanging their roles does not alter the objective function; (2) they enter as a product in the law of motion of infection probabilities. This does not guarantee symmetry in the optimal allocation. Depending on the parameterization of the preference shock, both symmetric and non-symmetric solutions can be optimal. For instance, if the preference shock follows the Exponential distribution, any solution with $q_{j,k} q_{k,j}$ staying constant yield the same value. If the preference shock follows an Pareto distribution with a thick tail, the optimal solution requires one side to always accept the match and the other side to set a interior threshold.

To characterize the planner's optimal allocation, we first take the first-order condition with respect to $q_{j,k}$:

$$q_{k,j}w'(q_{j,k}) + w(q_{k,j}) = (\hat{\mu}_j^s - \hat{\mu}_j^i)s_j i_k q_{k,j} + (\hat{\mu}_k^s - \hat{\mu}_k^i)s_k i_j q_{k,j}.$$

The optimal allocation equates the marginal values from matches to the marginal value from diffusion. Comparing the planner's condition to the individual's condition in the decentralized equilibrium, we notice the first source of inefficiency in the equilibrium. In the decentralized equilibrium, individuals ignore that when they increase matching probability, they also increase the probability that their meeting partners are getting matched with. This is correctly reflected in the social planner's problem as $w(q_{k,j}) - (\hat{\mu}_k^s - \hat{\mu}_k^i)i_j q_{k,j}$.

We define a planner counterpart of caution $\hat{\kappa}_j = (\hat{\mu}_j^s - \hat{\mu}_j^i)s_j$. With this definition and the proportionality in matching probability, the optimal condition for $q_{j,k}$ and $q_{k,j}$ implies:

$$w'(q_{j,k}) + \frac{w(q_{k,j})}{q_{k,j}} = w'(q_{k,j}) + \frac{w(q_{j,k})}{q_{j,k}} = \hat{\kappa}_j i_k + \hat{\kappa}_k i_j.$$

We then turn to the accounting equation for shadow values at the optimum:

$$\begin{aligned} \dot{\mu}_j^s &= r\mu_j^s + (\mu_j^s - \mu_j^i)\beta_0\lambda_0 \int_k q_{j,k}q_{k,j}i_k dk \\ \dot{\mu}_j^i &= (r + \gamma)\mu_j^i - r\phi_j + \beta_0\lambda_0 \int_k q_{j,k}q_{k,j}(\hat{\mu}_k^s - \hat{\mu}_k^i)s_k dk. \end{aligned}$$

The shadow value of susceptible probability is identical to the decentralized equilibrium. The risk of being infected is a private loss, so is fully internalized by individuals. On the other hand, the shadow value of infection probability has an extra time drift. This reflects the diffusion externality caused on others. When an individual is more likely to get infected, the chances that she passes the virus to others also increases. As a result, the social value of infection is no longer constant. Instead, it is a time-varying object that reflects the changing likelihood of reinfection in the economy. We again utilize the definition of $\hat{\kappa}_j$ to write:

$$\dot{\hat{\kappa}}_j = r\hat{\kappa}_j - \gamma\hat{\mu}_k^i s_j + r\phi_j s_j - \beta_0\lambda_0 s_j \int_k q_{j,k}q_{k,j}\hat{\kappa}_k dk.$$

Compared to the equilibrium caution as in equation (8), the caution according to planner's allocation cannot be trivially signed. As a result, the diffusion externality can take either positive or negative signs. The decentralized equilibrium can match too often or too little depending on who matches with whom. We will discuss this statement in more details

in the quantitative application.

6 Applications: COVID - 19

In this section we illustrate the insights we can learn from the framework. We consider the on-going pandemic of COVID-19. With the equilibrium model, we show sorting within age groups can play a vital role in reducing the reproduction number. By comparing the equilibrium outcome to the planner's solution, we show young groups match too little in the decentralized equilibrium, a novel prediction from the model presented in this paper.

6.1 Calibration

We simulate the model for the case of COVID-19. There are three sets of parameters to calibrate for our model: the preference/matching parameter and the epidemiology parameter.

Demographics.- We set the types to be six age groups: 0-18, 19-49, 50-59, 60-69, 70-79, and above 80. The share of these six types are set to the aggregate population share in the United states.

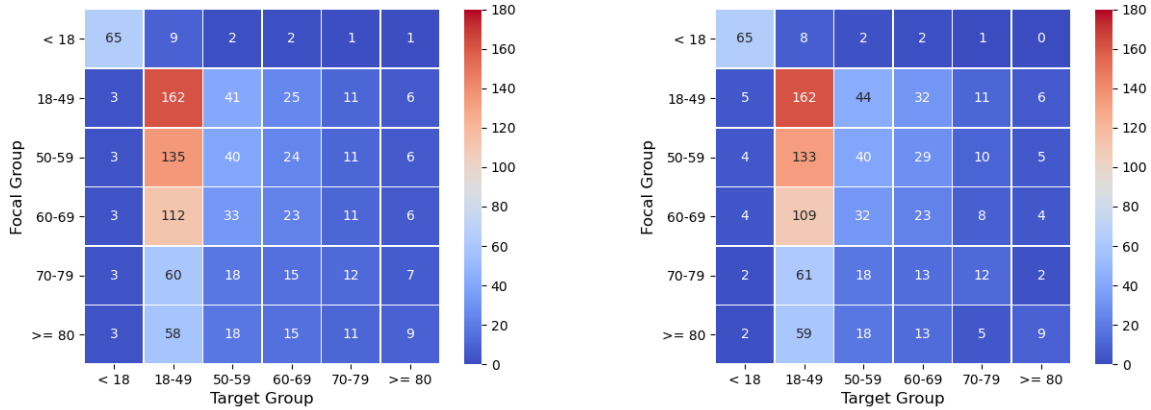
Preference/Matching.- We set the discount rate r to be $0.05/365$, which leads to a daily calibration. The preference shocks are parameterized by Normal distribution with type varying mean values. We normalize the variance of the preference shock to be 1. The average values from matchings and contacts are picked jointly in order to match the pre-COVID matching patterns among age groups, as in the recent work by Akbarpour et al. (2020). One salient feature of the pre-COVID matching matrix is that it already has a substantial degree of sorting. For example, the age group of 0 to 18 are significantly more likely to meet others with their own types. To not over attribute sorting to the endogenous channel in this paper, we allow the preference shocks to be matching pair specific. However, this creates an issue of identification because we do not have enough equations to pin down a pair-specific average value. To get around this issue, we assume the average value of matches for each types can take two possible values, one for matches within the same type and one for matches across types. Specifically: $\mu_{j,k} = \mathbb{I}\{j = k\}\mu_j^{self} + \mathbb{I}\{j \neq k\}\mu_j^{across}$. Thus exogenous homophily can be generated by a higher expected value from within-type matches. The detailed value of contact rates and expected value of matches are detailed in Table 1:

Epidemiology Parameters. - We start the economy with a initial infection population of 5.27×10^{-6} . To ensure the initial growth rate of infection is the same across all age groups, we parameterize the distribution of initial infection among different groups as the eigenvector of pre-COVID contact matrix associated with the largest eigenvalue. The detail is included

Table 1: Calibration: Matching Parameters

Age Group	Contact Rate (Daily)	E_{ν}^{within}	E_{ν}^{across}
≤ 18	35.69	-0.19	-1.92
19 – 49	100.00	-0.83	-0.87
50 – 59	42.06	-0.21	-0.21
60 – 69	34.15	-0.22	-0.20
70 – 79	27.18	-0.06	-0.53
≥ 80	31.66	-0.06	-0.69

Figure 2: Pre-Covid Contact Matrix: Data and Model



(a) Data: Contact Matrix - Chicago 2019 Qrt 1

(b) Model

in Table 2. From the model, the initial doubling time of a case can be approximated by the reproduction number using the pre-COVID matrix. We target a basic reproduction number $R_0 = 3.1$, this result in a baseline infection probability of 9.1×10^{-4} .

When the model is simulated in an environment without endogenous matching decisions, the exogenous matching rate is a normalization. Specifically, any scaled matrix that matches the basic reproduction number would produce the same results of infections. However, the level of contact rates matter in our model. When people meet more, we would calibrate a small baseline infection probability β_0 for a fixed R_0 , which would governs how much people respond to infection probability. We thus scale the contact matrix to different values, and compare the simulated path of equilibrium. If β_0 is close to zero, this our simulation is close to a model without endogenous choices of matching. As documented by Farboodi et al. (2021), during the first two weeks of the pandemic, there is a large drop in activity even without policy intervention (a 50% reduction in activity). We believe the individual choices are playing a vital role in the unfold of COVID-19, we thus pick the scaled β_0 (baseline times

Table 2: Calibration: Epidemiology Parameter

Parameter	Symbol	Value	Target
Termination Rate	r	0.05/365	Discount Rate
Recovery	γ	1/15	15-day Recovery
Initial Infection	ϵ	5.27×10^{-6}	
Baseline Infection Prob.	β_0	0.09	$R_0 = 3.1$

Table 3: Calibration: Age-Specific Parameter

Age Group	Loss (ϕ_j)	Population Share
≤ 18	-6,437	0.25
19 – 49	-12,171	0.40
50 – 59	-46,646	0.13
60 – 69	-80,193	0.12
70 – 79	-214,895	0.07
≥ 80	-429,948	0.03

100) as our preferable calibration.

The loss to infection ϕ_j is interpreted as the expected loss due to death. In order to calibrate this number, we use the value of statistical life based on [Hall et al. \(2020\)](#): people are willing to forgo $31.71 \times r$ of consumption to reduce death by probability by 0.1 percent. We thus calibrate ϕ_j to match the same reduction in utility from matches.

Results.- Figure 2 plots the time path of infection probability and susceptible probability by age groups, from both the model in this paper and from an SIR model that takes the pre-COVID matching matrix as given. Comparing the graph in panel (a) and panel (b) to the graph in panel (c) and panel (d), we observe several specific features of our model.

First, the peaks of infection rate for different age groups arrive at very different period in our model. This comes from the fact older groups suffer more loss from infection. At earlier stage of the pandemic, they optimally choose to avoid matching with others. This equilibrium force is missed in a conventional SIR model without endogenous response. This pattern is also featured in recent work by [Brotherhood et al. \(2020\)](#). Second, even within age groups, there can be multiple waves of infection. As the pandemic eases, older groups gradually reduce their caution. This comes from the fatigue force and the fact many infection cases of other groups are already recovered, and the risk of catching the virus is significantly lowered. The endogenous response of individuals in the equilibrium significantly reduces the peak-time infection of all groups, especially for the older groups.

Figure 3: Infection Probability (Baseline)

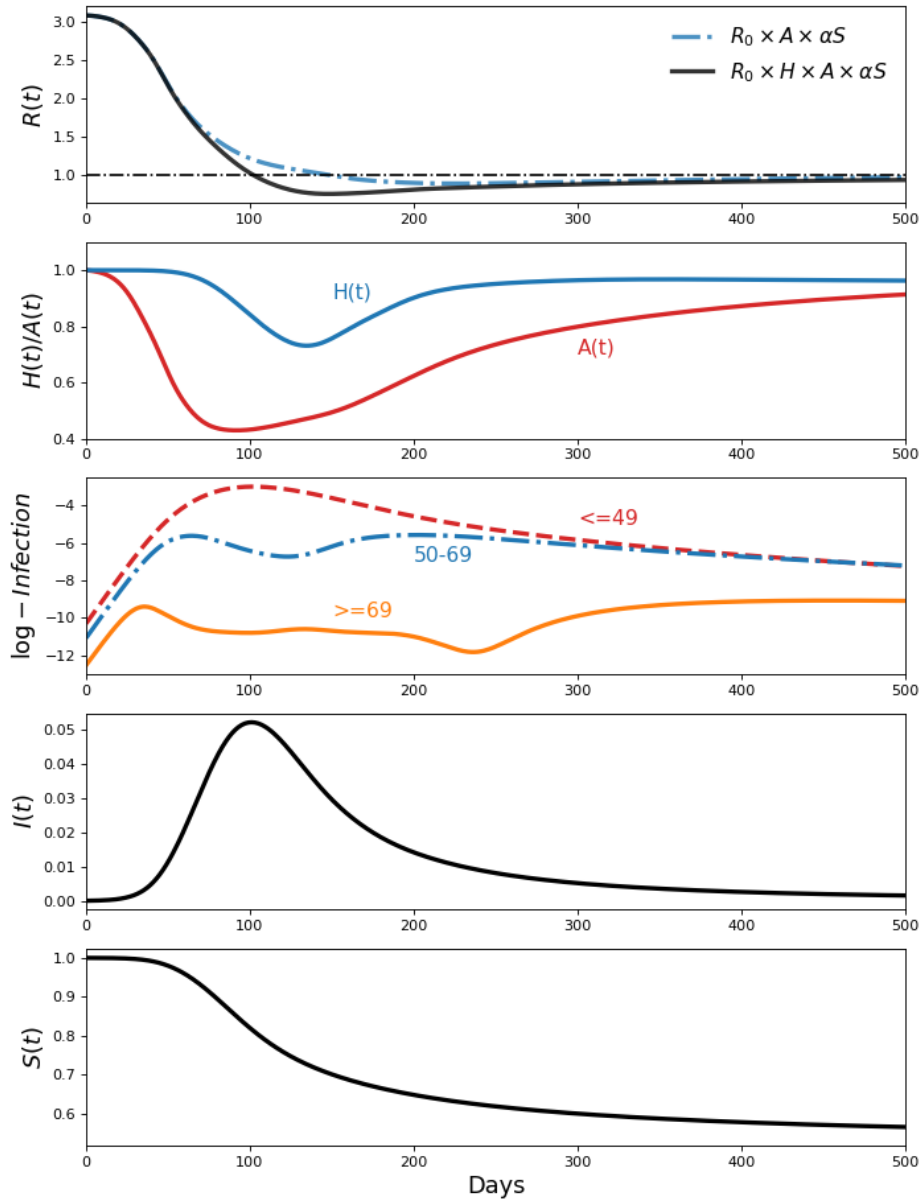


Figure 4: Infection Probability (Exogenous)

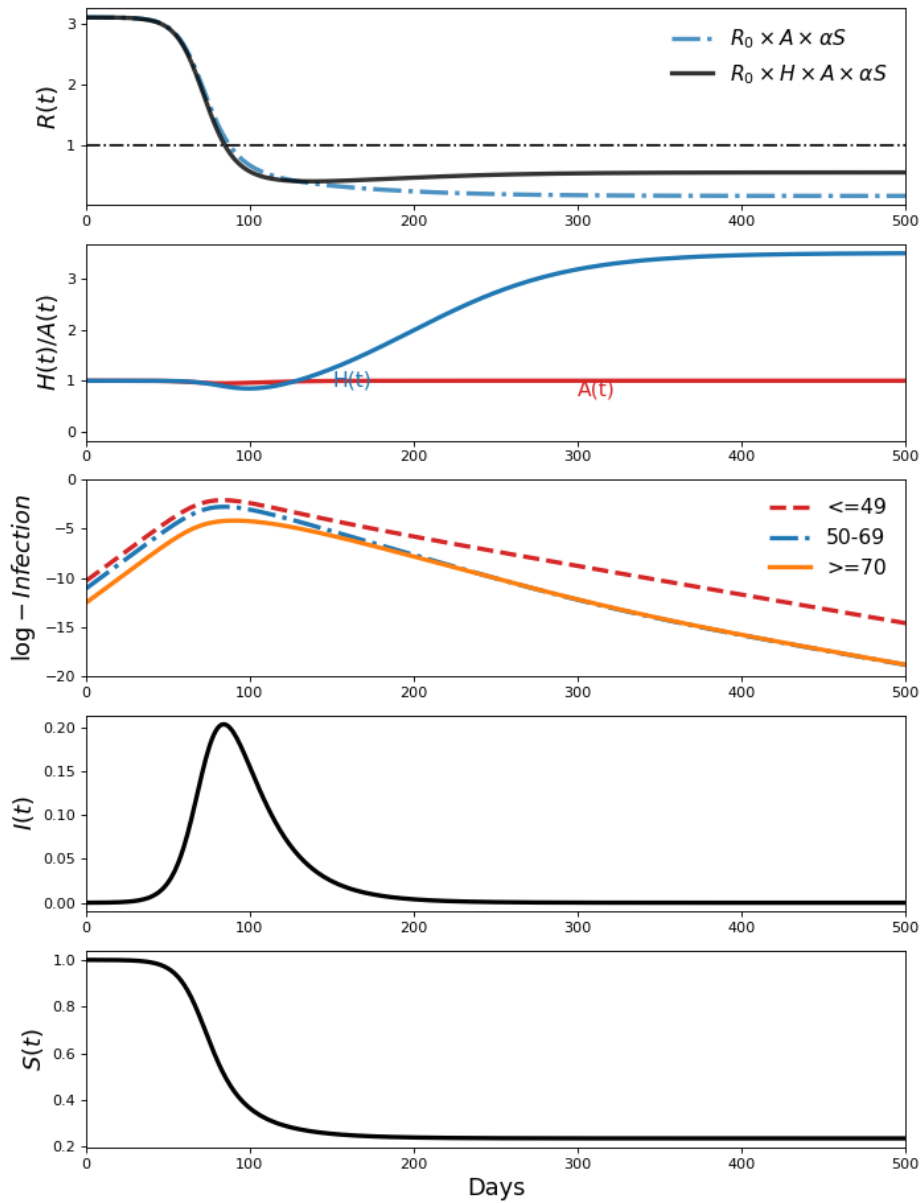


Figure 5: Sorting Pattern

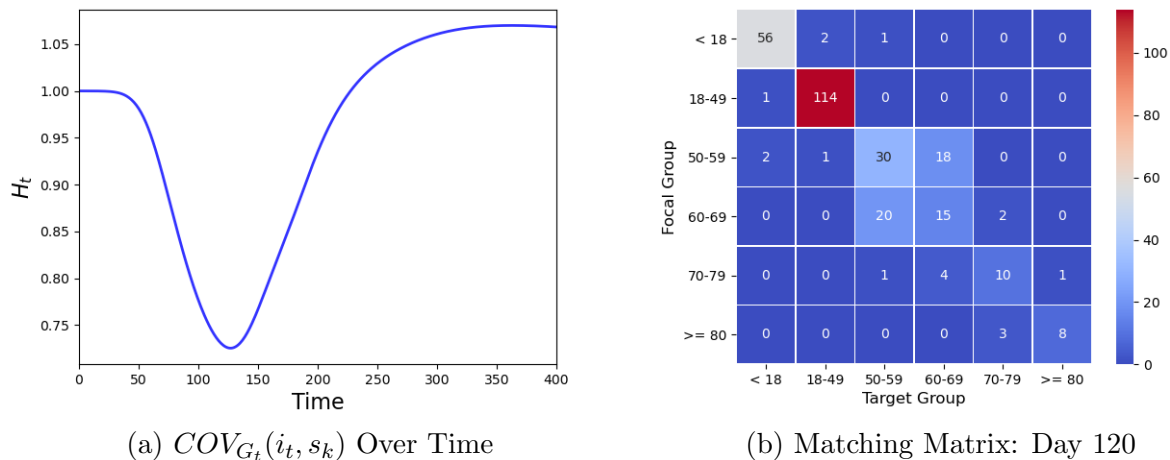


Table 4: Decomposition of R_t

Component	R_t^c	R_t^A	R_t^H
t=0	3.1	3.1	3.1
% γ		(-0)	(-0)
t=50	4.5	2.6	2.52
% γ		(-0.43)	(-0.03)
t=120	2.8	1.4	1.0
% γ		(-0.5)	(-0.27)
t=250	1.05	0.79	0.82
% γ		(-0.23)	(+0.02)

We further investigate the sorting pattern over different period of the pandemic. In Figure 5, we plot the measure of assortative matching on risk, the weighted covariance between the infection probability and susceptible probability of matched pairs. In the case with recovery, we still have assortative matching on risks over the whole episode of pandemic. The strength of sorting is time-varying. At the onset of pandemic and at the end of pandemic, sorting is zero. This result comes from the fact at both the beginning and the end of a pandemic, all types have the same probability to pass on the virus. In panel (b), we plot the matching matrix at day 120 of the pandemic, when the prevalence of infection is high. All agents cut their social interactions, more so for the older groups. Older groups cut more interactions with the young groups.

Utilizing the decomposition developed in section 2, we calculate the three reproduction numbers at different periods of the pandemic. At $t = 0$, there is no sorting. Thus, $R_t^c = R_t^A =$

R_t^H . As the virus becomes more prevalent, individuals start to reduce their interactions, which is reflected on a reduction from R_t^c to R_t^A . During the early periods, sorting plays a very small role of preventing transmission of virus, because different age groups are very similar in their relative infection probability. However, around the peak of the pandemic ($t = 120$), sorting plays a sizable role. The homophily index reduces the effective reproduction number by 27%.

6.2 Efficiency: Lock-down or Open-up?

We now compare the decentralized equilibrium outcomes to the social planner's solution. As noted in section 5, there are two sources of inefficiency in this economy. One comes from the local increasing return to scale of matches, and another due to the diffusion externality. The novel and more interesting inefficiency is the diffusion externality. In order to highlight this inefficiency, we suppose the planner can use tools to motivate individuals in meetings such that their matching decisions reflect the increasing return-to-scale. We refer to this allocation as equilibrium in this section.

Figure 6 plots the time paths of infection probability within each age group, for both the equilibrium outcome and the planner's solution. We note several features of their disparities. First, the discrepancy between these two allocations depends on age groups. For the younger groups (age less than 59), the planner allocates a higher and faster infection probability for all groups. For older groups (age more than 60), the planner chooses a lower infection probability in the first wave, and faster second waves. Second, peak of the infection arrives faster in planner's allocation than in the equilibrium allocation for all groups. Figure 8 further shows how would the planner creates such different pattern. The most striking feature is that the planner chooses to increase interactions among young groups at the onset of the pandemic. Put in another words, it is socially beneficial for young individuals to meet more and spread the virus faster. This increasing in activity is associated with a stronger reduction in the interaction between old and young groups. Put in another word: with sorting, the society could pass the peak faster by increasing interactions. This type of allocation can never be achieved in decentralized equilibrium. As we showed in earlier sections, the private caution is always positive, meaning individuals reduce their activities with everyone else, regardless of their ages.

Why would the planner wants to increase young groups' activity during a pandemic? To understand this, we compare three types of meetings: young-young meetings, old-old meetings, and young-old meetings. The planner wants to protect the old more from infection because they suffer more from infection. The most risky meeting type is the young-old when

Figure 6: Comparison between Planner and Equilibrium Allocation

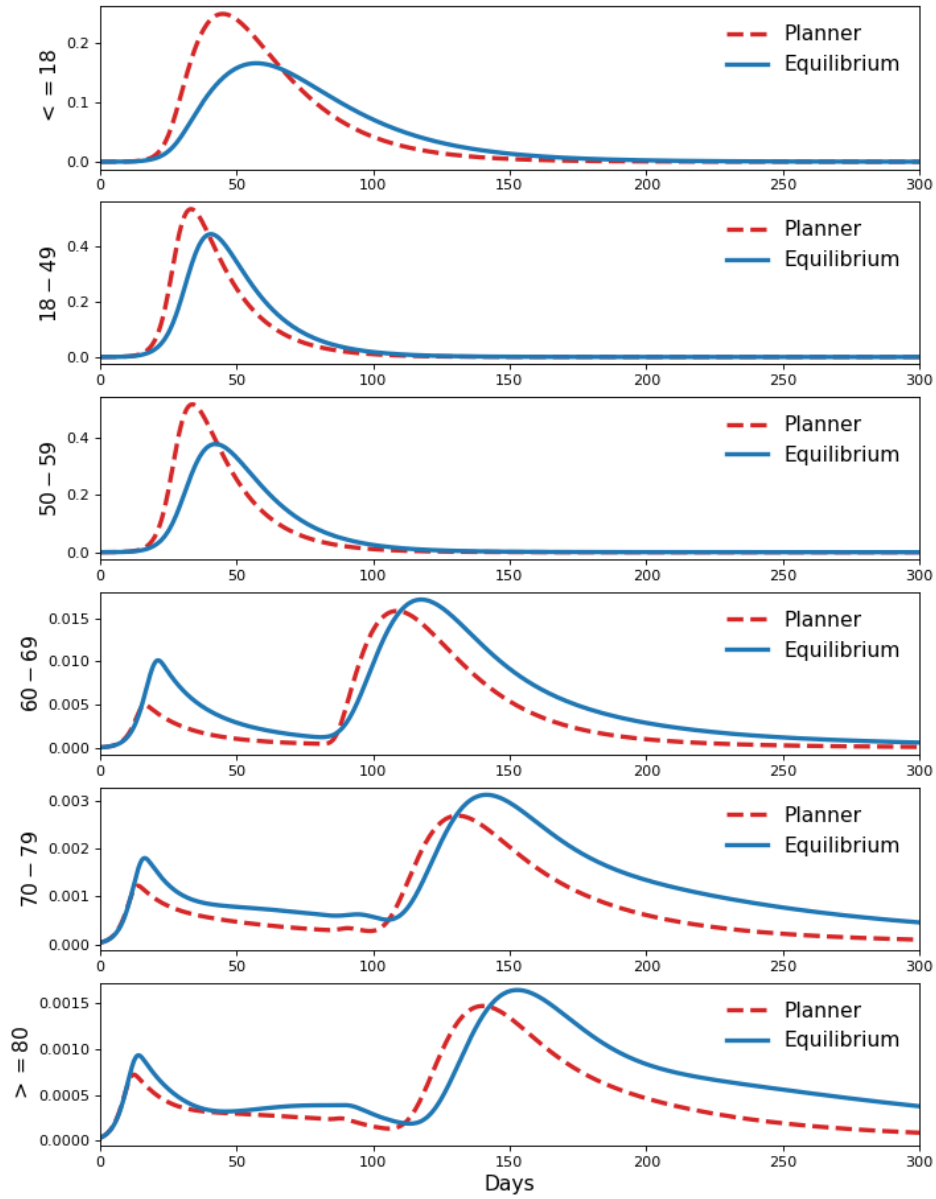
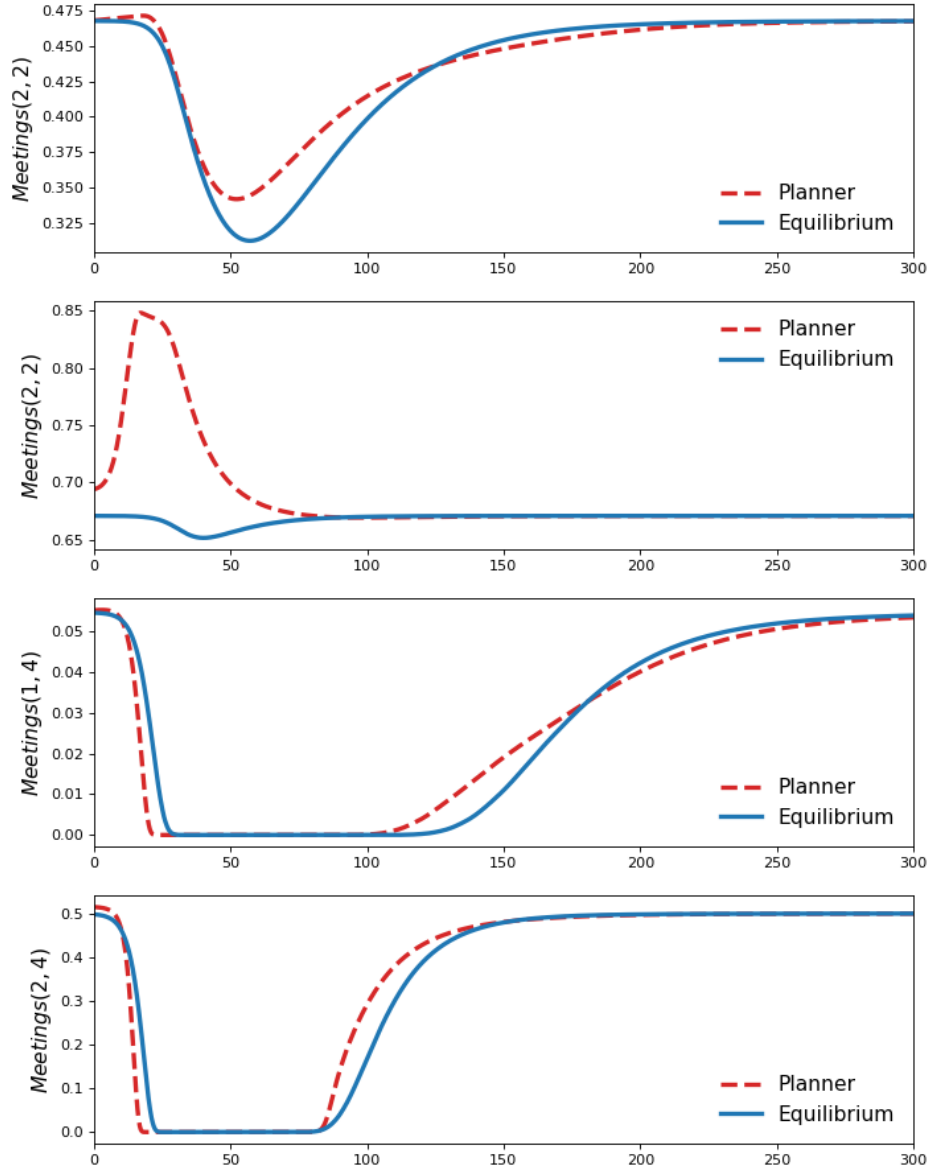


Figure 7: Comparison between Planner and Equilibrium Allocation

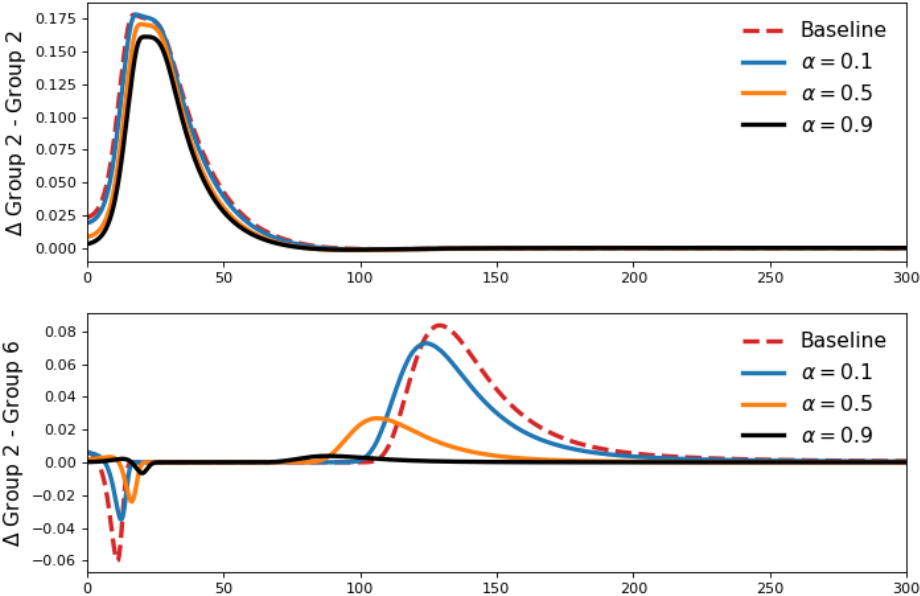


the old population are still largely susceptible and the young population is largely infected. The planner can reduce these types of meetings in two ways: either to reduce these meetings all along or make sure young population are already recovered when the meetings between

young and old resume. The first method requires a long period of restriction in activity, while the second requires a shorter period of reduction. Given the distribution of preference in our calibration, it is more worthwhile for the young to increase activity, to get infected and recover faster, and shorten the period of reduction in activity.

Sorting is important for the planner’s choice of increasing meeting probabilities among the young population. During the period where she instructs young population to interact more with each other, she also reduces the meeting probabilities between young group and old group to a very low level. In reality, this policy might not be feasible. For example, some interactions between young and old are impossible to avoid, such as nursing homes. In order to illustrate this logic and tests the robustness of our findings, we impose a lower bound on interactions among types. More specifically, we assume α fraction of meetings must result in a match. Taking the nursing home example, assume α fraction of old population needs to live with the help of young workers. They draw a utility shock of positive infinity from interactions. In our next numerical simulation, we focus on the social planner’s choice of match probabilities as a function of α .

Figure 8: Robustness with Different α



As shown in Figure 8, when more meetings between young and old group are unavoidable, the gap between planner’s solution and the equilibrium allocation are smaller. However, the planner still wants to increase meetings within group 2 with even a very high share of

unavoidable meetings ($\alpha = 0.9$). This comes from the fact (1) young groups take a large population share and tend to meet with each other and (2) old group takes a very minimum share of meetings among young cohort. This result would change if these patterns are different. We believe a small share of unavoidable meetings between young and old cohorts fit the reality better⁴.

7 Conclusion

How heterogeneous individuals interact with each other is an important determinant of diffusion process. We provide a theory of endogenous formation of these matching patterns, and characterize its time variation. Through both analytical results and numeric simulation, we highlight the endogenous sorting pattern that emerges in equilibrium, and its role in accelerating or slowing down the diffusion of infectious disease or valuable information. We show the optimal intervention can either increase or decrease activity during a pandemic, depending on the heterogeneity among types and how they interact with each others.

⁴In the U.S., only 8.5 percent of old population lives in a nursing home.

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8 Appendix

8.1 Details of Exogenous Matching Case

In the exogenous matching case, we define:

$$z(\lambda, t) = \beta \int_0^t \int_{\lambda'} m(\lambda, \lambda') i(\lambda', \tau) dG(\lambda') d\tau$$

Taking the time derivative:

$$\dot{z}(\lambda, t) = \beta \int_{\lambda'} m(\lambda, \lambda') i(\lambda', t) dG(\lambda')$$

Utilizing the fact recovery, susceptible and infected probability add up to 1:

$$\dot{z}(\lambda, t) = \beta \int_{\lambda'} m(\lambda, \lambda') \left(1 - s(\lambda', t) - r(\lambda', t)\right) dG(\lambda')$$

Further:

$$\begin{aligned} \int_{\lambda'} m(\lambda, \lambda') dG(\lambda') &= \lambda \\ \int_{\lambda'} m(\lambda, \lambda') s(\lambda', t) d\lambda' &= \int_{\lambda'} m(\lambda, \lambda') (1 - \epsilon_{\lambda'}) e^{-z(\lambda', t)} dG(\lambda') \\ \int_{\lambda'} m(\lambda, \lambda') r(\lambda', t) dG(\lambda') &= \gamma z(j, t) \end{aligned}$$

so we get:

$$\dot{z}(j, t) = \beta \lambda - \beta \int_{\lambda'} m(\lambda, \lambda') (1 - \epsilon_{\lambda'}) e^{-z(\lambda', t)} dG(\lambda') - \gamma z(j, t)$$

8.2 A parametric case

Suppose

$$\begin{aligned} m(\lambda, \lambda') &= \lambda \lambda' m(\lambda' | \lambda) \\ G(\lambda) &= 1 - e^{-\alpha \lambda} \end{aligned}$$

And the meetings are generated by the Farlie-Gumbel-Morgenstern Copula:

$$C(u, v) = uv + \theta u(1 - u)v(1 - v)$$

8.3 Formal Derivation of Optimal Conditions

We first write out the Hamiltonian of individuals of type j :

$$\begin{aligned} \mathbf{H} = & \max_{q_{j,k}} \int_0^1 \lambda_j \lambda_k Q_{k,j}(t) w(q_{j,k}(t)) dk + r \phi_j i_j(t) \\ & - (\mu_j^s - \mu_j^i) \beta_0 s_j(t) \int_k \lambda_j \lambda_k Q_{k,j}(t) q_{j,k}(t) I_k(t) dk - \gamma \mu_j^i i_j \end{aligned} \quad (10)$$

We take the first-order condition and simplifies:

(Optimal)

$$q_{j,k} = 1 - F(\beta_0(\mu_j^s - \mu_j^i) s_j I_k)$$

(Accounting)

$$\begin{aligned} \dot{\mu}^s &= r \mu^s + \beta_0 s_j(t) (\mu_j^s - \mu_j^i) \int_k \lambda_j \lambda_k Q_{k,j}(t) q_{j,k}(t) I_k(t) dk \\ \dot{\mu}^i &= (r + \gamma) \mu^s - r \phi_j \end{aligned}$$

(Transversality)

$$\begin{aligned} \lim_{t \rightarrow \infty} e^{-rt} \mu_j^s(t) &= 0 \\ \lim_{t \rightarrow \infty} e^{-rt} \mu_j^i(t) &= 0 \end{aligned}$$

We define two terms:

(Caution)

$$\begin{aligned} \kappa_j(t) &= s_j(t) (\mu_j^s - \mu_j^i) \\ \beta_j(t) &= \beta_0 (\mu_j^s - \mu_j^i) \int_k \lambda_j \lambda_k Q_{k,j}(t) q_{j,k}(t) I_k(t) dk \end{aligned}$$

Combining these two definitions and the optimal condition we reach exactly the same equation systems as in Lemma 3.

8.4 Proof of Proposition 1

Case 1: $\lambda_1 = \lambda_2$ while $0 > \phi_1 > \phi_2$.

Suppose we find a interval of time $[t_1, t_2]$ during which:

$$i_1(t) \leq i_2(t)$$

The rational decision of other individuals implies that for any $t \in [t_1, t_2]$

$$Q_{j,1}(t) \geq Q_{j,2}(t)$$

For type 1 and type 2 to start with the same infection probability yet end up with $i_1 \leq i_2$, there must be a sub periods during which:

$$\beta_2(t) > \beta_1(t).$$

Call the collection of the these sub periods T_1 . We want to show this type of interval must not exist if both types are behaving optimally.

Step a. Consider the following deviation:

$$\tilde{\beta}(t) = \min\{\beta_2(t), \beta_1(t)\}.$$

For type 2 to not want to deviate to $\tilde{\beta}$, it must be:

$$\int_{T_1} e^{-rt} W(\beta_2; 2, t) dt + r\phi_2 \int_0^\infty e^{-rt} i_2(t) dt \geq \int_{T_1} e^{-rt} W(\beta_1; 2, t) dt + r\phi_2 \int_0^\infty e^{-rt} \tilde{i}_2(t) dt,$$

where \tilde{i}_2 is the path of infection probability for type 2 if she uses $\tilde{\beta}$ strategy.

Step b. Consider the following deviation

$$\hat{\beta}(t) = \max\{\beta_2(t), \beta_1(t)\}$$

For type 1 to not want to deviate to $\hat{\beta}$, it must be:

$$\int_{T_1} e^{-rt} W(\beta_2; 1, t) dt + r\phi_1 \int_0^\infty e^{-rt} \hat{i}_1(t) dt \leq \int_{T_1} e^{-rt} W(\beta_1; 1, t) dt + r\phi_1 \int_0^\infty e^{-rt} i_1(t) dt,$$

Step c. we show this creates a contradiction.

Due to super-modularity of W :

$$\int_{T_1} e^{-rt} \left(W(\beta_2; 2, t) - W(\beta_1; 2, t) \right) dt \leq \int_{T_1} e^{-rt} \left(W(\beta_2; 1, t) - W(\beta_1; 1, t) \right) dt$$

Because $\tilde{i}_2 < i_2$:

$$r\phi_2 \int_0^\infty e^{-rt} (i_2 - \tilde{i}_2) dt < r\phi_1 \int_0^\infty e^{-rt} (i_2 - \tilde{i}_2) dt = r\phi_1 \int_0^\infty e^{-rt} (\tilde{s}(t) - s_2(t)) dt$$

We want to show:

$$\int_0^\infty e^{-rt} (\tilde{s}(t) - s_2(t)) dt > \int_0^\infty e^{-rt} (s_1(t) - \hat{s}(t)) dt$$

To show this, we first note:

$$\log(\tilde{s}(t)) - \log(s_2(t)) = \int_{T_1 \cap [0, t]} (\beta_1(\tau) - \beta_2(\tau)) d\tau = \log(s_1(t)) - \log(\hat{s}(t)) > 0$$

Then we note:

$$\log s_1(t) = \log \epsilon - \int_0^t \beta_1(\tau) d\tau < \log \epsilon - \int_0^t \tilde{\beta}_1(\tau) d\tau = \log \tilde{s}(t)$$

Because e^h is convex and $\log s_1(t) < \log \hat{s}(t)$ so:

$$\tilde{s}(t) - s_2(t) > s_1(t) - \hat{s}(t)$$

As a result:

$$\int_0^\infty e^{-rt} (\tilde{s}(t) - s_2(t)) dt > \int_0^\infty e^{-rt} (s_1(t) - \hat{s}(t)) dt$$

Because $\phi_1 < 0$:

$$r\phi_1 \int_0^\infty e^{-rt} (\tilde{s}(t) - s_2(t)) dt < r\phi_1 \int_0^\infty e^{-rt} (s_1(t) - \hat{s}(t)) dt$$

This means if:

$$\int_{T_1} e^{-rt} W(\beta_2; 2, t) dt + r\phi_2 \int_0^\infty i_2(t) dt \geq \int_{T_1} e^{-rt} W(\beta_1; 2, t) dt + r\phi_2 \int_0^\infty \tilde{i}_2(t) dt,$$

then

$$\int_{T_1} e^{-rt} W(\beta_2; 1, t) dt + r\phi_1 \int_0^\infty \hat{i}_1(t) dt > \int_{T_1} e^{-rt} W(\beta_1; 1, t) dt + r\phi_1 \int_0^\infty i_1(t) dt,$$

A contradiction! For $i_1(t) < i_2(t)$, there must be periods like T_1 . Non-existence of T_1 rules out any interval of time with $i_1(t) < i_2(t)$.

Case 2: $\lambda_1 > \lambda_2$ while $0 > \phi_1 = \phi_2$.

This case can be proved using the same method as Case 1.

Case 3: $\lambda_1 = \lambda_2$ while $\phi_1 > \phi_2 > 0$.

Take any crossing point where $i_1(T) = i_2(T)$. We want to show:

$$\kappa_1(T) < \kappa_2(T).$$

Suppose it is the opposite. Using the $\dot{\kappa}_j$ equation we get:

$$\dot{\kappa}_1(T) - \dot{\kappa}_2(T) = r(\kappa_1(T) - \kappa_2(T)) + \frac{r^2(\phi_1 - \phi_2)}{r + \gamma} s_1(T) > 0$$

We can find a small neighborhood of $(T, T + t)$ such that:

$$s_1 > s_2, \quad \kappa_1 > \kappa_2$$

For any of these instant of time

$$\dot{\kappa}_1 - \dot{\kappa}_2 = r(\kappa_1(T) - \kappa_2(T)) + \frac{r^2\phi_1}{r + \gamma} s_1(T) - \frac{r^2\phi_2}{r + \gamma} s_2(T) > r(\kappa_1(T) - \kappa_2(T)) + \frac{r^2\phi_2}{r + \gamma} s_1(T) - \frac{r^2\phi_2}{r + \gamma} s_2(T) > 0$$

Also at this small neighborhood, $i_1 < i_2$. Type 2 is more likely to have matches because they are more likely to be informed. This leads to:

$$\dot{i}_1 < \dot{i}_2$$

This argument can goes on forever until the transversality condition is violated. This rules out any crossing of probability such that:

$$\kappa_1(T) \geq \kappa_2(T)$$

This means at any crossing point of p_1 and p_2 , type 1 must accept more matches than type 2 and ends up with higher probability of infection. This leads to a unique crossing, from $t = 0$. Thus we have shown:

$$i_1(t) \geq i_2(t).$$

Case 4: $\lambda_1 > \lambda_2$ while $\phi_1 = \phi_2 > 0$.

This case can be proved in exactly the same method as in Case 3.

8.5 Proof of Corollary 1

From proposition 1, we show the equilibrium must be rank preserving in terms if infection probability. As a result it must also be rank preserving in terms of susceptible probability. According to the solution of $\kappa_j(t)$ as a function of $s_j(t)$:

$$\kappa_j(t) = -\frac{r^2\phi_j}{r + \gamma} \int_t^\infty e^{-r(\tau-t)} s_j(\tau) d\tau$$

Case 1: $\lambda_1 = \lambda_2$ while $0 > \phi_1 > \phi_2$.

In this case we showed:

$$s_1(t) < s_2(t)$$

Thus:

$$\kappa_1(t) = -\frac{r^2\phi_1}{r+\gamma} \int_t^\infty e^{-r(\tau-t)} s_1(\tau) d\tau < -\frac{r^2\phi_2}{r+\gamma} \int_t^\infty e^{-r(\tau-t)} s_1(\tau) d\tau < -\frac{r^2\phi_2}{r+\gamma} \int_t^\infty e^{-r(\tau-t)} s_2(\tau) d\tau = \kappa_2(t)$$

Case 2: $\lambda_1 > \lambda_2$ while $0 > \phi_1 = \phi_2 = \phi$.

In this case we showed:

$$s_1(t) < s_2(t)$$

Thus:

$$\kappa_1(t) = -\frac{r^2\phi}{r+\gamma} \int_t^\infty e^{-r(\tau-t)} s_1(\tau) d\tau < -\frac{r^2\phi}{r+\gamma} \int_t^\infty e^{-r(\tau-t)} s_2(\tau) d\tau = \kappa_2(t)$$

Case 3: $\lambda_1 > \lambda_2$ while $0 < \phi_1 = \phi_2 = \phi$.

In this case we showed:

$$s_1(t) < s_2(t)$$

Thus:

$$\kappa_1(t) = -\frac{r^2\phi}{r+\gamma} \int_t^\infty e^{-r(\tau-t)} s_1(\tau) d\tau > -\frac{r^2\phi}{r+\gamma} \int_t^\infty e^{-r(\tau-t)} s_2(\tau) d\tau = \kappa_2(t)$$

8.6 Discussion of Symmetry in Planner's Solution

Consider the following problem:

$$\max_{q_1, q_2} q_1 w(q_2) + q_2 w(q_1)$$

s.t.

$$q_1 q_2 = Q$$

We can re-write as a unconstrained problem

$$\max_q q w\left(\frac{Q}{q}\right) + \frac{Q}{q} w(q)$$

The second order derivative reads:

$$\frac{Q}{q^2} \left(2\left(\frac{w(q)}{q} - w'(q)\right) + qw''(q) + \frac{Q}{q} w''\left(\frac{Q}{q}\right) \right)$$

Evaluate this at the symmetric point $q = \sqrt{Q}$

$$D = 2 \left(\frac{w(q)}{q} - w'(q) + qw''(q) \right)$$

Using the definition of $w(q)$

$$\frac{w(q)}{q} - w'(q) = \mathbb{E}[\nu - z | \nu \geq z]$$

$$qw''(q) = -\frac{1 - F(z)}{f(z)}$$

As a second order test, we ask if $D < 0$ at the symmetry point:

$$D = \mathbb{E}[\nu - z | \nu \geq z] - \frac{1 - F(z)}{f(z)}$$

For Exponential: $\mathbb{E}[\nu - z | \nu \geq z] = \frac{1}{\lambda} = \frac{1 - F(z)}{f(z)}$, so $D = 0$.

For Pareto: $\mathbb{E}[\nu - z | \nu \geq z] = \infty > \frac{1 - F(z)}{f(z)}$, so $D > 0$.

For Normal: $\mathbb{E}[\nu - z | \nu \geq z] = \mathbb{E}[\nu] - z + \frac{f(z)}{1 - F(z)} \cdot \frac{1 - F(z)}{f(z)}$.