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Abstract

We develop a quantitative theory to study disease dynamics and the welfare consequences of endogenous travel choices. Travel responds to and influences disease dynamics, serving as an infection avoidance strategy and a vector for pandemic transmission. We integrate a metapopulation SIRD epidemiological framework with an economic model to recover consistent travel choices. We analytically characterize the role of travel as an infection avoidance mechanism, distinct from and in addition to home isolation. We then quantify its relative importance during a pandemic. We show local pandemic policies lead to suboptimal Nash equilibrium outcomes and train surrogate models to assess parameter impacts.

Keywords: epi-econ metapopulation model, endogenous travel, pandemic federalism, travel bans, surrogate models.
JEL Classification: C6, C7, E6, H7, I1, Z3.

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1 Introduction

The interconnection of regions via travel is a crucial component in the emergence of pandemics. Dating back to the Plague of Justinian in the 6th Century, it is travel that ultimately puts the "pan" in pandemics. While travel constitutes a small fraction of a typical household's time and budget, its key role in disease spread makes it a primary policy concern. Household travel choices, responding to differences in infection rates, may lead to intermixing of populations and will likely influence the trajectory of a pandemic, especially in presence of uncoordinated, unilateral, public policy interventions across different locales ("pandemic federalism").

In this paper, we develop a coupled epidemiological-economic (epi-econ) metapopulation model to examine how individuals' travel decisions interact with the spread of disease. Our quantitative theory comprises two heterogeneous populations connected through interregional travel. At its core, our framework integrates a standard SIRD epidemiological system, capturing individuals' transitions through disease states (Susceptible, Infectious, Recovered, Deceased), with an economic model, where the SIRD compositions of each population segment influence agents' decisions regarding travel, consumption, labor, and home isolation. Central to our approach is establishing the connection between epidemiological dynamics and economic behavior through the probability of disease transmission. These infection probabilities dictate the pandemic's course, shaping individual activity and travel decisions, which in turn affect transmission probabilities, creating a feedback loop. Our theoretical framework leads to sharp analytical results, which we use to cleanly calibrate parameters and interpret our quantitative findings.

We analytically characterize travel and home isolation choices during a pandemic. In our metapopulation model, susceptible individuals (those at risk of becoming sick) can either isolate at home or travel in response to disease progression in their region. We show travel can act as either an infection avoidance asset or liability, depending on whether traveling to a foreign region reduces or increases the likelihood of getting infected. We further identify the conditions under which travel is preferred over home isolation as an avoidance strategy. In this context, we highlight key drivers, including activity-specific contact rates with infected individ-

uals and the relative share of infectious in home and foreign populations. We also demonstrate that travel is preferred to home isolation when infection rates are low.

We calibrate our metapopulation model to the U.S. economy. In order to conduct positive and normative analysis for regions of different size, we focus on a two-region version of our model. This environment leads to a fast solution algorithm that allows us to explore over one million epidemiological-economic parameter configurations. Our quantitative analysis is conducted in two policy-relevant scenarios: one with regions of similar size (symmetric) and another where one region is significantly smaller than the other (asymmetric).

Our positive analysis focuses on the feedback loop between the economic and epidemiological modules. We first study how susceptible individuals respond to the evolution of the pandemic. In the symmetric case, susceptible individuals prefer travel to isolation at the onset and ending of a pandemic. Our theory shows that in these circumstances, isolation is less effective than travel due to both low infection rates and the high utility cost of foregone travel. In the asymmetric case, susceptibles in the small region use travel and susceptibles in the large region use isolation for avoidance throughout the pandemic. In both environments, the role of travel as infectious avoidance asset or liability evolves over time and is quantitatively driven by both relative contact rates and differential rates of exposure at home and abroad. We then investigate how the disease dynamics are affected by these behavioral responses. To isolate the connectivity role of travel, we compare a metapopulation model without travel against different specifications of the model incorporating exogenous and endogenous travel choices. Both exogenous and endogenous travel synchronizes peak infectious across regions. When regions are asymmetric, endogenous traveling behavior leads to a nearly threefold increase in peak infectious in the small region.

Our normative analysis of pandemic federalism reveals that the Nash game between local planners leads to the classic Prisoner's Dilemma outcome. Local policymakers' best responses are to impose the most stringent travel restrictions on incoming travelers (to mitigate the externality effect), while at same time impose no restrictions on their own citizens' outgoing travel, such that they can take advantage of the avoidance and utility benefits of travel. With both policymakers

following this strategy, the resulting equilibrium is very close to that under travel autarky (effectively a travel ban), leading to significant welfare losses relative to the optimal coordinated policy by a centralized authority.

The scope of our quantitative results is inherently constrained by the specific parameterization of our model. To cope with this challenge, we perform a comprehensive sensitivity analysis over one million combinations of structural parameters governing epidemiological and economic characteristics. Across the parameter space, we confirm travel to be an effective infection avoidance asset for low infection rates (that is, at the beginning and ending of the pandemic) and the Prisoner's Dilemma outcome to be a Nash equilibrium in 99% of economies. We find endogenous travel reduces peak infection rates, uncovering a more relevant role of endogenous travel for mitigating the heat of the pandemic than that indicated by our benchmark (COVID-19) calibration. Unlike our benchmark, we also find socially optimal policy to involve positive restrictions on travel for 32% of economies. These last findings caution against drawing general conclusions from a single parametrization.

Finally, we then use Deep Neural Networks to estimate surrogate models of the unknown mapping from epidemiological-economic parameters to model outcomes. We identify the basic reproductive number, R_0, as the primary driver of: *(i)* disease dynamics, which, in turn, govern the effectiveness of travel as an avoidance mechanism; *(ii)* welfare losses associated with the Prisoner's Dilemma outcome. Travel is preferred more often to isolation for highly contagious diseases (R_0 greater than 6). For several prominent diseases with lower reproductive numbers, such as Ebola, H1N1, the seasonal flu, SARS, and COVID-19, we find the effectiveness of travel choices to be highly sensitive to the value of R_0, making early accurate estimates of transmissibility a primary priority for determining travel policies.

The rest of the paper is organized as follows: Section 2 summarizes the contribution of this work to the related literature, Section 3 describes the model and theoretical results, Section 4 lays out the calibration, Section 5 presents the quantitative results, Section 6 discusses the parameters space exploration, and Section 7 concludes. Additional results are presented in the Online Appendix.

2 Contribution

The incorporation of behavioral considerations into epidemiological models (e.g. the classic SIR model of Kermack and McKendrick (1927)) has been an important advance in the literature in recent years. Building on influential groundwork by Kremer (1996) and Fenichel et al. (2011), the COVID-19 pandemic produced a flurry of coupled epidemiological-economic models (e.g. Eichenbaum, Rebelo, and Trabandt, 2021, Farboodi, Jarosch, and Shimer, 2021, Ash et al., 2022, Boppart et al., 2024). Although these models differ in their quantitative and qualitative features, a key component is that disease dynamics both influence and respond to the behavioral choices made by individuals. We contribute to this literature by endogenizing travel decisions between regions in a quantitative epi-econ metapopulation model.[1]

Our work is most closely related to Antràs, Redding, and Rossi-Hansberg (2023), which studies how globalization and trade affect the evolution of pandemics. While both works feature endogenous travel decisions in a metapopulation model, they differ in several important dimensions. First, we focus on different questions; we study pandemic federalism, whereas they examine the international general equilibrium implications. Second, we propose different mechanisms for how travel affects inter-population disease diffusion. In their open-economy model, locations are connected by movements of both goods and people, while our closed-economy emphasizes the movement of people. Accordingly, their framework allows them to quantify the amplification effect of endogenous travel decisions on trade outcomes, whereas our framework allow us to *theoretically* isolate and quantify the insurance role of travel, where individuals may choose to travel to less infectious regions solely to reduce infection risk. This result is consistent with evidence from developing countries documented in Burlig, Sudarshan, and Schlauch (2021). Third, in their model, agents do not know their disease state (susceptible, infectious, recovered etc.), while we allow susceptibles to internalize the effect that their choices have on their probability of becoming infected. Finally, our theoretical framework yields closed-form solutions that address the computational challenges highlighted

[1] Metapopulation models with endogenous choices by economic agents have also been considered in natural resource contexts (e.g. Sanchirico and Wilen, 2001; Kaffine and Costello, 2011; Fabbri, Faggian, and Freni, 2024).

in their paper, enabling us to study the robustness of our results for over a million of combinations of parameter vectors and to conduct normative analysis.[2]

We contribute methodologically to the quantitative epi-econ literature by proposing surrogate models to explore the robustness of our results across the parameter space. Surrogate models combine sampling, fitting, and validation techniques to create a simplified representation of complex systems, like the epi-econ model at the heart of our analysis.[3] More precisely our work extends the regression approach in Iverson, Karp, and Peri (2022), by applying recent techniques developed in Chen, Didisheim, and Scheidegger (2023) to study how the relevance of travel for disease transmission and welfare costs varies with different policy- (and time-) invariant aspects of disease transmission and individual behavioral responses.

Our framework provides theoretical support for empirical findings that people both avoid traveling to areas with high disease burdens (Fenichel, Kuminoff, and Chowell, 2013; Brinkman and Mangum, 2022; Ojo et al., 2023) and travel away from areas with high disease burdens (Gibbs et al., 2020; Coven, Gupta, and Yao, 2023). Travel is also linked to increases in cases in the receiving destination (Kraemer et al., 2020; Julliard, Shi, and Yuan, 2023; Boto-García, 2023). Given these travel responses and the potential for jurisdictional spillovers, our paper also contributes to the literature on policy responses to pandemics (Gersovitz and Hammer, 2004; Rowthorn, Laxminarayan, and Gilligan, 2009; Kraemer et al., 2020; Boppart et al., 2024), geographic implications (Bisin and Moro, 2022), and pandemic federalism (Graff Zivin and Sanders, 2020; Renne, Roussellet, and Schwenkler, 2020; Iverson and Barbier, 2021). Our findings on decentralized policymaking related to pandemic travel are consistent with the general literature on environmental federalism, whereby externalities between regions generally leads decentralized planning

[2] As they note, the feedback between disease dynamics and agent choices increases the numerical complexity of the problem and makes it difficult to solve the model for many parameter variations. This will be true of this class of epi-econ models in general, such as Boppart et al. (2024), whose multi-generation, single-region epi-econ model conducts sensitivity analysis on a few selected parameter calibrations.

[3] From the seminal work by Box and Wilson (1951) surrogate models (also known as response surface models or metamodels) have been routinely used in chemistry (McBride and Sundmacher, 2019), physics (Tripathy and Bilionis, 2018), engineering (Alizadeh, Allen, and Mistree, 2020), econometrics (Bai and Perron, 2003), climate (Friedl et al., 2023) and finance (Scheidegger and Treccani, 2021, Chen, Didisheim, and Scheidegger, 2023).

to be suboptimal (Eichner and Runkel, 2012; Fell and Kaffine, 2014), except in certain circumstances (Landry, 2021).

3 Model

Our epi-econ model is organized in three modules. The epidemiological module in Section 3.1 dictates the evolution of the aggregate disease states; this framework expands the canonical compartmental modeling structure to multiple regions (a metapopulation model). The economic module in Section 3.2 describes individuals' behavioral responses to the state of the pandemic; the economy consists of $\mathscr{I}$ distinct regions populated by atomistic agents who optimize over consumption, labor, travel, and isolation choices, and interact with one another both within and across regional boundaries. The infection probability module in Section 3.3 establishes the connection between the epidemiological and economic modules. Subsection 3.4 describes the equilibrium. Subsection 3.5 concludes by analytically characterizing the equilibrium.

3.1 Epidemiological Module

We use an SIRD metapopulation model to describe the evolution across regions and over time of the aggregate measure of individuals over the disease states: susceptible, infectious, recovered, and deceased, $\mathscr{T}_t = \{S_{i,t}, I_{i,t}, R_{i,t}, D_{i,t}\}_{i=1}^{\mathscr{I}}$ (also referred to as compartments). Interaction and transitions across compartments in our metapopulation model are governed by the following system of equations:

$$S_{i,t+1} = \left(1 - \mathscr{P}_i^{SI}(\mathscr{A}_t, \mathscr{T}_t) - P^{D^*}\right) S_{i,t} + \gamma\left(S_{i,t} + I_{i,t} + R_{i,t}\right), \tag{1}$$

$$I_{i,t+1} = \left(1 - P^{D^*} - P^R - P^D\right) I_{i,t} + \mathscr{P}_i^{SI}(\mathscr{A}_t, \mathscr{T}_t) S_{i,t}, \tag{2}$$

$$R_{i,t+1} = \left(1 - P^{D^*}\right) R_{i,t} + P^R I_{i,t}, \tag{3}$$

$$D_{i,t+1} = D_{i,t} + P^D I_{i,t} + P^{D^*}\left(S_{i,t} + I_{i,t} + R_{i,t}\right), \tag{4}$$

where S_i, I_i, R_i, D_i are the population in each disease state for region i, γ is the population growth rate, P^R is the probability of transition from infectious to recovered, P^D is the probability of transition from infectious to deceased, and P^{D^*} is the probability of death from all other outside causes.

The force of infection $\mathscr{P}_i^{SI}(\mathscr{A}_t, \mathscr{T}_t)$ denotes the endogenous share of susceptibles that become infectious. This function depends on the share of the population in each of the disease states, $\mathscr{T}_t$, and economic activities, $\mathscr{A}_t$ (regional aggregate consumption, labor, and travel flows in and out of regions).[4] The force of infection plays the key role of connecting the epidemiological module with the economic module discussed in the following section. Section 3.3 formalizes this link.

3.2 Economic Module

The economy consists of a continuum of agents of measure one who live in one of $\mathscr{I} = 2$ home and foreign regions with competitive firms that hire labor to produce a homogeneous consumption good. The population share of region i is given by λ_i for $i \in \{1,2\}$. Although individuals may temporarily travel from one region to another, citizens of region i remain so forever.

3.2.1 Individuals

Agents make choices over consumption, labor, travel, and isolation to maximize their lifetime utility. As in Antràs, Redding, and Rossi-Hansberg (2023) and Boppart et al. (2024), we abstract from saving and borrowing decisions, due to the short time horizon. The flow utility, $u_i(c,m)$, is an increasing and concave function of a numeraire consumption good, c, and the share of time spent traveling, m. The utility satisfies the Inada conditions $\lim_{c\to 0} \partial u_i(c,m)/\partial c = \infty$, $\lim_{c\to\infty} \partial u_i(c,m)/\partial c = 0$, $\lim_{m\to 0} \partial u_i(c,m)/\partial m = \infty$, $\lim_{m\to\infty} \partial u_i(c,m)/\partial m = 0$. Additionally, we allow the utility to depend on region i to reflect different preferences for travel.[5]

[4] In the classic SIR framework, new infections at time t are equal to $\beta I_t S_t$ where β is the average number of contacts multiplied by the probability of infection per contact. In our framework, $\mathscr{P}_i^{SI}(\mathscr{A}_t, \mathscr{T}_t)$ corresponds to βI_t while allowing the number of contacts and probability of infection to vary over the course of the pandemic due to choices.

[5] To establish uniqueness of optimal travel choices, we further require q-complementarity between consumption and travel choices, $\partial^2 u_i(c,m)/\partial c \partial m \geq 0$. This assumption is satisfied by the utility

Before the start of (and after the end of) the pandemic, an agent in region i solves:

$$U_{i,t}^{pre} = \max_{\{c_t^{pre}, l_t^{pre}, m_t^{pre}\}} u_i(c_t^{pre}, m_t^{pre}) + \delta \left[(1 - P^{D^*}) U_{i,t+1}^{pre} + P^{D^*} U_{i,t+1}^{D} \right] \quad (5)$$

$$\text{s.t.} \quad c_t^{pre} = w_t \bar{l} l_t^{pre},$$
$$m_t^{pre} + l_t^{pre} = 1,$$

where w_t is the wage rate, l_t^{pre} is the labor share choice, $\bar{l}$ is the time endowment, $U_{i,t+1}^{D}$ is the value function of being deceased in the next period, where the individual exogenous probability of dying for natural causes P^{D*} coincides with the share of individuals that die for natural causes in (1)-(4). We model the start of the pandemic ($t = 0$) as an unanticipated MIT shock. We assume the pandemic ends deterministically at $t = T$. Thus, $U_{i,t}^{pre} = \bar{U}_i^{pre}$ for all $t < 0$ and $t > T$.

During the pandemic, agents observe their *individual* disease states, $z \in \{S, I, R, D\}$, the aggregate distribution of individuals over regions and disease states $\mathscr{T}_t$, and aggregate economic activities, including inflows and outflows for traveling reasons, $\mathscr{A}_t$. Susceptible agents $z = S$ solve

$$U_{i,t}^{S} \equiv U_i^S(\mathscr{A}_t, \mathscr{T}_t) = \max_{\{c_t^S, l_t^S, m_t^S, h_t^S\}} u_i(c_t^S, m_t^S) + \delta \Big[P_i^I(m_t^S, l_t^S, h_t^S; \mathscr{A}_t, \mathscr{T}_t) U_{i,t+1}^{I}$$
$$+ (1 - P_i^I(m_t^S, l_t^S, h_t^S; \mathscr{A}_t, \mathscr{T}_t) - P^{D^*}) U_{i,t+1}^{S} + P^{D^*} U_{i,t+1}^{D} \Big] \quad (6)$$
$$\text{s.t.} \quad c_t^S = w_t \bar{l} l_t^S,$$
$$m_t^S + l_t^S + h_t^S = (1 - \theta_i),$$
$$m_t^S \in [0, \min\{1 - \bar{m}_j^{in}, 1 - \bar{m}_i^{out}\}],$$
$$h_t^S \in [0, \bar{h}],$$

where θ_i imposes a limit on time spent on economic activities (e.g. local social distancing restrictions) and $\bar{m}_i^{out}, \bar{m}_j^{in}$ are restrictions on outbound travel from region i and inbound travel to region j respectively.

Crucially, in our model agents know their disease state and respond accordingly

function utilized in our quantitative analysis.

when making choices. Specifically, a key feature of our framework is that susceptibles internalize how their labor, l_t^S, travel, m_t^S, and home isolation choices, h_t^S, affect their probability of infection $P_i^I(m_t^S, l_t^S, h_t^S; \mathscr{A}_t, \mathscr{T}_t)$.[6] Note that during a pandemic, agents can also choose to isolate at home, in which case they can reduce the likelihood of getting infected, but do not receive any direct utility benefits. Section 3.3 describes the probability of infection $P_i^I(m_t^S, l_t^S, h_t^S; \mathscr{A}_t, \mathscr{T}_t)$ and its dependence on the alternative infection channels.[7]

Unlike susceptible agents, the decision of infectious and recovered individuals do not depend on the aggregate states, $\mathscr{T}_t$ and $\mathscr{A}_t$. Infectious individuals $z = I$ solve:

$$
\begin{aligned}
U_{i,t}^I = \max_{\{c_t^I, l_t^I, m_t^I, h_t^I\}} & u_i^I(c_t^I, m_t^I) + \delta \Big[(1 - P^R - P^D - P^{D^*}) U_{i,t+1}^I + P^R U_{i,t+1}^R \\
& + (P^D + P^{D^*}) U_{i,t+1}^D \Big] \qquad (7) \\
\text{s.t.} \quad & c_t^I = \phi w_t \bar{l} l_t^I, \\
& m_t^I + l_t^I + h_t^I = 1 - \theta_i, \\
& m_t^I \in [0, \min\{1 - \bar{m}_j^{in}, 1 - \bar{m}_i^{out}\}], \\
& h_t^I \in [0, \bar{h}],
\end{aligned}
$$

where ϕ is a productivity penalty associated with being sick. In addition, we allow for a different utility function for infectious $u_i^I(\cdot)$ to accommodate a reduced preference for travel while sick. Infectious individuals eventually transition to either recovered or deceased.

[6] This is in contrast to the assumption in Farboodi, Jarosch, and Shimer (2021) and Antràs, Redding, and Rossi-Hansberg (2023), where agents are uncertain and probabilistically infer their type and make decisions based on *S, I, R* type shares in the population.

[7] Alternatively, one could complicate the model and assume that after exposure to the virus, susceptible agents transition to an "exposed" type. If these exposed agents are pre-symptomatic and do not realize they have become exposed, they would make the same choices as susceptible agents. Following an incubation period, exposed individuals will then develop symptoms and become infectious. As shown in Online Appendix E.1, the presence of exposed agents introduces delays in peak infectious due to disease-specific incubation periods that yield more realistic disease dynamics, but does not alter the fundamental insights of the analysis.

The recovered problem $z = R$ is

$$U_{i,t}^R = \max_{\{c_t^R, l_t^R, m_t^R, h_t^R\}} u_i(c_t^R, m_t^R) + \delta \left[(1 - P^{D^*}) U_{i,t+1}^R + P^{D^*} U_{i,t+1}^D \right] \tag{8}$$

$$\begin{aligned} \text{s.t.} \quad & c_t^R = w_t \bar{l} l_t^R, \\ & m_t^R + l_t^R + h_t^R = 1 - \theta_i, \\ & m_t^R \in [0, \min\{1 - \bar{m}_j^{in}, 1 - \bar{m}_i^{out}\}], \\ & h_t^R \in [0, \bar{h}]. \end{aligned}$$

Infectious and recovered individuals take the transition probabilities as given. Furthermore, they do not internalize the externality associated with their choices and the effect on the infection probability $P_i^I(\cdot)$ in the susceptible problem in (6). Accordingly, the decisions that solve their dynamic problem are static.[8] Absent direct utility benefits from isolation, infectious and recovered individuals choose zero isolation, $h_t^I = h_t^R = 0$.

Those that die, either from the virus or other causes, receive a one time payoff of Ω_i, so that the value for the deceased is

$$U_{i,t}^D = \Omega_i. \tag{9}$$

3.2.2 Production

Individuals supply labor to perfectly competitive firms which use labor to produce an homogeneous consumption good (the numeraire) with a linear production technology $Y_t = AL_t$. We do not distinguish between the production technologies of different regions. Instead, we assume that the markets are sufficiently integrated such that all firms operate with identical technology and marginal products are equalized across regions.

[8] The model does not feature general equilibrium price effects from the pandemic, implying that infectious and recovered choose a constant travel share.

3.3 Infection Probability Module

The epidemiological module describes the evolution of the aggregate disease states given the behavior of individuals, while the economic module captures the behavioral response of individuals to the aggregate disease state of the economy. Connecting these two modules is the force of infection $\mathscr{P}_i^{SI}(\mathscr{A}_t, \mathscr{T}_t)$ and the infection probability $P_i^I(m_t^S, l_t^S, h_t^S; \mathscr{A}_t, \mathscr{T}_t)$.[9] In equilibrium, the coupled modules' consistency requires that

$$\mathscr{P}_i^{SI}(\mathscr{A}_t, \mathscr{T}_t) = P_i^I(m_t^S(\mathscr{A}_t, \mathscr{T}_t), l_t^S(\mathscr{A}_t, \mathscr{T}_t), h_t^S(\mathscr{A}_t, \mathscr{T}_t); \mathscr{A}_t, \mathscr{T}_t). \tag{10}$$

The previous equation formalizes the handshake between the epidemiological and economic module, and determines the channel through which agents' behavioral responses affect the course of the pandemic and vice-versa. To connect economic activities with contacts and ultimately infection probabilities, we extend Ash et al. (2022) and Boppart et al. (2024)'s linear random meeting technology to multiple regions to account for travel activities and their corresponding contacts.

The general formulation for the probability of infection given $\mathscr{C}$ contacts and the epidemiological probability τ that a contact with an infectious person leads to infection is

$$P(\text{infection}) = 1 - \left(1 - \tau \cdot \frac{I}{N}\right)^{\mathscr{C}}.$$[10]

We extend this logic to a model with multiple regions and endogenous choices over activities. The probability that a susceptible is infected depends on the share of time the susceptible spend in each activity, the likelihood that a contact during

[9] Because all region i susceptible individuals are ex ante identical, all make the same choices, and the infection probability $P_i^I(m_t^S, l_t^S, h_t^S; \mathscr{A}_t, \mathscr{T}_t)$ is the same for all susceptible individuals in i.

[10] Given n independent draws (contacts), the probability of getting infected is the probability of at least one success, $P(\text{at least one success}) = 1 - P(\text{failure})^n$. The probability that a contact does not lead to infection is $P(\text{failure}) = 1 - P(\text{infection from a contact}) = 1 - P(\text{Infectious Contact}) \cdot P(\text{contact with Infectious leads to infection})$. Thus, the probability that a contact is infectious, $P(\text{Infectious Contact})$, corresponds to the share of the relevant population that belongs to I, while the probability that a contact with an infectious person leads to infection is τ.

that activity leads to infection, and the number of contacts during the activity,

$$P_i^I(m_t^S, l_t^S, h_t^S; \mathscr{A}_t, \mathscr{T}_t) \equiv 1 - \Bigg[\underbrace{\left(\frac{l_t^S}{1-\theta_i}\right)}_{\text{Work share}} \left(1 - \underbrace{\tau \cdot \frac{(I_{i,t} - M_{i,t}^I + M_{j,t}^I)}{N_{i,t}}}_{\text{Home Exposure}} \right)^{\mathscr{C}_{i,t}^l} \tag{11}$$

$$+ \underbrace{\left(\frac{m_t^S}{1-\theta_i}\right)}_{\text{Travel share}} \left(1 - \underbrace{\tau \cdot \frac{(I_{j,t} - M_{j,t}^I + M_{i,t}^I)}{N_{j,t}}}_{\text{Foreign Exposure}} \right)^{\mathscr{C}_{i,t}^m} + \underbrace{\left(\frac{h_t^S}{1-\theta_i}\right)}_{\text{Isolation share}} \left(1 - \underbrace{\tau \cdot \frac{(I_{i,t} - M_{i,t}^I + M_{j,t}^I)}{N_{i,t}}}_{\text{Home Exposure}} \right)^{\mathscr{C}_{i,t}^h} \Bigg].$$

The three infection channels isolate the probability of getting infected per contact while working, isolating, or traveling. Agents allocate their time endowment accounting for the fact that each activity (l_t^S, m_t^S, h_t^S) leads to a differential activity-and-region specific likelihood of not getting infected, denoted by the terms $(\cdot)^{\mathscr{C}^z}$, $z \in \{l, m, h\}$. Crucially agent takes the latter as given, introducing a key behavioral externality into the disease dynamics.

In a metapopulation model, the infectious population share in i depends on the infectious residents of i who leave and travel to the other regions, $M_{i,t}^I = I_{i,t} m_{i,t}^I$, as well as the infectious residents of other regions who travel to region i, $M_{j,t}^I = I_{j,t} m_{j,t}^I$ (recall in equilibrium infectious do not isolate). The total population engaged in economic activities in region i at time t, $N_{i,t}$, includes the residents of i, less those traveling to the other region, plus residents of j traveling into i:

$$N_{i,t} = \sum_{Z \in \{S,I,R\}} Z_{i,t} - M_{i,t}^Z + M_{j,t}^Z, \tag{12}$$

where $M_{i,t}^Z$ are type Z travelers.[11]

The number of contacts an agent has depends on the activities they participate

[11] In Online Appendix E.2, we consider an alternative specification for the infection probability where isolating agents are removed from the relevant local population. We show quantitatively that this leads to higher peak infectious, but otherwise similar dynamics.

in. The contacts of an agent who chooses to work (and consume) are given by

$$\mathscr{C}^l_{i,t} = \underbrace{\rho^C w_t \bar{l}(1-\theta_i) C^*_{i,t}}_{\text{Consumption contacts}} + \underbrace{\rho^L \bar{l}(1-\theta_i) L^*_{i,t}}_{\text{Work contacts}} + \underbrace{\mathscr{C}^u}_{\text{Unavoidable contacts}} . \tag{13}$$

The unavoidable contacts can be thought of primarily as contacts occurring at home, but could also include things such as religious activities. Consumption contacts are determined by the dollar amount spent by the individual for consumption activities, $w_t \bar{l}(1-\theta_i)$, the average dollar value of consumption activities by others in region i, $C^*_{i,t}$, and the parameter ρ^C which converts individual and average consumption into contacts. Likewise, work contacts depend on the number of hours the individual works, $\bar{l}(1-\theta_i)$, the average number of hours others in region i work, $L^*_{i,t}$, and ρ^L which converts individual and average labor into contacts.[12]

The average dollar value of consumption and average number of work hours in i are given by

$$C^*_{i,t} = \sum_{Z \in \{S,I,R\}} \left(\underbrace{c^Z_{i,t} \frac{Z_{i,t} - M^Z_{i,t}}{N_{i,t}}}_{\text{Consumption by } i \text{ agents}} + \underbrace{\kappa \frac{M^Z_{j,t}}{N_{i,t}}}_{\text{Consumption by } j \text{ agents}} \right), \tag{14}$$

$$L^*_{i,t} = \sum_{Z \in \{S,I,R\}} \bar{l} \cdot l^Z_{i,t} \frac{Z_{i,t}}{S_{i,t} + I_{i,t} + R_{i,t}}. \tag{15}$$

$C^*_{i,t}$ is a weighted average of the consumption by those individuals the susceptible agent may encounter in region i (i.e. those in each disease state in i less those that are traveling or isolating) and consumption dollars spent by those traveling from j into i, where κ converts travel shares into dollars.[13] $L^*_{i,t}$ is the weighted average labor hours by agents in region i. We do not adjust $L^*_{i,t}$ for those traveling because this is already captured by the time share spent on work $l^Z_{i,t}$.

When traveling the agent has contacts from consumption activities in the foreign

[12] From the budget and time constraints in (6), if the agent chooses zero travel and isolation $m^S_{i,t} = h^S_{i,t} = 0$, then labor hours are $\bar{l}(1-\theta_i)$ and consumption is $w_t \bar{l}(1-\theta_i)$ as in (13).

[13] The parameter κ allows for the possibility that people spend more on consumption activities when traveling than at home.

region as well as unavoidable contacts,

$$\mathscr{C}^m_{i,t} = \underbrace{\rho^M \bar{l}(1-\theta_i)C^*_{j,t}}_{\text{Travel contacts}} + \underbrace{\mathscr{C}^u}_{\text{Unavoidable contacts}}, \tag{16}$$

where $C^*_{j,t}$ is defined as in (14), and ρ^M converts the dollars-hours product into contacts. Finally, if the susceptible individual chooses to isolate, they have only the unavoidable contacts,

$$\mathscr{C}^h_{i,t} = \mathscr{C}^u. \tag{17}$$

Notice that the choices of individual agents affect the probability of infection both directly through the work, travel, and isolation activity shares in (11), and indirectly through the contacts associated with those activities (12) - (17). Because agents are atomistic, they take contacts $\mathscr{C}^l_{i,t}, \mathscr{C}^m_{i,t}$ and population shares $N_{i,t}$ as given when making their choices. Our solution algorithm in Online Appendix B recovers the individual choices consistent with these aggregates.

3.4 Equilibrium

An equilibrium is defined as a set of individual agents' choices $(c^z_{i,t}, l^z_{i,t}, m^z_{i,t}, h^z_{i,t})$ and associated value functions $U^z_{i,t}$ for all regions i, time t, and agents disease state $z \in \{S, I, R, D\}$, an aggregate wage rate w_t, the time-varying regional disease states of the economy $\mathscr{T}_{i,t} = (S_{i,t}, I_{i,t}, R_{i,t}, D_{i,t})$, such that, in every period, t and region, i:

1. **Individual Choices:** Given the wage, aggregate disease states of the economy $\mathscr{T}_{i,t}$, agents' choices solve (6), (7), and (8);

2. **Firms' Zero-profit Condition:** Labor is paid its marginal product: $w_t = A$;

3. **Market Clearing:** Given the individual choices, aggregate disease states of the economy $\mathscr{T}_{i,t}$, aggregate labor supply equals labor demand:

$$\sum_i \left[l^S_{i,t} S_{i,t} + \phi l^I_{i,t} I_{i,t} + l^R_{i,t} R_{i,t} \right] = L_t$$

4. **Epidemiological Module:** The disease states $\mathscr{T}_t = \{S_{i,t}, I_{i,t}, R_{i,t}, D_{i,t}\}_{i=1}^{\mathscr{I}}$ follow the law of motion described by the system of equations in (1)-(4)

5. **Infection Probabilities' Module:** Given the wage, the individual choices, and the law of motion for the disease states, coupled models' consistency requires that equation (10) is satisfied in every period.

Online Appendix B describes the solution algorithm.

3.5 Equilibrium Characterization: Infection Avoidance and Arbitrage

What are the determinants of travel and isolation decisions during a pandemic? This section analytically characterizes the infection avoidance role of travel and the infection arbitrage opportunity provided by home isolation. To do so, we initially shut down the home isolation channel in Proposition 1, and then later reintroduce it in Proposition 2 and 3. Online Appendix A contains all proofs.

In the pre-pandemic period, agents choose travel by comparing their marginal utility benefits and opportunity costs,

$$\frac{\partial u_i}{\partial m} = \frac{\partial u_i}{\partial c} w_t \bar{l}.$$

The pandemic introduces new considerations in the susceptible agents' travel decisions,

$$\underbrace{\frac{\partial u_i}{\partial m}}_{\text{Direct Benefit}} \underbrace{- \delta (U^S_{i,t+1} - U^I_{i,t+1}) \frac{\partial P^I_i}{\partial m}}_{\text{Pandemic Considerations}} = \underbrace{\frac{\partial u_i}{\partial c} w_t \bar{l}}_{\text{Opportunity Cost of Travel}},$$

which account for the extent by which traveling to a region with different infec-

tiousness levels affects their likelihood of becoming infected,

$$\frac{\partial P_i^I}{\partial m} \equiv \frac{1}{1-\theta_i}\left[\left(1-\underbrace{\tau\cdot\frac{(I_{i,t}-M_{i,t}^I+M_{j,t}^I)}{N_{i,t}}}_{\text{Home Exposure}}\right)^{\mathscr{C}_{i,t}^l} - \left(1-\underbrace{\tau\cdot\frac{(I_{j,t}-M_{j,t}^I+M_{i,t}^I)}{N_{j,t}}}_{\text{Foreign Exposure}}\right)^{\mathscr{C}_{i,t}^m}\right] \quad (18)$$

and the associated discounted payoff of moving from a susceptible to an infectious state in the next period, $\delta(U_{i,t+1}^S - U_{i,t+1}^I)$. Crucially, pandemic considerations are absent among infectious and recovered agents, whose traveling decisions have no bearing on their continuation value.

We focus our analysis on the policy-relevant case where the pandemic is deadly or costly enough that agents do not want to *strategically* contract the virus, $U_{i,t}^S - U_{i,t}^I > 0$. Lemma 1 in Online Appendix A.1 shows that the following condition eliminates these *selfish incentives for herd immunity*, and implies $U_{i,t}^R > U_{i,t}^I$, $U_{i,t}^R \geq U_{i,t}^S$.[14]

Assumption 1. ***No selfish incentives for herd immunity.*** *For all m,t,*

$$\Big(u_i(c^R(m),m) - u_i^I(c^I(m),m)\Big) - \delta(1-P^D)(U_{i,t}^R - \Omega_i) \geq 0,$$

where $c^R(m) \equiv w_t\bar{l}(1-\theta_i-m)$ and $c^I(m) \equiv \phi w_t\bar{l}(1-\theta_i-m)$.

In the presence of endogenous travel choices, traveling acts not only as a pathway for the virus to spread, but also an insurance mechanism by which susceptible people may avoid infection. When the case rate is high (low) in their home region susceptibles have an additional incentive (disincentive) to travel. This mechanism is absent in models with exogenous travel choices. Proposition 1 formalizes the conditions under which travel provide an infection avoidance asset or liability.

Proposition 1. ***The Infection Avoidance Role of Travel.*** *Under Assumption 1 and absent home isolation, $h = 0$, endogenous travel choices provide susceptibles with:*

[14] This condition is intuitively satisfied when the infectious state productivity loss is large enough (ϕ small) or the probability of death due to the virus, P^D is large enough.

(a) ***an infection avoidance asset*** *if the likelihood of contracting the virus in the foreign region while traveling is lower than the likelihood of contracting the virus in the home region while working,* $\frac{\partial P_i^I}{\partial m} < 0$. *In this case, susceptible agents travel strictly more than if they were recovered. In the absence of direct benefits from travel, susceptible travel weakly more than if they were recovered.*[15]

(b) ***an infection avoidance liability*** *if the likelihood of contracting the virus in the foreign region while traveling is higher than the likelihood of contracting the virus in the home region while working,* $\frac{\partial P_i^I}{\partial m} > 0$. *In this case, susceptible agents travel less than if they were recovered. Absent direct benefits from travel, susceptible choose not to travel.*

Next, consider the case where susceptibles have access to an additional infection avoidance asset: isolation at home, $h^S > 0$. Home isolation yields lower contact rates than travel ($\mathscr{C}^h \leq \mathscr{C}^m$) but does not offer direct utility benefits. Crucially, the presence of both traveling and home isolation provide susceptibles with an infection avoidance arbitrage opportunity, which we formalize in Proposition 2.

Proposition 2. ***Avoidance Channels and Infection Arbitrage.*** ***(a)*** *Under Assumption 1, in the absence of utility benefits from travel,* $u(c,m) = u(c)$, *susceptibles will choose the infection avoidance asset (travel or home isolation) that yields the greater reduction in the likelihood of infection. Susceptibles weakly prefer travel if home isolation is less effective in reducing the exposure risk,*

$$\left(1 - \underbrace{\tau \cdot \frac{(I_{i,t} - M_{i,t}^I + M_{j,t}^I)}{N_{i,t}}}_{\text{Home Exposure}}\right)^{\mathscr{C}_{i,t}^h} - \left(1 - \underbrace{\tau \cdot \frac{(I_{j,t} - M_{j,t}^I + M_{i,t}^I)}{N_{j,t}}}_{\text{Foreign Exposure}}\right)^{\mathscr{C}_{i,t}^m} \leq 0. \quad (19)$$

Effectiveness of home isolation in reducing infection exposure $(+/-)$

[15] Distinct from Antràs, Redding, and Rossi-Hansberg (2023), in our metapopulation model, the avoidance role of travel emerges even in the absence of direct benefits from travel, which in their model arise due to gains from trade.

This is true when

$$\frac{\partial P_i^I}{\partial m} \leq \frac{\partial P_i^I}{\partial h} = \frac{1}{1-\theta_i} \underbrace{\left[\left(1 - \tau \cdot \underbrace{\frac{(I_{i,t} - M_{i,t}^I + M_{j,t}^I)}{N_{i,t}}}_{\textit{Home Exposure}} \right)^{\mathscr{C}_{i,t}^I} - \left(1 - \tau \cdot \underbrace{\frac{(I_{i,t} - M_{i,t}^I + M_{j,t}^I)}{N_{i,t}}}_{\textit{Home Exposure}} \right)^{\mathscr{C}_{i,t}^h} \right]}_{\textit{Effectiveness of home isolation in reducing home-infection exposure } (-)} \leq 0,$$

which depends on the number of contacts associated with each activity given by equations (16) *and* (17)*, as well as the share of infectious in the home and foreign populations. Conversely, susceptibles weakly prefer home isolation if the previous inequality is flipped* ($\geq$)*.* ***(b)*** *Under Assumption 1, independent of whether agents receive direct utility from travel, susceptibles will not isolate* ($h = 0$) *if infection rates in the home region are sufficiently low that isolation's opportunity cost (in terms of consumption) is too high. Susceptibles will also not isolate and instead travel for avoidance purposes if these conditions hold: (i) travel is more effective than home isolation in reducing the likelihood of infection or home and foreign infection rates are sufficiently small; (ii) the marginal utility benefits of travel are sufficiently large,* $\partial u / \partial m$*; and (iii) travel is either an interior solution or the shadow value on its upper-bound constraint is not too large that isolating may become attractive.*

Next, we use assumptions on the functional form of the utility function adopted in our quantitative analysis to provide closed-form solutions of traveling choices.

Proposition 3. ***Susceptible Travel Choices.*** *Let* $u_i(c,m) = (1-\beta_i)\ln(c) + \beta_i \ln(m)$*, then pre-pandemic agents, infectious and recovered types choose a region-specific constant share of time traveling*

$$m_{i,t}^{pre} = \beta_i, \qquad m_{i,t}^I = \beta_i^I (1-\theta_i), \qquad m_{i,t}^R = \beta_i (1-\theta_i). \tag{20}$$

Under Assumption 1, if susceptibles do not choose to isolate ($h = 0$)*—or absent isolation options—they travel more than recovered individuals when traveling re-*

duces the likelihood of becoming infectious ($\partial P^I_{i,t}/\partial m^S_{i,t} < 0$) and less otherwise,[16]

$$\frac{m^S_{i,t}}{m^R_{i,t}} = 1 + \begin{cases} \underbrace{\frac{1-2\beta_i}{2\beta_i} + \frac{1}{2\beta_i\tilde{B}}\left(1+\sqrt{1+\tilde{B}(\tilde{B}+2(1-2\beta_i)}\right)}_{\geq 0}, & \frac{\partial P^I_{i,t}}{\partial m^S_{i,t}} < 0, \quad \textit{Inf. Avoid. Asset} \\ \underbrace{\frac{1-2\beta_i}{2\beta_i} + \frac{1}{2\beta_i\tilde{B}}\left(1-\sqrt{1+\tilde{B}(\tilde{B}+2(1-2\beta_i)}\right)}_{\leq 0}, & \frac{\partial P^I_{i,t}}{\partial m^S_{i,t}} > 0, \quad \textit{Inf. Avoid. Liability} \end{cases} \tag{21}$$

where $\tilde{B} \equiv (1-\theta_i)\delta(U^S_{i,t+1} - U^I_{i,t+1})\partial P^I_{i,t}/\partial m$. If susceptibles isolate, $h > 0$, this expression becomes

$$\frac{m^S_{i,t}}{m^R_{i,t}} = \frac{1}{\delta(U^S_{i,t+1} - U^I_{i,t+1})\left[\underbrace{\left(1-\tau\cdot\frac{(I_{i,t}-M^I_{i,t}+M^I_{j,t})}{N_{i,t}}\right)^{\mathscr{C}^h_{i,t}} - \left(1-\tau\cdot\frac{(I_{j,t}-M^I_{j,t}+M^I_{i,t})}{N_{j,t}}\right)^{\mathscr{C}^m_{i,t}}}_{\textit{Effectiveness of home isolation in reducing infection exposure}(+,h>0)}\right]} \tag{22}$$

and susceptibles travel even less, relative to recovered individuals, when home isolation is increasingly effective at reducing the likelihood of becoming infected.

This section isolates the key considerations driving traveling choices during a pandemic, specifically, regional differences in relative contact rates and infectious population shares. Section 5 explores the quantitative relevance of these key trade-offs over the evolution of a pandemic.

4 Model Calibration

We calibrate the model to match features of the U.S. economy prior to the COVID-19 pandemic. We examine a two region version of the model, $i \in \{home, foreign\}$. A period in the model corresponds to one day and the pandemic lasts two years, $T = 730$, at which point a vaccine arrives and the pandemic ends with certainty.[17]

[16] Susceptibles do not isolate, $h = 0$, if $\hat{m}^S_{i,t} > \tilde{m}^S_{i,t}$ where $\hat{m}^S_{i,t}$ satisfies (21) and $\tilde{m}^S_{i,t}$ satisfies (22).

[17] In our exploration of the parameter space in Section 6, across over one million parameter combinations, the pandemic ends (infectious population less than one per million) after an average of 395 days.

Table 1 summarizes the parameter values used in our benchmark analysis.[18]

4.1 Preferences

We discipline the preferences of agents to capture pre-pandemic (long-run) traveling and consumption decisions. We follow Prescott (1986) and assume that agents have preferences

$$u_i(c,m) = \frac{\left(c^{1-\beta_i} \cdot m^{\beta_i}\right)^{1-\sigma} - 1}{1-\sigma} \qquad i \in \{home, foreign\}$$

over a composite good $c^{1-\beta_i} \cdot m^{\beta_i}$, where $\beta_i > 0$ is the travel share parameter, and $1/\sigma > 0$ is the elasticity of intertemporal substitution. As income and substitution effects tend to compensate each other in the long-run (e.g. Prescott, 1986, Kimball and Shapiro, 2008), we focus our quantitative analysis on the case where $\sigma \to 1$,

$$u_i(c,m) = (1-\beta_i)\ln(c) + \beta_i \ln(m) \qquad i \in \{home, foreign\}$$

The log-utility proves useful for two reasons. First, it provides unique closed-form solutions to the susceptible agent's problem, dispensing with the need for root-finding procedures and enhancing the interpretability of our quantitative results.[19] Second, it allows us to cleanly identify the travel preference parameters $\{\beta_{home}, \beta_{foreign}\}$ to match the pre-pandemic share of time residents travel. In particular, under this utility specification, agents display a constant share of time traveling, $m^{pre}_{i,t} = \beta_i$, with $i \in \{home, foreign\}$. We calculate these time shares with information from the Bureau of Transportation Statistics American Travel Survey (ATS). Using ATS, we calculate the share of time spent traveling from the aver-

[18] The values for pre-pandemic contacts during consumption $\mathscr{C}^{c^*}$ and travel $\mathscr{C}^{m^*}$, and the consumption and travel contacts conversion factors ρ^C, ρ^M, depend on the relative population of the home region (see Section 4.2). For these, Table 1 reports values for both symmetric ($\lambda_{home} = 0.5$) and asymmetric ($\lambda_{home} = 0.00001$) regions.

[19] This assumption eliminates the dependence of travel choices on exogenously fixed aspects of our economic environment, such as the opportunity costs of travel $(\phi, \bar{l})$ and the wage, which is set to A in equilibrium. Since our analysis abstracts from targeted labor market interventions (such as the Paycheck Protection Program in the U.S.) or the long-run effect of a pandemic on productivity, this assumption is not overly restrictive for our purposes.

age (across households) number of days spent on travel of at least 100 miles per year. Crucially, the prediction of a constant share of time traveling is consistent with recent evidence suggesting the absence of trends in traveling choices.[20]

Finally, to account for potential health effects of the disease on preferences for traveling activities, we allow infectious agents to receive different (lower) utility from travels, $\beta_i^I < \beta_i$. In particular, we choose β_i^I so that the time share spent traveling by infectious relative to that of recovered is adjusted by the share of infectious with severe symptoms, $m_i^I = (1-\phi^{severe})m_i^R$, where ϕ^{severe} is the share of infectious with severe symptoms.[21]

4.2 Contacts

We follow Ash et al. (2022) to calculate the parameters ρ^C, ρ^L, ρ^m which convert economic activities into contacts. Ash et al. (2022) use contact data from Prem, Cook, and Jit (2017) to calculate average daily contacts by activity. They separate contacts into those occurring during labor activities $\mathscr{C}^{l^*}$, contacts from home activities, which we assign to unavoidable contacts $\mathscr{C}^u$, and contacts from other activities, which we assign to consumption $\mathscr{C}^{c^*}$ and travel $\mathscr{C}^{m^*}$. We choose the contact parameters ρ^C, ρ^L, ρ^m so that average pre-pandemic contacts match these values,

$$\sum_i \lambda_i \rho^L \bar{l} l_i^{pre} L_i^{pre^*} = \mathscr{C}^{l^*},$$
$$\sum_i \lambda_i \left(\rho^C c_i^{pre} C_i^{pre^*} + \rho^M \bar{l} m_i^{pre} C_j^{pre^*} \right) = \mathscr{C}^{c^*} + \mathscr{C}^{m^*},$$

[20] An annual survey from Expedia shows that travel behavior has remained relatively constant over time at ~ 11 (business) days per year. *Source:* Expedia - 2007, Expedia - 2024.

[21] Ash et al. (2022) calculate the share of infectious with severe symptoms ϕ^{severe} as a duration-weighted average of time spent in different states of infection severity, with major sufferers being those who become hospitalized weighted by their time spent symptomatic, giving a value of $\phi^{severe} = 0.1445$.

Table 1: Parameter Values

Parameter	Value	Description	Source
Epidemiological Parameters			
R_0	2.6	Initial reproduction number	Ash et al. (2022)
d	5.1	Duration of infectiousness	Ash et al. (2022)
μ	0.015	Case fatality rate	Ash et al. (2022)
$\mathscr{C}^{c^*}$	[3.984, 4.546]	Pre-pandemic contacts during consumption	Described in Section 4.2
$\mathscr{C}^{m^*}$	[1.183, 0.621]	Pre-pandemic contacts during travel	Described in Section 4.2
$\mathscr{C}^{l^*}$	7.513	Pre-pandemic contacts during labor	Prem, Cook, and Jit (2017)
$\mathscr{C}^{u}$	3.549	Unavoidable contacts	Prem, Cook, and Jit (2017)
τ	0.033	Infections per contact	Described in Section 4.3
P^R	0.193	Daily probability of recovery	$=(1/d)(1-\mu)$
P^D	0.003	Daily probability of death	$=(1/d)\mu$
P^{D^*}	3.6e-5	Daily probability of death (outside cause)	$=1/(76*365)$
γ	3.6e-5	Population growth rate	Set $\gamma = P^{D^*}$
Economic Parameters			
δ	$0.96^{1/365}$	Daily discount factor	Implied risk-free interest rate of $\sim$0.04
ϕ	0.8555	Share minor sufferers	Ash et al. (2022)
β_i	0.063	Travel share	Described in Section 4.1
β_i^I	0.054	Infectious travel share	Described in Section 4.1
$\bar{h}$	0.7	Share essential workers	Iverson, Karp, and Peri (2022)
κ	345.58	Daily travel cost	Described in Section 4.2
Ω_i	-33,420	Value for deceased	Described in Section 4.4
A	19.86	Technology	Implies pre-pandemic GDP per capita of $58,000
$\bar{l}$	8.54	Time endowment	Implies pre-pandemic individual labor supply = 8 hours
λ_{home}	[1e-5, 0.5]	Home population share	Set externally
ρ^C	[1.6e-4, 1.7e-4]	Consumption contacts conversion factor	Described in Section 4.2
ρ^M	[0.006, 0.007]	Travel contacts conversion factor	Described in Section 4.2
ρ^L	0.117	Labor contacts conversion factor	Described in Section 4.2

Note: For parameters in brackets, the first value corresponds to the case with asymmetric regions, and the second to symmetric regions.

where λ_i are regional population shares and

$$C_i^{pre^*} = c_i^{pre} \frac{\lambda_i(1-m_i^{pre})}{\lambda_i(1-m_i^{pre})+\lambda_j m_j^{pre}} + \kappa \frac{\lambda_j m_j^{pre}}{\lambda_i(1-m_i^{pre})+\lambda_j m_j^{pre}},$$
$$L_i^{pre^*} = \bar{l} l_i^{pre},$$

are the pre-pandemic average consumption and labor activities in region i.[22] We calculate the parameter κ, which converts time spent on travel into dollars using data from Business Travel News (BTN) and the Federal Highway Administration Next-Generation National Household Travel Survey (NHTS). We weight the total daily cost for travel to a given city in the BTN data by the annual total trips to the city in the NHTS data to arrive at an average daily cost of travel.

4.3 Transmissibility of the Virus

We calculate the transmissibility of the virus τ by backing it out from the formula for the reproductive number R_0. R_0 denotes the expected number of new cases caused by a single case in a population of entirely susceptibles. R_0 is given by

$$R_0 = \tau \mathscr{C}^* d,$$

where $\tau \in [0,1]$ is infections per contact, $\mathscr{C}^*$ is the number of contacts between the infectious person and susceptibles per day, and d is the number of days a person is infectious. For our two region model, we calculate contacts $\mathscr{C}^*$ as a weighted average of contacts across regions,

$$\mathscr{C}^* = \sum_{i \in \{home, foreign\}} \lambda_i \left(\rho^C c_i^I C_i^{pre^*} + \rho^L l_i^I \bar{l} L_i^{pre^*} + \rho^M m_i^I \bar{l} C_j^{pre^*} + \mathscr{C}^u \right), \quad (23)$$

[22] We compute the model-implied values for $\mathscr{C}^{c^*}$ and $\mathscr{C}^{m^*}$ individually as $\mathscr{C}^{c^*} = \sum_i \lambda_i \rho^C c_i^{pre} C_i^{pre^*}$ and $\mathscr{C}^{m^*} = \sum_i \lambda_i \rho^M \bar{l} m_i^{pre} C_j^{pre^*}$. These values are reported in Table 1.

with $j \neq i$.[23] Given epidemiological estimates of R_0 and d we can compute the transmissibility τ implied by $R_0 = \tau \mathscr{C}^* d$.

4.4 Value of Being Deceased

We calibrate the value of deceased Ω_i to be consistent with a value of a statistical life (VSL) of \$10 million.[24] The VSL provides a measure of how much individuals are willing to give up in order to reduce the likelihood of death. That is, for an increase in the probability of dying of ΔP^{D^*}, agents require $VSL \cdot \Delta P^{D^*}$ additional wealth. Alternatively, for an increase in wealth of Δa, individuals will accept an increase in the probability of death of $\Delta a / VSL$. Thus, we can find the implied Ω_i from the following equality:

$$U_{i,t}^{pre} = U_{i,t}^{VSL},$$

where $U_{i,t}^{pre}$ is pre-pandemic value given in (5), and $U_{i,t}^{VSL}$ is given by the solution to

$$U_{i,t}^{VSL} = \max_{\{c_t^{VSL}, l_t^{VSL}, m_t^{VSL}\}} \{u(c_t^{VSL}, m_t^{VSL}) + \delta((1 - P^{D^*} - \frac{\Delta a}{VSL}) U_{i,t+1}^{pre} + (P^{D^*} + \frac{\Delta a}{VSL}) U_{i,t+1}^{D})\},$$
$$\text{s.t. } c_t^{VSL} = w_t \bar{l} l_t^{VSL} + \Delta a, \qquad m_t^{VSL} + l_t^{VSL} = 1.$$

We set Δa to a 10% increase in daily wage, $\Delta a = 0.1 \bar{l} w$.

4.5 Initial Conditions of the Pandemic

The model requires an initial infectious seed to start the pandemic. Unless otherwise specified, we set the population of infectious at time zero to $I_{0,i} = 0.0001\%$ in both regions $i = \{H, F\}$.

[23] When travel between regions is not permitted, $m_i^I = 0$, $c_i^I = \phi w \bar{l}$, $C_i^{pre^*} = w\bar{l}$, and $L_i^{pre^*} = \bar{l}$, which implies $\mathscr{C}^* = \sum_i \lambda_i \left(\rho^C \phi (w\bar{l})^2 + \rho^L \bar{l}^2 + \mathscr{C}^u \right)$, analogous to the single region contact model in Ash et al. (2022).

[24] Setting the value associated with death $\Omega_i = 0$, as is sometimes done in the literature, can lead to unintended individual behavior. As an illustrative example, take the case of CRRA preferences $u(c) = c^{1-\sigma}/(1-\sigma)$. For $\sigma > 1$, discounted lifetime utility is < 0 implying death would *raise* lifetime utility, contrary to how we typically think people view dying.

5 Quantitative Analysis

This section examines the quantitative relevance of travel choices and disease containment strategies in the context of pandemic federalism. Section 5.1 studies the determinants of endogenous travel and isolation choices, while Section 5.2 analyzes the effect of behavioral responses on disease dynamics, both in a laissez-faire environment. Section 5.3 examines pandemic federalism, the role of travel restrictions, and endogenous policy under a non-cooperative (Nash) game between two local policymakers. Additional results are provided in Online Appendix C.

5.1 Endogenous Travel and Isolation Choices

When is travel an asset or a liability for infection avoidance purposes? When do agents prefer travel to isolation? How do these incentives change over the evolution of a pandemic? We build on the theoretical results in Section 3.5 to explore these quantitative questions. We examine two economies, one where regions are identical in every aspect (symmetric economy), and another where the home-region is relatively small (asymmetric economy). The symmetric case is more relevant when considering federal policies and travel across roughly similar-sized countries (e.g. Germany and France), while the second case is more relevant for local policies when there is travel between very heterogeneous countries (e.g. U.S. and Cayman Islands) with different potentials to shape the course of the pandemic, to which we will return in Section 5.3.

We begin by analyzing the endogenous travel decisions of susceptibles within a symmetric economy. This is illustrated in Figure 1. In this environment, home- and foreign- infection exposures are identical (Panel C) along the equilibrium path. Accordingly, the relative contact rates across activities are the key determinants of travel and isolation choices. Panel B shows that contact rates of traveling are lower than working, $\partial P^I_{i,t}/\partial m^S_{i,t} < 0$; therefore, travel is an infection avoidance asset over the course of the entire pandemic (Proposition 1). However, the relative effectiveness of travel and home isolation as infectious avoidance channels varies over the evolution of the pandemic.

Proposition 2 states that when the likelihood of infection in the home region

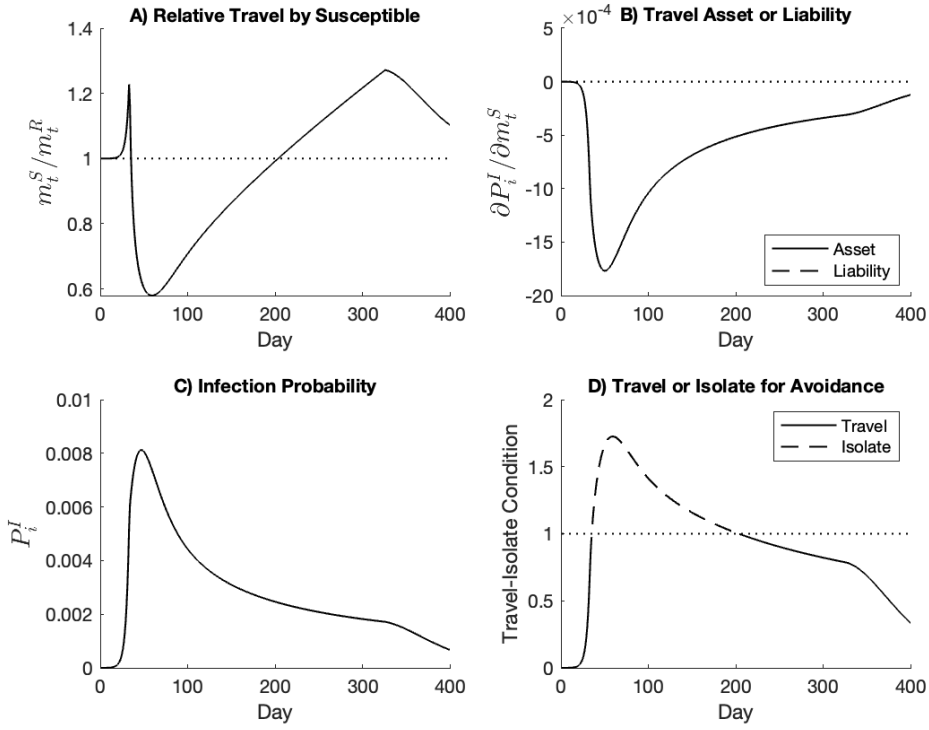

Figure 1: Determinants of travel choice with symmetric regions.

is low, isolation is not an effective avoidance tool. Consistent with this insight, Panel D shows that at the beginning and end of the pandemic, agents do not isolate, and prefer to travel for infectious avoidance purposes.[25] Finally, Panel A shows that susceptible agents travel more than recovered agents when traveling is a more effective infectious avoidance mechanism than isolation (Proposition 3), which is particularly true when infection rates are low (Proposition 2), at the beginning and ending of the pandemic.[26]

Next, in Figure 2 we study the travel and isolation choices of susceptibles when the home and foreign regions are asymmetric in population shares (home is small,

[25]The travel-isolate condition corresponds to the reciprocal of (22). When this quantity is smaller than one and $\partial P_{i,t}^I/\partial m_{i,t}^S < 0$, travel is an effective asset and susceptible agents increase travel relative to recovered.

[26]The sharp spike in travel as the initial infectious share rises is consistent with observed outflows of people from early hot spots such as Wuhan (Kraemer et al., 2020) and New York City (Coven, Gupta, and Yao, 2023) during the COVID-19 pandemic.

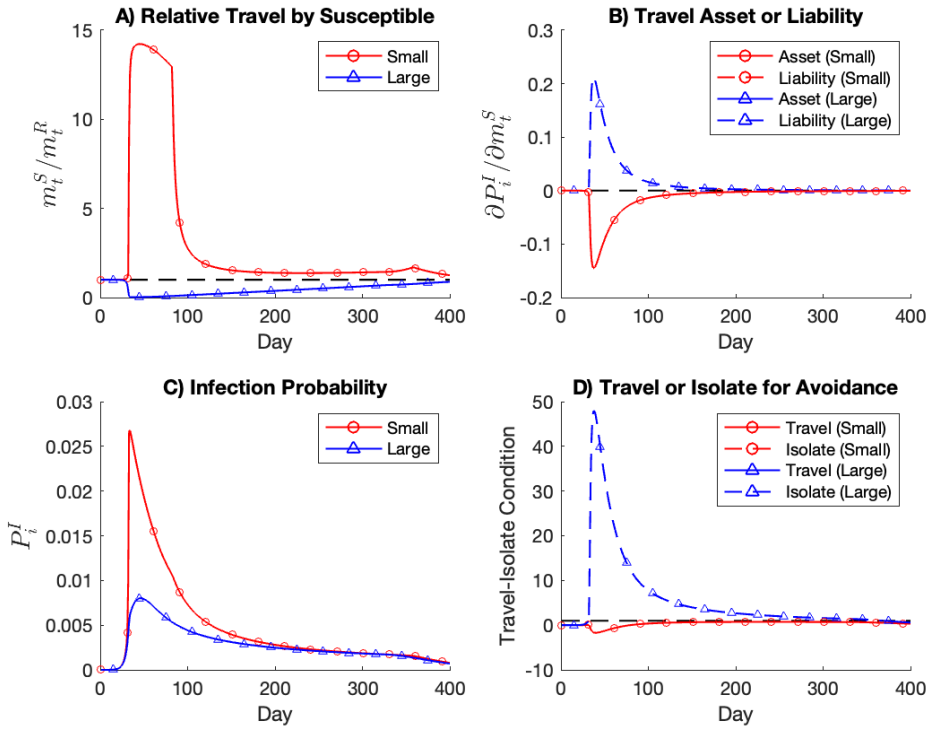

Figure 2: Determinants of travel choice with asymmetric regions.

foreign is large).[27] The role of travel as an asset or liability depends on whether the agent resides in the small or the large region. In particular, and in contrast to the symmetric case, travel becomes a liability ($\partial P_{i,t}^I/\partial m_{i,t}^S > 0$) for the agents in the large region when infection rates are high, shown in Panel B . Hence Panel A shows agents reduce their travel relative to the recovered (Proposition 1) beginning in the peak infection period and continuing throughout the pandemic. On the other hand, travel is an asset for agents in the small region even during the peak. Furthermore, travel remains an *effective* asset (Proposition 3), as shown in Panel D, such that susceptibles in the small region favor travel over isolation for avoidance over the entirety of the pandemic, unlike the symmetric case where agents switch to isolation during the peak. However, despite travel being a relatively more attractive

[27] We set the home population share to 0.001% to illustrate a small nation facing travelers from the rest of the world.

avoidance mechanism than isolation, susceptibles in the small region are unable to meaningfully reduce their infection probability, as the evolution of the pandemic in *both* regions is driven by the behavior of agents in the large region. This illustrates one of our main quantitative results: As the relative size between the two economies increases, the effectiveness of travel as an avoidance mechanism (for susceptibles in both regions) drops.[28]

To sum up, in the symmetric environment, susceptibles prefer travel to isolation as an infectious avoidance tool for a significant (77%) portion of a two-year pandemic, especially at the beginning and the end, when isolation is very costly relative to the benefits due to low infection rates. By contrast, in the asymmetric environment, agents in the small region use travel and those in the large region use isolation for avoidance throughout the pandemic. Notably, the role of travel as an infection avoidance asset or liability evolves over time and is driven by both relative contact rates and the differential of exposure at home and abroad. Finally, travel for avoidance becomes less relevant as the size disparity between regions grows.

5.2 Disease Dynamics and Behavioral Responses

How do behavioral responses affect the disease dynamics in a metapopulation framework? In our metapopulation model, travel works as a pathway for the virus to spread. Here, we show how exogenous and endogenous travel choices affect these disease dynamics, accounting for the fact susceptibles can also isolate. Numerical results for all model comparisons are reported in Table 2.

To assess the role of home isolation, we first contrast an economy where agents cannot travel and isolate (first row),[29] with one where agents cannot travel but are able to isolate (second row).[30] Home isolation is an effective avoidance strategy,

[28] For completeness, Online Appendix C.1 replicates this analysis under the assumption that susceptibles do not receive utility benefits from travel. Here, susceptibles travel only if the foreign-exposure is lower than the home-exposure (Proposition 1). Accordingly, in absence of any asymmetry between regions, agents do not travel.

[29] The model with no isolation and traveling coincides with an SIRD model where the share of susceptibles becoming infected, $\mathscr{P}_i^{SI}(\mathscr{A}_t, \mathscr{T}_t)$ is consistent with the probability of getting infected implied by agents spending their entire time working in order to consume (equation (10)).

[30] The no behavioral response and home isolation autarky outcomes are reported for the case of symmetric regions. When regions are asymmetric, these outcomes change due to the updated

reducing peak infectious from more than 25% down to 3.51%, close to that under our baseline symmetric economy (3.58%).

Table 2: Peak Infectious and Days Between Peaks Across Different Model Specifications

	Peak Infectious - Home (%)		Days Between Peaks	
	$I_{0,H} = I_{0,F}$	$I_{0,H} < I_{0,F}$	$I_{0,H} = I_{0,F}$	$I_{0,H} < I_{0,F}$
Autarky				
No Behavioral Response	26.52	26.52	0	24
Home Isolation	**3.51**	3.51	0	**24**
Symmetric Size				
Exogenous Travel	3.68	3.71	0	**2**
Endogenous Travel	3.58	3.56	0	**5**
Asymmetric Size				
Exogenous Travel	**4.17**	4.18	2	**2**
Endogenous Travel	**9.44**	9.48	9	**9**

Note: No Behavioral Response model has no travel or endogenous avoidance. Home Isolation has no travel, but allows positive home isolation. Exogenous Travel enforces travel at the pre-pandemic rate, with endogenous home isolation response. Endogenous Travel allows both travel and home isolation choices to respond endogenously. The table reports the peak home infectious as percent of the home population and the number of days between the home and foreign peaks. Outcomes are shown for the case with home and foreign infectious seeds set to 0.0001% ($I_{0,H} = I_{0,F}$) and for the home seed set to 0.0001% and the foreign seed set to 0.1% ($I_{0,H} < I_{0,F}$).

Next, to understand the connectivity role of travel, we contrast the home isolation autarky model with four alternative specifications: models with either symmetric or asymmetric region sizes under exogenous or endogenous travel choices (the models with endogenous choices coincide with those presented in Section 5.1).[31] Under exogenous travel, the share of time spent traveling during the pandemic is set to its pre-pandemic period level, $m^{pre} = m^R = m^I = m^S$. This specification isolates the role of travel as a vector of pandemic transmission, by abstracting from

transmissibility parameter τ (Section 4.3), though the differences are not economically significant. We report additional results in Online Appendix C.2.1.

[31] We assume under travel autarky that agents do not receive utility benefits from travel, $\beta_i = 0$. For comparison purposes, we also assume that agents do not receive utility from travel under exogenous travel.

endogenous pandemic considerations of susceptibles.

Travel synchronizes disease dynamics across regions relative to home isolation autarky, reducing the time between peak infections by three weeks under exogenous travel, and slightly less (two weeks) under endogenous travel, independently of relative region size. Accordingly, modeling travel as exogenous performs reasonably well in capturing its impact on the synchronization of disease dynamics under our benchmark calibration, a point we will revisit in our robustness analysis.

Although relative regional sizes do not affect the synchronicity of disease dynamics, they do affect the severity of the pandemic. When regions are symmetric in size, travel has little to no impact on the height of peak infectious relative to the home isolation autarky. In contrast, when regions are asymmetric, travel exacerbates the severity of the pandemic in the small region, particularly under endogenous travel, where peak infectious experience a *threefold* increase relative to home isolation autarky. This is due to the fact that, in the asymmetric case, the disease dynamics in the small region are dominated by those in the large region. While the time to peak infectious in the large region shifts minimally (by only 2 days) compared to autarky, inflows of infectious from the large region induce an earlier peak in the small region, occurring 24 (33) days earlier under exogenous (endogenous) travel. Notably, these dynamics reverse the effect of a reduction in the infectious seed of the small region that occurs under home isolation autarky, accelerating rather than delaying the peak of the pandemic in the small region (Appendix Figure 7). Taken together, these results indicate potential benefits for small regions to restrict incoming travel (smaller and delayed onset of peak infectious), to which we turn in the next subsection.

To sum up: travel matters. When regions are similar in size, travel synchronizes peaks across regions. When regions differ in size, endogenous travel leads to severe outcomes for the small region, for which disease dynamics are dictated by their larger neighbors. Clearly, these analyses hinge on initial assumptions over many important drivers of disease dynamics in a metapopulation model (e.g the reproductive number). Our parameter space exploration in Section 6, will check the robustness of these results across the parameter space.

5.3 Public Policy under Pandemic Federalism

What are the welfare consequences of travel bans and social distancing? In a pandemic federalism setting, local policies not only affect the strategic decisions of policymakers in other regions, but they are also influenced by them. We analyze the Nash equilibrium in a game between policymakers in the home and foreign regions who choose a fixed public intervention to begin at time $t \geq 1$. Given the relevance to our research question and space constraints, we focus our discussion on travel restrictions, a widely adopted yet less understood public intervention policy. Additional results on the Nash equilibria for social distancing policies, as well as outcomes under exogenous policy, are provided in Online Appendix C.3.

Consider a local policymaker who chooses fixed incoming and outgoing travel restrictions $x_i \equiv \{\bar{m}_i^{in}, \bar{m}_i^{out}\}$ to maximize the welfare in its own region i

$$\max_{x_i \in [0,1)\times[0,1)} \sum_{Z_{i,1} \in \{S_{i,1}, I_{i,1}, R_{i,1}, D_{i,1}\}} \frac{Z_{i,1}}{\lambda_i} U_{i,1}^{Z}(x_i, x_j)$$

taking as given the other planner's policies, $x_j \equiv \{\bar{m}_j^{in}, \bar{m}_j^{out}\}$, and subject to the agents' optimization problems (6)-(9) and the transition equations (1)-(4).

Note that the region i policymaker can choose both a policy that restricts their own population $\bar{m}_i^{out}$, as well as one which restricts the j population $\bar{m}_i^{in}$. We restrict the feasible set of policies to 25%, 50%, 75%, or 99% reductions relative to pre-pandemic travel, or the planner can impose no restriction.[32] Online Appendix C.3.4 provides additional details about the local policymaker's problem.

We study the Nash equilibria of the travel restriction game under two environments, one in which regions are symmetric and another in which the home region is small and the foreign region is large.[33] Table 3 summarizes the Nash equilibrium

[32] We prefer a 99% over a 100% reduction due to the log-utility specification, which implies welfare is not defined in an economy with no travel. This assumption is also reasonable, as some leakage between regions is likely to occur when travel is fully banned. Also note that we implicitly assume full commitment on travel restrictions across time from policymakers, which is restrictive but reasonable given the relatively short-horizon of pandemics and the COVID-19 pandemic experience.

[33] Initial conditions for the two regions are identical for all results presented in this section. In Online Appendix C.3.4, we show results are similar when the foreign region begins with a higher infectious share than the home region.

Table 3: Nash Outcomes under Alternative Travel Policies

	Peak Infectious (%)		Welfare	
	Fixed	Infections-Guided	Fixed	Infections-Guided
Symmetric	3.58	[3.43, 3.58]	-23.30	[-23.30, -3.34]
Asymmetric Small	4.21	[4.08, 4.21]	-4.84	[-4.84, 4.85]
Asymmetric Large	3.52	[3.45, 3.52]	-21.30	[-21.30, -5.35]

Note: Peak Infectious is a percent of the region population. Welfare is the percent change from the economy with no travel restrictions. Under a Fixed policy rule, the Nash equilibrium outcome is unique for both symmetric and asymmetric regions. Under an Infections-Guided rule, there are multiple Nash equilibrium outcomes that fall within the range reported in brackets.

outcomes in each environment, considering both a benchmark where the travel bans are fixed in place for the entire pandemic and the case for which bans only become active when the infectious population share passes a threshold.

For both symmetric and asymmetric regions, there is a unique Nash equilibrium outcome under the fixed benchmark timing, where policymakers impose a 99% reduction in travel for both regions. It is always the best response of local policymakers to shut down incoming travel, leading to dynamics similar to those under the home isolation autarky specification presented in Section 5.2.

In the symmetric case, welfare losses relative to the benchmark with no travel restrictions are large, with a -23.30% change in welfare. These losses accrue due to both the direct loss in utility (agents like to travel) as well as the inability to utilize travel for avoidance (Proposition 1). Further, the symmetric environment implies both regions see identical welfare losses, and both could improve welfare by coordinating to achieve the no-restriction benchmark, similar to the textbook Prisoner's Dilemma.

Turning to the case where regions are asymmetric in size, the Nash equilibrium outcome once again leads to a Prisoner's Dilemma. However, unlike the symmetric

case, welfare losses are not shared equally between the two regions. While the large region sees welfare losses similar to that under symmetric economies, the small region experiences much smaller losses. In the small region, Nash travel restrictions still reduce the utility benefits of traveling, but this loss is partially offset by beneficial reductions in peak infectious, down to 4.21% from 9.44% under the model with no travel restrictions (Table 2).[34]

We next study how the timing of policy interventions affect welfare outcomes in the Nash game. To do so, we allow for a policy decision rule under which travel bans are in place when the global infectious population share is above a threshold value ("Infections-Guided"). Each policymaker commits to both travel restrictions (inbound and outbound) and the threshold at which restrictions become active, choosing a triple $x_i = \{\bar{m}_i^{in}, \bar{m}_i^{out}, I_i^{\text{thresh.}}\}$.[35] This enables policymakers to choose strategies for which travel restrictions do not bind during the beginning and end of the pandemic, when travel is most effective (Section 5.1).

When policymakers use infections to guide the imposition of travel bans, welfare losses are significantly reduced compared to those under a fixed policy rule. For symmetric economies, the equilibrium which maximizes welfare sees policymakers wait to enforce bans until the pandemic is well underway–each chooses a threshold value of 1%–leading to a 20 percentage point improvement in welfare from the fixed policy Nash equilibrium. Likewise for asymmetric regions, delayed policy can improve welfare in both regions. In fact, the small region can see welfare gains (relative to the case without policy restrictions) up to 4.85%. This is despite the equilibrium thresholds for asymmetric regions being quite low: in the welfare-maximizing equilibrium, travel bans are in place in the small region when the infectious population share is above 0.25% and in the large region at 0.5%.

We note that allowing policymakers to choose when to impose travel bans may still result in undesirable Nash equilibria. For symmetric and asymmetric regions, the lowest-welfare equilibrium features full travel restrictions in place for the entire pandemic, equivalent to the fixed-policy economies.[36] Thus, coordination mecha-

[34] In Online Appendix C.3.3, we exogenously vary policies to identify those that maximize aggregate welfare.

[35] Policymakers choose a threshold from the set $I^{\text{thresh.}} \in X^{\text{thresh.}} \equiv \{0\%, 0.25\%, 0.5\%, ..., 3\%\}$.

[36] Note, the global-welfare maximizing solution within the feasible policy set does not include any

nisms would be needed to allow policymakers to achieve the welfare-maximizing Nash equilibrium. We leave the study of such mechanisms for future work.

In summary, local policy under pandemic federalism can lead to a suboptimal prisoners' dilemma for which all travel is banned. When regions are symmetric, this outcome leads to large welfare losses relative to no restrictions. When regions are asymmetric in size, with disease dynamics dominated by the large region, welfare losses are much less for the small region when travel is restricted. In both cases, welfare can be improved by enforcing travel bans only when infection rates are high. In Section 6, we use the surrogate model to study an intermediate case, where regions are asymmetric, but the small region is not so small that it is unable to influence aggregate dynamics.

6 Robustness

Is the quantitative relevance of travel choices a feature of our specific calibration to the COVID-19 pandemic or does it extend to alternative diseases? What are the key parameters driving the direction and magnitude of our positive and normative findings? To answer these questions, we conduct a comprehensive exploration of the parameter space, by solving our epi-econ model for *one million* unique epidemiological-economic parameter combinations.[37] We then use these model solutions to train surrogates of key features of our model in order to isolate the direction and magnitude of each parameter's influence on our results.[38]

We begin our parameter space exploration by documenting how often travel is effective across the one million economies. On average, susceptibles prefer travel to isolation for 37% of a pandemic period.[39] Consistent with Proposition 2, this

travel restrictions under an Infections-Guided decision rule. However, modest travel restrictions are optimal when following an Infections-Guided rule with asymmetric regions.

[37] Online Appendix D.1 details how we use estimates of disease characteristics from the epidemiological literature to discipline the boundaries of our parameter space.

[38] A surrogate model is an estimate $\hat{f}$ of an unknown mapping $f : \mathbb{X} \to \mathbb{R}^m$ from an input space $\mathbb{X}$ to outcomes $y = f(\underline{\mathbf{x}})$, $\underline{\mathbf{x}} \in \mathbb{X}$. In our case, $\mathbb{X}$ is the space of model parameters, while y represents a model outcome of interest (e.g. model-generated moments). We use deep neural networks to estimate the surrogate $\hat{f}$. Details concerning the surrogate model are provided in Online Appendix D. Throughout our surrogate analysis, we report results for the home (small) region.

[39] Online Appendix D.4 defines the statistic used to evaluate the share of days in which travel is an

percentage goes up to 50% if we restrict attention to the pandemic periods when the global infectious population share is low (below 1%).

We next use surrogate models to study what factors drive this result. The reproductive number R_0 emerges as the key parameter, underscoring the predominant role of disease contagiousness for endogenous avoidance strategies.[40] Figure 3 illustrates how the share of time during which travel is effective varies with R_0 when we consider either the entire pandemic (Panel A) or periods when the infectious population share is below 1% (Panel B).[41] These panels offer two key insights. First, susceptibles are more likely to prefer travel to home isolation for higher values of the reproductive number, with a predicted average effectiveness of 30% when $R_0 \leq 6$ and 38% otherwise (average effectiveness increases to 34% for $R_0 \leq 6$ and 56% for $R_0 > 6$ when we restrict attention to only the periods where the infectious population is below 1%). Second, the effectiveness of travel is highly sensitive to R_0 for prominent diseases with reproductive numbers below 6, such as Ebola, H1N1, the seasonal flu, SARS, and COVID-19. This finding highlights the value of early accurate estimates of this key epidemiological parameter.[42]

Next, we study how characteristics of the virus and economy dictate disease dynamics, and the extent to which these are driven by endogenous vs. exogenous travel response. Section 5.2 shows both exogenous and endogenous travel significantly reduce the time to peak infectious in the home region compared to a home isolation autarky, by synchronizing infection rates across regions. This quantitative finding remains robust across our parameter space exploration for exogenous travel, though the reduction in time between peaks is far more modest (0.37 days

effective asset over the days in which global infections are larger than a small threshold (1e-6). This statistic ensures comparability among diseases with different contagiousness of transmission.

[40] We use the permutation feature importance methodology to determine which features (parameters) drive the results. Details and results for all features are provided in Online Appendix D.5.

[41] Figure 3 is called a Partial Dependence (PD) plot. This graph reports the dependence of a model outcome on a parameter (horizontal axis), computed as the average of the predicted surrogate model outcomes across all occurrences of the other parameters in the training data. Online Appendix D.6 provides further details.

[42] Table 6 in the Online Appendix provides estimates from the literature of the R_0 for these and other diseases. We verified that this non-monotonicity is not the numerical feature of an overly sparse grid by confirming its persistence after we replicate the analysis for $R_0 \in [1.01, 6]$ (Online Appendix E.4).

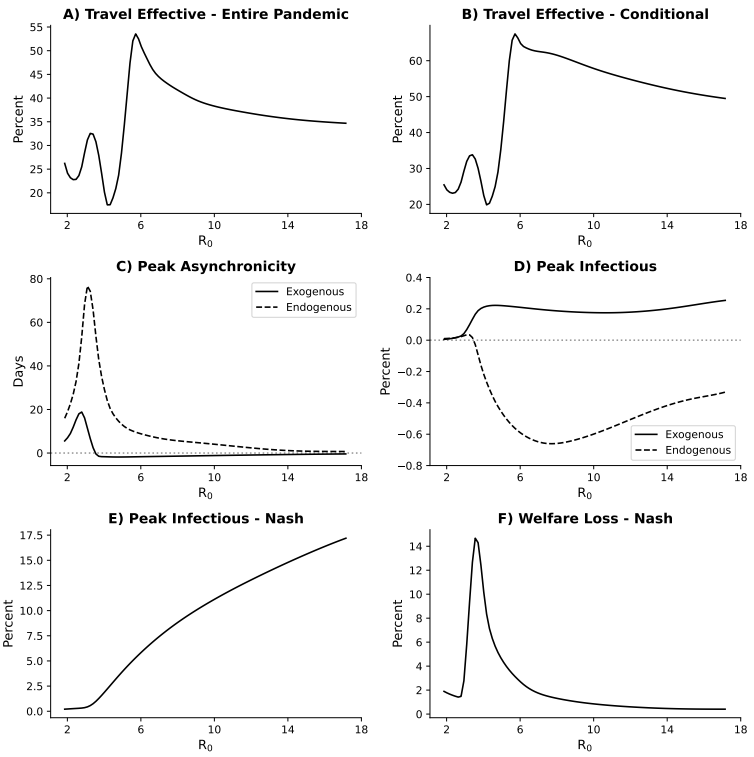

Figure 3: Partial dependence plot of model outcomes (vertical axis) on R_0 (horizontal axis). A) Average percent of days travel is an effective avoidance asset. B) Average percent of days travel is an effective avoidance asset when the infectious population share is above 1%. C) Change in the time between home and foreign peaks from a home isolation autarky model to models with exogenous (solid line) or endogenous (dashed line) travel. D) Percentage point change in peak infectious (percent of population) from a home isolation autarky model to models with exogenous (solid line) or endogenous (dashed line) travel. E) Peak infectious under the aggregate-welfare-minimizing Prisoner's Dilemma. F) Change in welfare (consumption equivalent units) from the aggregate-welfare-maximizing coordinated solution to the aggregate-welfare-minimizing Prisoner's Dilemma.

on average). Conversely, we find endogenous travel reduces synchronization, by increasing the time between peaks by an average of 10.8 days. This result reveals a qualitatively and quantitatively distinct impact of endogenous compared to exogenous travel than indicated by our benchmark calibration.

Over our parameter space exploration, peak infectious is on average 9.13% under home isolation autarky. In contrast to our COVID-19 calibration in Section 5.2, endogenous travel reduces (instead of increases) peak infectious, but the effect is modest (-6%). In line with our original calibration, exogenous travel increases the peak relative to the home isolation autarky, but by a smaller amount (9%).

Figure 3 shows how R_0 drives the impact of travel on disease dynamics through peak synchronization (Panel C) and peak infectious (Panel D). Panel C reports the absolute value of the time difference between peaks under endogenous and exogenous travel, minus the same statistic computed under home isolation autarky. A value above zero denotes a reduction in synchronicity. Under endogenous travel, marginal increases in R_0 significantly reduce synchronicity for low values of the reproductive number (up to 3.1), and increase synchronicity after that, though to a lesser degree for $R_0 > 6$. In the case of exogenous travel, low values of R_0 are associated with a reduction in synchronicity, but for $R_0 > 3.6$ exogenous travel is associated with stronger synchronicity between peaks, albeit modest, as noted above. Panel D shows infectious peaks vary with the reproductive number (relative to home isolation autarky). Endogenous travel is associated with a modest decrease (instead of a significant increase) in peak infectious for most values of R_0. This result suggests a qualitatively distinct role of travel for mitigating the heat of the pandemic, as opposed to that indicated by the COVID-19 calibration. These results underscore the implications of the avoidance role of endogenous travel choices vis-á-vis exogenous travel choices on disease dynamics.

We close our analysis by exploring the determinants of welfare effects under time-invariant travel restrictions.[43] In particular, we use the Nash equilibrium of the game between local policymakers (Section 5.3) to study how equilibrium welfare varies throughout the parameter space. Across the one million parameter combina-

[43] Solving for the Infections-Guided Nash equilibrium is computationally intensive for a single economy and infeasible for parameter space exploration.

tions, we find 99% lead to a Nash equilibrium that is not Pareto optimal, consistent with the results in Section 5.3. However unlike under the COVID-19 calibration with fixed policy rules, we find the socially optimal policy involves positive restrictions on travel for 32% of economies. This suggests the implementation of globally-optimal travel bans necessitates consideration of the particular epidemiological and economic characteristics.[44]

We once again use the surrogate model to examine the impact of R_0–the primary driver of the results–on outcomes in the local policymakers' game.[45] Figure 3 Panel E shows home peak infectious population share under the (global) welfare-minimizing Nash equilibrium. Panel F displays the loss in aggregate welfare relative to the globally-optimal coordinated solution. As was the case in the absence of public policy, most variation in welfare losses associated with the Prisoner's Dilemma outcome arises for $R_0 < 6$, where we see non-monotonic effects on welfare. Interestingly, for R_0 higher than this, average welfare losses relative to the coordinated solution *decrease* with R_0, indicating a smaller role for coordinated policy with very infectious diseases.[46]

To sum up, this section explores the robustness of our COVID-19 calibrated-predictions of behavioral responses to disease dynamics, the response of disease dynamics to travel choices, and welfare outcomes under pandemic federalism across the parameter space. Our analysis reveals that several of the quantitative results of Section 5 depend on the particular disease under consideration. Critically, we find endogenous travel alleviates the severity of the pandemic, in contrast to what is suggested by the benchmark calibration to COVID-19. This highlights the importance of parameter space exploration of complex systems and sounds a note of caution against drawing general conclusions from a single parametrization.

[44] As in Section 5.3, when considering Pareto optimality, we restrict the feasible set of travel restrictions to those which limit travel to no more than 1%, 25%, 50%, or 75% of average pre-pandemic travel, or no restrictions on travel.

[45] We drop the 1% of economies which do not have a Prisoner's Dilemma Nash equilibrium for our surrogate analysis of the local policymakers' game.

[46] While higher R_0 leads to a decline in *relative* welfare losses, the absolute change in welfare can be large, as suggested by the increasing peak infectious share in Figure 3 Panel E.

7 Conclusions

We develop a coupled epidemiological-economic metapopulation model to examine the role of travel choices in a pandemic. Our analysis focuses on a two region model. This assumption yields analytical tractability, clean interpretability of our quantitative results, and allows us to study the Nash game between local social planners. In our future research, we plan to relax this assumption in order to quantitatively investigate the effect of endogenous travel choices on disease dynamics in a multi-region model.

We highlight the role of travel as an infection avoidance strategy as well as a vector of pandemic transmission. Quantitatively, we find susceptible individuals are more likely to use travel as an avoidance strategy during the early run-up and late-stages of a pandemic, but rely more on home isolation strategies during peak infection periods. We also find modeling endogenous travel decisions is particularly quantitatively important when considering regions of asymmetric size. Our normative analysis shows locally-chosen travel policies under pandemic federalism lead to a suboptimal Prisoner's Dilemma outcome whereby both regions implement strict travel bans on incoming travelers, to the detriment of overall welfare.

Finally, we study the robustness of our quantitative results by solving the model for one million combinations of structural parameters. This analysis confirms the role of travel as an effective avoidance mechanism when infection rates are low, as well as the robustness of the Prisoner's Dilemma Nash equilibrium outcome across parametrizations. Crucially, we find endogenous travel mitigates the severity of the pandemic, contrary to the implications of our benchmark calibration. We use neural networks to estimate surrogates of the key model outcomes. This analysis shows the basic reproductive number R_0 is the most important parameter in driving disease dynamics and ultimately welfare consequences of pandemics.

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Online Appendix

A Equilibrium Characterization

This section collects the proofs of the propositions in the paper. We start by deriving/describing objects and listing assumptions that will be useful in the next subsections.

Pre-pandemic value function. The first order condition for the pre-pandemic agent's problem (5) is

$$\frac{\beta_i}{m_t^{pre}} = \frac{(1-\beta_i)}{1-m_t^{pre}},$$

which implies $m_t^{pre} = \beta_i$. Since the pre-pandemic problem is stationary with $U_{i,t}^{pre} = U_{i,t+1}^{pre} = U_i^{pre}$, we can rewrite the pre-pandemic value function in (5) as

$$U_i^{pre} = \frac{1}{1-\delta(1-P^{D^*})}\left[(1-\beta_i)\ln(w\bar{l}(1-\beta_i)) + \beta_i \ln(\beta_i) + \delta P^{D^*}\Omega_i\right].$$

Recovered value function. The first order condition for the recovered agent's problem (8) is

$$\frac{\beta_i}{m_t^R} = \frac{(1-\beta_i)}{(1-\theta_i)-m_t^R},$$

which implies $m_t^R = (1-\theta_i)\beta_i$. In the final period of the pandemic $t = T$, the recovered value function in (8) is

$$\begin{aligned} U_{i,T}^R &= (1-\beta_i)\ln(w\bar{l}(1-\theta_i)(1-\beta_i)) + \beta_i \ln((1-\theta_i)\beta_i) + \delta\left((1-P^{D^*})U_{i,T+1}^R + P^{D^*}\Omega_i\right) \\ &= (1-\beta_i)\ln(w\bar{l}(1-\theta_i)(1-\beta_i)) + \beta_i \ln((1-\theta_i)\beta_i) + \delta\left((1-P^{D^*})U_i^{pre} + P^{D^*}\Omega_i\right), \end{aligned}$$

since $U_{i,t}^R = U_i^{pre}$ for $t > T$ after the arrival of a vaccine. Likewise in period $t =$

$T-1$, the recovered value function is,

$$\begin{aligned}
U^R_{i,T-1} &= (1-\beta_i)\ln(w\bar{l}(1-\theta_i)(1-\beta_i)) + \beta_i \ln((1-\theta_i)\beta_i) + \delta\left((1-P^{D^*})U^R_{i,T} + P^{D^*}\Omega_i\right) \\
&= (1-\beta_i)\ln(w\bar{l}(1-\theta_i)(1-\beta_i)) + \beta_i \ln((1-\theta_i)\beta_i) \\
&+ \delta\Bigg[(1-P^{D^*})\Big((1-\beta_i)\ln(w\bar{l}(1-\theta_i)(1-\beta_i)) + \beta_i \ln((1-\theta_i)\beta_i) \\
&+ \delta\left((1-P^{D^*})U^{pre}_i + P^{D^*}\Omega_i\right)\Big) + P^{D^*}\Omega_i\Bigg].
\end{aligned}$$

For general $U^R_{i,T-j}$ with $j \in \{0,...,T-1\}$, we have

$$\begin{aligned}
U^R_{i,T-j} &= \sum_{t=0}^{j}\left(\delta(1-P^{D^*})\right)^t\left[(1-\beta_i)\ln(w\bar{l}(1-\theta_i)(1-\beta_i)) + \beta_i\ln((1-\theta_i)\beta_i)\right] \\
&+ \sum_{t=0}^{j}\left(\delta P^{D^*}\right)^{t+1}\Omega_i + \left(\delta(1-P^{D^*})\right)^{j+1}U^{pre}_i. \qquad (24)
\end{aligned}$$

Infectious value function. The first order condition for the infectious agent's problem (7) is

$$\frac{\beta^I_i}{m^I_t} = \frac{(1-\beta^I_i)}{(1-\theta_i)-m^I_t},$$

which implies $m^I_t = (1-\theta_i)\beta^I_i$. In the final period of the pandemic $t=T$, the infectious value function in (7) is

$$\begin{aligned}
U^I_{i,T} &= (1-\beta^I_i)\ln(\phi w\bar{l}(1-\theta_i)(1-\beta^I_i)) + \beta^I_i\ln((1-\theta_i)\beta^I_i) \\
&+ \delta[(1-P^R-P^D-P^{D^*})U^I_{i,T+1} + P^R U^R_{i,T+1} + (P^D+P^{D^*})\Omega_i] \\
&= (1-\beta^I_i)\ln(\phi w\bar{l}(1-\theta_i)(1-\beta^I_i)) + \beta^I_i\ln((1-\theta_i)\beta^I_i) + \delta[(1-P^D-P^{D^*})U^{pre}_i \\
&+ (P^D+P^{D^*})\Omega_i],
\end{aligned}$$

since $U^R_{i,t} = U^I_{i,t} = U^{pre}_i$ for $t > T$ after the arrival of a vaccine. Likewise in period

$t = T-1$, the infectious value function is,

$$\begin{aligned}U^I_{i,T-1} &= (1-\beta^I_i)\ln(\phi w\bar{l}(1-\theta_i)(1-\beta^I_i)) + \beta^I_i \ln((1-\theta_i)\beta^I_i) + \delta[(1-P^R-P^D-P^{D^*})U^I_{i,T} \\ &\quad + P^R U^R_{i,T} + (P^D+P^{D^*})\Omega_i] \\ &= (1-\beta^I_i)\ln(\phi w\bar{l}(1-\theta_i)(1-\beta^I_i)) + \beta^I_i \ln((1-\theta_i)\beta^I_i) + \delta\Big[(1-P^R-P^D-P^{D^*}) \\ &\quad \Big((1-\beta^I_i)\ln(\phi w\bar{l}(1-\theta_i)(1-\beta^I_i)) + \beta^I_i \ln((1-\theta_i)\beta^I_i) + \delta[(1-P^D-P^{D^*})U^{pre}_i \\ &\quad + (P^D+P^{D^*})\Omega_i]\Big) + P^R U^R_{i,T} + (P^D+P^{D^*})\Omega_i\Big].\end{aligned}$$

For general $U^I_{i,T-j}$ with $j \in \{0,...,T-1\}$, we have

$$\begin{aligned}U^I_{i,T-j} &= \sum_{t=0}^{j}\Big(\delta(1-P^R-P^D-P^{D^*})\Big)^t\Big[(1-\beta^I_i)\ln(\phi w\bar{l}(1-\theta_i)(1-\beta^I_i)) + \beta^I_i\ln((1-\theta_i)\beta^I_i)\Big] \\ &\quad + \sum_{t=0}^{j}\Big(\delta P^R\Big)^{t+1} U^R_{T+1-j+t} + \sum_{t=0}^{j}\Big(\delta(P^D+P^{D^*})\Big)^{t+1}\Omega_i + \Big(\delta(1-P^R-P^D-P^{D^*})\Big)^{j+1} U^{pre}_i,\end{aligned}$$

with $U^R_{T+1} = U^{pre}_i$ and U^R_t given by (24) for $t \in \{1,...T\}$.

Assumption 2. ***Restrictions on*** θ_i. *We focus on the interesting case where social distancing is low enough,* $\theta_i \in [0,\bar{\theta})$, *such that* $U^R_{i,t}, U^S_{i,t}, U^I_{i,t} \geq \Omega_i$. *We define* $0 < \bar{\theta} \leq 1$ *as the upper bound to the social distancing restriction such that* $U^I_{i,1} = \Omega_i$. *Under the conditions of Lemma 1,* $\theta_i \in [0,\bar{\theta})$ *is a sufficient condition for* $U^I_{i,t} > \Omega_i$ $\forall t$.

This is a quantitative assumption that rules out drastic social policy interventions where agents prefer to be dead than alive. The parameter $\bar{\theta}$ can be derived analytically and it is available upon request.

A.1 No selfish incentives for herd immunity

The following lemma establishes a sufficient condition that eliminates the *selfish incentives for herd immunity*.

Lemma 1. *The value function of being recovered is always larger than the value function of being infectious or susceptible,* $U^R_{i,t} > U^I_{i,t}$, $U^R_{i,t} \geq U^S_{i,t}$. *The value of*

being susceptible is larger than the one of being infectious, $U^R_{i,t} \geq U^S_{i,t} > U^I_{i,t}$, when catching the disease is costly enough, that is

$$\Big(u_i(c^R(m),m) - u^I_i(c^I(m),m)\Big) - \delta(1-P^D)(U^R_{i,t} - \Omega_i) \geq 0, \qquad \forall m,t$$

where $c^R(m) \equiv w\bar{l}(1-\theta_i - m)$ and $c^I(m) \equiv \phi w\bar{l}(1-\theta_i - m)$. That is, when the infectious state productivity loss is large enough (ϕ small) or the probability of death due to the virus, P^D is large enough.

We prove the value functions ordering presented in Lemma 1

$$U^R_{i,t} \geq U^S_{i,t} > U^I_{i,t} > \Omega_i \qquad \forall t \in \{1,2,\ldots,T\}$$

by backward induction. First, use the budget constraint for each disease type S, I, R to define functions:

$$\begin{aligned} c^S(m) &\equiv w\bar{l}(1-\theta_i - m), \\ c^I(m) &\equiv \phi w\bar{l}(1-\theta_i - m), \\ c^R(m) &\equiv w\bar{l}(1-\theta_i - m), \end{aligned}$$

The proof proceeds in three steps.

Step 1. We show that $U^R_{i,t} > U^I_{i,t}$ $\forall t \in \{1,2,\ldots,T\}$. In the last period of the pandemic, T,

$$\begin{aligned} U^R_{i,T} &= \max_{m_T}\{u_i(c^R(m_T),m_T) + \delta\Big((1-P^{D^*})U^R_{i,T+1} + P^{D^*}\Omega_i\Big)\} \\ &= \max_{m_T}\{u_i(c^R(m_T),m_T) + \delta\Big((1-P^{D^*})U^{pre}_{i,T+1} + P^{D^*}\Omega_i\Big)\} \\ &> \max_{m_T}\{u_i(c^R(m_T),m_T) + \delta\Big((1-P^D-P^{D^*})U^{pre}_{i,T+1} + (P^D+P^{D^*})\Omega_i\Big)\} \\ &= \max_{m_T}\{u_i(c^R(m_T),m_T) + \delta\Big((1-P^R-P^D-P^{D^*})U^{pre}_{i,T+1} + P^R U^{pre}_{i,T+1} + (P^D+P^{D^*})\Omega_i\Big)\} \\ &> \max_{m_T}\{u_i(c^I(m_T),m_T) + \delta\Big((1-P^R-P^D-P^{D^*})U^{pre}_{i,T+1} + P^R U^{pre}_{i,T+1} + (P^D+P^{D^*})\Omega_i\Big)\} \\ &= \max_{m_T}\{u_i(c^I(m_T),m_T) + \delta\Big((1-P^R-P^D-P^{D^*})U^I_{i,T+1} + P^R U^I_{i,T+1} + (P^D+P^{D^*})\Omega_i\Big)\} \\ &= U^I_{i,T}, \end{aligned}$$

where the first inequality follows because $U_{i,t}^{pre} > \Omega_i$ as explained in Section 4, and the second inequality uses the fact that $c^R(m) = w\bar{l}((1-\theta_i)-m) > \phi w\bar{l}((1-\theta_i)-m) = c^I(m)$ for all $m \in [0, 1-\theta]$, $\phi < 1$. Now, let $t = T-1$

$$
\begin{aligned}
U_{i,t}^R &= \max_{m_t}\{u_i(c^R(m_t), m_t) + \delta\left((1-P^{D^*})U_{i,t+1}^R + P^{D^*}\Omega_i\right)\} \\
&> \max_{m_t}\{u_i(c^R(m_t), m_t) + \delta\left((1-P^{D}-P^{D^*})U_{i,t+1}^R + (P^{D}+P^{D^*})\Omega_i\right)\} \\
&= \max_{m_t}\{u_i(c^R(m_t), m_t) + \delta\left((1-P^{R}-P^{D}-P^{D^*})U_{i,t+1}^R + P^R U_{i,t+1}^R + (P^{D}+P^{D^*})\Omega_i\right)\} \\
&> \max_{m_t}\{u_i(c^R(m_t), m_t) + \delta\left((1-P^{R}-P^{D}-P^{D^*})U_{i,t+1}^I + P^R U_{i,t+1}^R + (P^{D}+P^{D^*})\Omega_i\right)\} \\
&> \max_{m_t}\{u_i(c^I(m_t), m_t) + \delta\left((1-P^{R}-P^{D}-P^{D^*})U_{i,t+1}^I + P^R U_{i,t+1}^R + (P^{D}+P^{D^*})\Omega_i\right)\} \\
&= U_{i,t}^I,
\end{aligned}
$$

where the first inequality uses $U_{i,t}^R > \Omega_i$, the second uses $U_{i,t+1}^R > U_{i,t+1}^I$, and the third uses $c^R(m) > c^I(m)$. This establishes $U_{i,T-1}^R > U_{i,T-1}^I$. Proceeding backward and recursively in a similar fashion for $t \in \{T-2, T-3, \ldots 1\}$ proves the result. **Step 2.** We show that $U_{i,t}^R \geq U_{i,t}^S$ for $t \in \{1, 2, \ldots, T\}$. Proceeding by backward induction, in the final period, T,

$$
\begin{aligned}
U_{i,T}^S &= \max_{m_T}\{u_i(c^S(m_T), m_T) + \delta[(1-P_i^I(m_T, 1-\theta_i-m_T, 0; \mathscr{T}_T) - P^{D^*})U_{i,T+1}^S \\
&\quad + P_i^I(m_T, 1-\theta_i-m_T, 0; \mathscr{T}_T)U_{i,T+1}^I + P^{D^*}\Omega_i]\} \\
&= \max_{m_T}\{u_i(c^S(m_T), m_T) + \delta[(1-P_i^I(m_T, 1-\theta_i-m_T, 0; \mathscr{T}_T) - P^{D^*})U_{i,T+1}^{pre} \\
&\quad + P_i^I(m_T, 1-\theta_i-m_T, 0; \mathscr{T}_T)U_{i,T+1}^{pre} + P^{D^*}\Omega_i]\} \\
&= \max_{m_T}\{u_i(c^R(m_T), m_T) + \delta[(1-P^{D^*})U_{i,T+1}^{pre} + P^{D^*}\Omega_i]\} \\
&= U_{i,T}^R,
\end{aligned}
$$

since $c^S(m) = c^R(m)$ for all $m \in [0, \bar{m}]$. Accordingly, we have that $U_{i,T}^S = U_{i,T}^R > U_{i,T}^I$.

Proceeding backward and recursively, note that in $T-1$,

$$\begin{aligned}
U^R_{i,T-1} &= \max_{m_{T-1}}\{u_i(c^R(m_{T-1}),m_{T-1}) + \delta\left((1-P^{D^*})U^R_{i,T} + P^{D^*}\Omega_i\right)\} \\
&= \max_{m_{T-1}}\{u_i(c^S(m_{T-1}),m_{T-1}) + \delta\left((1-P^{D^*})U^S_{i,T} + P^{D^*}\Omega_i\right)\} \\
&= \max_{m_{T-1}}\{u_i(c^S(m_{T-1}),m_{T-1}) + \delta[(1-P^I_i(m_{T-1},1-\theta_i-m_{T-1},0;\mathscr{T}_{T-1}) \\
&\qquad - P^{D^*})U^S_{i,T} + P^I_i(m_{T-1},1-\theta_i-m_{T-1},0;\mathscr{T}_{T-1})U^S_{i,T} + P^{D^*}\Omega_i]\} \\
&> \max_{m_{T-1}}\{u_i(c^S(m_{T-1}),m_{T-1}) + \delta[(1-P^I_i(m_{T-1},1-\theta_i-m_{T-1},0;\mathscr{T}_{T-1}) \\
&\qquad - P^{D^*})U^S_{i,T} + P^I_i(m_{T-1},1-\theta_i-m_{T-1},0;\mathscr{T}_{T-1})U^I_{i,T} + P^{D^*}\Omega_i]\} \\
&= U^S_{i,T-1},
\end{aligned}$$

where the inequality follows from $U^S_{i,T} = U^R_{i,T} > U^I_{i,T}$. Next, consider $t = T-2$. Then,

$$\begin{aligned}
U^R_{i,t} &= \max_{m_t}\{u_i(c^R(m_t),m_t) + \delta\left((1-P^{D^*})U^R_{i,t+1} + P^{D^*}\Omega_i\right)\} \\
&= \max_{m_t}\{u_i(c^S(m_t),m_t) + \delta[(1-P^I_i(m_t,1-\theta_i-m_t,0;\mathscr{T}_t) - P^{D^*})U^R_{i,t+1} \\
&\qquad + P^I_i(m_t,1-\theta_i-m_t,0;\mathscr{T}_t)U^R_{i,t+1} + P^{D^*}\Omega_i]\} \\
&> \max_{m_t}\{u_i(c^S(m_t),m_t) + \delta[(1-P^I_i(m_t,1-\theta_i-m_t,0;\mathscr{T}_t) - P^{D^*})U^S_{i,t+1} \\
&\qquad + P^I_i(m_t,1-\theta_i-m_t,0;\mathscr{T}_t)U^I_{i,t+1} + P^{D^*}\Omega_i]\} \\
&= U^S_{i,t},
\end{aligned}$$

where the inequality follows since $U^S_{i,t+1} < U^R_{i,t+1}$ and $U^I_{i,t+1} < U^R_{i,t+1}$. This establishes $U^R_{i,T-2} > U^S_{i,T-2}$. Proceeding backward and recursively in a similar fashion for $t \in \{T-3,\dots 1\}$ proves the result.

Step 3. We show that $U^S_{i,t} > U^I_{i,t}$ for $t \in \{1,2,\dots,T\}$. Under the conditions of Lemma 1, we have the following inequality:

$$\left(u_i(c^S(m),m) - u^I_i(c^I(m),m)\right) - \delta(1-P^D)(U^R_{i,t} - \Omega_i) \geq 0, \qquad \forall m,t,$$

since $c^S(m) = c^R(m)\ \forall m$. With a slight abuse of notation, let $U^S_{i,t}(m)$, $U^I_{i,t}(m)$ denote

the objective function of the susceptible and infectious, respectively, evaluated at a given m (not necessarily the optimum). In the last period of the pandemic, we have $U^S_{i,T} = U^R_{i,T} > U^I_{i,T}$. Proceeding backward and recursively, let $t = T-1$. The difference $U^S_{i,t}(m) - U^I_{i,t}(m)$ is given by

$$\begin{aligned}
U^S_{i,t}(m) - U^I_{i,t}(m) &= u_i(c^S(m_t), m_t) - u^I_i(c^I(m_t), m_t) + \delta[(1 - P^I_i(m_t, 1-\theta_i - m_t, 0; \mathscr{T}_t) - P^{D^*})U^S_{i,t+1} \\
&\quad + P^I_i(m_t, 1-\theta_i - m_t, 0; \mathscr{T}_t)U^I_{i,t+1} - (1 - P^R - P^D - P^{D^*})U^I_{i,t+1} - P^R U^R_{i,t+1} - P^D \Omega_i] \\
&> u_i(c^S(m_t), m_t) - u^I_i(c^I(m_t), m_t) + \delta[(1 - P^I_i(m_t, 1-\theta_i - m_t, 0; \mathscr{T}_t) - P^{D^*})U^S_{i,t+1} \\
&\quad + P^I_i(m_t, 1-\theta_i - m_t, 0; \mathscr{T}_t)U^I_{i,t+1} - (1 - P^R - P^D - P^{D^*})U^R_{i,t+1} - P^R U^R_{i,t+1} - P^D \Omega_i] \\
&= u_i(c^S(m_t), m_t) - u^I_i(c^I(m_t), m_t) + \delta[(1 - P^I_i(m_t, 1-\theta_i - m_t, 0; \mathscr{T}_t) - P^{D^*})U^S_{i,t+1} \\
&\quad + P^I_i(m_t, 1-\theta_i - m_t, 0; \mathscr{T}_t)U^I_{i,t+1} - (1 - P^D - P^{D^*})U^R_{i,t+1} - P^D \Omega_i] \\
&> u_i(c^S(m_t), m_t) - u^I_i(c^I(m_t), m_t) + \delta[(1 - P^I_i(m_t, 1-\theta_i - m_t, 0; \mathscr{T}_t) - P^{D^*})U^I_{i,t+1} \\
&\quad + P^I_i(m_t, 1-\theta_i - m_t, 0; \mathscr{T}_t)U^I_{i,t+1} - (1 - P^D - P^{D^*})U^R_{i,t+1} - P^D \Omega_i] \\
&= u_i(c^S(m_t), m_t) - u^I_i(c^I(m_t), m_t) + \delta[(1 - P^{D^*})U^I_{i,t+1} - (1 - P^D - P^{D^*})U^R_{i,t+1} - P^D \Omega_i] \\
&> u_i(c^S(m_t), m_t) - u^I_i(c^I(m_t), m_t) + \delta[(1 - P^{D^*})\Omega_i - (1 - P^D - P^{D^*})U^R_{i,t+1} - P^D \Omega_i] \\
&= u_i(c^S(m_t), m_t) - u^I_i(c^I(m_t), m_t) - \delta[(1 - P^D - P^{D^*})(U^R_{i,t+1} - \Omega_i)] \\
&> u_i(c^S(m_t), m_t) - u^I_i(c^I(m_t), m_t) - \delta[(1 - P^D)(U^R_{i,t+1} - \Omega_i)] \\
&\geq 0,
\end{aligned}$$

where the first inequality follows from $U^R_{i,t+1} > U^I_{i,t+1}$, the second from $U^S_{i,t+1} > U^I_{i,t+1}$, the third from $U^I_{i,t+1} > \Omega_i$, the fourth from $U^R_{i,t+1} > \Omega_i$, and the fifth by assumption. Since this holds for all m and the feasible set $[0, \bar{m}]$ is the same for both disease types, $m^S_{i,t}, m^I_{i,t} \in [0, \bar{m}]$, the inequality must also hold in the max, $U^S_{i,t} = \max_m U^S_{i,t}(m) > \max_m U^I_{i,t}(m) = U^I_{i,t}$. This establishes $U^S_{i,T-1} > U^I_{i,T-1}$. Proceeding backward and recursively in a similar fashion for $t \in \{T-2, \ldots 1\}$ proves the result.

A.2 Proof of Proposition 1

A.2.1 Case 1: Utility benefits from travel

Consider first the case where agents receive direct utility benefits from travel, $\frac{\partial u_i}{\partial m} > 0$. By Inada conditions, travel choices are strictly positive, $m > 0$. In an interior

solution, the first order condition (FOC) for the susceptible agent is

$$\frac{\partial u_i}{\partial m} = -\frac{\partial u_i}{\partial c}\cdot\frac{\partial c}{\partial m} + \delta(U^S_{i,t+1} - U^I_{i,t+1})\frac{\partial P^I_{i,t}}{\partial m}. \tag{25}$$

Uniqueness. Define the continuous function

$$E(m) \equiv \frac{\partial u_i(c(m),m)}{\partial m} + \frac{\partial u_i(c(m),m)}{\partial c(m)}\cdot\frac{\partial c(m)}{\partial m} - \delta(U^S_{i,t+1} - U^I_{i,t+1})\frac{\partial P^I_{i,t}(m)}{\partial m},$$

where $c(m) \equiv w_t\bar{l}(1-\theta_i-m)$ is defined from the Susceptible's budget constraint in (6) and

$$P^I_{i,t}(m) \equiv 1 - \left[\left(\frac{1-\theta_i-m}{1-\theta_i}\right)\left(1-\tau\cdot\frac{(I_{i,t}-M^I_{i,t}+M^I_{j,t})}{N_{i,t}}\right)^{\mathscr{C}^l_{i,t}} + \left(\frac{m}{1-\theta_i}\right)\left(1-\tau\cdot\frac{(I_{j,t}-M^I_{j,t}+M^I_{i,t})}{N_{j,t}}\right)^{\mathscr{C}^m_{i,t}}\right],$$

provides the probability of infection in the absence of home isolation. We then have $E(m) = 0$ at an optimum. Notice

$$\begin{aligned}\frac{dE(m)}{dm} &= \frac{\partial^2 u_i(c(m),m)}{\partial m\partial c(m)}\cdot\frac{\partial c(m)}{\partial m} + \frac{\partial^2 u_i(c(m),m)}{\partial m^2} + \left[\frac{\partial^2 u_i(c(m),m)}{\partial c(m)^2}\cdot\frac{\partial c(m)}{\partial m} + \right.\\ &\left.\frac{\partial^2 u_i(c(m),m)}{\partial c(m)\partial m}\right]\frac{\partial c(m)}{\partial m} + \frac{\partial u_i(c(m),m)}{\partial c(m)}\cdot\underbrace{\frac{\partial^2 c(m)}{\partial m^2}}_{=0} - \delta(U^S_{i,t+1} - U^I_{i,t+1})\underbrace{\frac{\partial^2 P^I_{i,t}}{\partial m^2}}_{=0}\\ &= \underbrace{\frac{\partial c(m)}{\partial m}}_{<0}\left[2\cdot\underbrace{\frac{\partial^2 u_i(c(m),m)}{\partial m\partial c(m)}}_{\geq 0} + \underbrace{\frac{\partial^2 u_i(c(m),m)}{\partial c(m)^2}\cdot\frac{\partial c(m)}{\partial m}}_{\geq 0}\right] + \underbrace{\frac{\partial^2 u_i(c(m),m)}{\partial m^2}}_{\leq 0} < 0\end{aligned}$$

where the inequalities follow from the susceptible's budget constraint and by assumptions on the functional form of utility. It follows that $E(m)$ is strictly decreasing in m.

Now note that by Inada conditions on u_i

$$\lim_{m\to 0} E(m) = \infty, \qquad \lim_{m\to(1-\theta_i)} E(m) = -\infty,$$

$E(m)$ is therefore a continuous strictly decreasing monotonic function of m between $(0, 1-\theta)$ that goes from $+\infty$ to $-\infty$. Then by application of the Intermediate Value Theorem to Monotonic Functions in an open set, the solution $E(m) = 0$ is unique.

The susceptible's optimal travel choice m is decreasing in B. Let $B \equiv \delta(U^S_{i,t+1} - U^I_{i,t+1})\frac{\partial P^I_{i,t}}{\partial m}$, which does not depend on m. By the implicit function theorem, from (25) we have,

$$\frac{\partial^2 u_i(c(m),m)}{\partial m \partial c(m)} \cdot \frac{\partial c(m)}{\partial m} \cdot \frac{\partial m}{\partial B} + \frac{\partial^2 u_i(c(m),m)}{\partial m^2} \cdot \frac{\partial m}{\partial B} =$$
$$- \left[\frac{\partial^2 u_i(c(m),m)}{\partial c(m)^2} \cdot \frac{\partial c(m)}{\partial m} \cdot \frac{\partial m}{\partial B} + \frac{\partial^2 u_i(c(m),m)}{\partial c(m) \partial m} \frac{\partial m}{\partial B} \right] \frac{\partial c(m)}{\partial m} - \frac{\partial u_i(c(m),m)}{\partial c(m)} \cdot \underbrace{\frac{\partial^2 c(m)}{\partial m^2}}_{=0} \cdot \frac{\partial m}{\partial B} + 1$$

Simple algebra shows that

$$\frac{\partial m}{\partial B} = \frac{1}{\underbrace{\frac{\partial c(m)}{\partial m}}_{<0} \left[2 \cdot \underbrace{\frac{\partial^2 u_i(c(m),m)}{\partial m \partial c(m)}}_{\geq 0} + \underbrace{\frac{\partial^2 u_i(c(m),m)}{\partial c(m)^2} \cdot \frac{\partial c(m)}{\partial m}}_{\geq 0} \right] + \underbrace{\frac{\partial^2 u_i(c(m),m)}{\partial m^2}}_{\leq 0}} < 0.$$

The susceptibles' optimal travel choice m converges to the recovered when $B \to 0$. As $B \to 0$ the FOC of the susceptible (25) reduces to FOC of the recovered,

$$\frac{\partial u_i}{\partial m} = \frac{\partial u_i}{\partial c} w \bar{l}.$$

Thus for $B < 0$, $m^S_{i,t} > m^R_{i,t}$ since $m^S_{i,t}$ decreases in B. Likewise, for $B > 0$, we have $m^S_{i,t} < m^R_{i,t}$.

Relative travel by susceptible depends on travel as infection avoidance asset or liability. Under the conditions of Lemma 1, $B < 0$ if and only if $\partial P^I_i / \partial m < 0$ (infection avoidance asset). In this case, $m^S_{i,t} > m^R_{i,t}$. Conversely if $\partial P^I_i / \partial m > 0$, then $B > 0$. Consequently $m^S_{i,t} < m^R_{i,t}$.

A.2.2 Case 2: No utility benefits from travel

Consider now the case where agents do not receive any direct utility benefits from travel, $u_i(c,m) = u_i(c)$. In this environment recovered agents do not travel (unless travel is costless). The susceptible's first order and Kuhn-Tucker conditions imply

$$\frac{\partial u_i}{\partial c} \cdot \frac{\partial c}{\partial m} - \delta(U^S_{i,t+1} - U^I_{i,t+1}) \frac{\partial P^I_{i,t}}{\partial m} \leq 0, \tag{26}$$

with equality if $m^S_{i,t} > 0$.

Uniqueness. Define the continuous function

$$F(m) \equiv -\delta(U^S_{i,t+1} - U^I_{i,t+1}) \frac{\partial P^I_{i,t}(m)}{\partial m} + \frac{\partial u_i(c(m))}{\partial c(m)} \cdot \frac{\partial c(m)}{\partial m}$$

such that $F(m) = 0$ at an interior solution. Differentiating with respect to m gives,

$$\begin{aligned} \frac{dF(m)}{dm} &= -\delta(U^S_{i,t+1} - U^I_{i,t+1}) \underbrace{\frac{\partial^2 P^I_{i,t}}{\partial m^2}}_{=0} + \frac{\partial^2 u_i(c(m))}{\partial c(m)^2} \cdot \left(\frac{\partial c(m)}{\partial m} \right)^2 + \frac{\partial u_i(c(m))}{\partial c(m)} \cdot \underbrace{\frac{\partial^2 c(m)}{\partial m^2}}_{=0} \\ &= \underbrace{\frac{\partial^2 u_i(c(m))}{\partial c(m)^2}}_{\leq 0} \cdot \left(\frac{\partial c(m)}{\partial m} \right)^2 \leq 0 \end{aligned}$$

where the inequality follow from the assumptions on the functional form of utility. By Inada conditions on u_i, we have that

$$\lim_{m \to (1-\theta_i)} F(m) = -\infty.$$

Notice $F(0) < \infty$. If $F(0) > 0$, there is a unique $m^S_{i,t} > 0$. Otherwise $m^S_{i,t} = 0$.

The susceptible travels more than recovered only if B<0. From (26),

$$-\frac{\partial u_i}{\partial c}(w\bar{l}) - \underbrace{\delta(U^S_{i,t+1} - U^I_{i,t+1}) \frac{\partial P^I_{i,t}}{\partial m}}_{=B} \leq 0.$$

Since $\partial u_i/\partial c > 0$, a necessary condition for $m^S_{i,t} > 0$ (and (26) to hold with equality) is $B < 0$. Equivalently, $m^S_{i,t} > 0$ implies $\partial P^I_{i,t}/\partial m < 0$. Thus when travel is an infection avoidance asset ($\partial P^I_{i,t}/\partial m < 0$), $m^S_{i,t} \geq 0$. Otherwise travel is an infection avoidance liability and the susceptible's traveling choice mimics the one of the recovered $m^S_{i,t} = m^R_{i,t} = 0$.

A.3 Proof of Proposition 2

We first prove Proposition 2.(a). First note that by definition,

$$\frac{\partial P^I_i}{\partial h} \equiv \frac{1}{1-\theta_i}\left[\underbrace{\left(1-\tau\cdot\underbrace{\frac{(I_{i,t}-M^I_{i,t}+M^I_{j,t})}{N_{i,t}}}_{\text{Home Exposure}}\right)^{\mathscr{C}^l_{i,t}} - \left(1-\tau\cdot\underbrace{\frac{(I_{i,t}-M^I_{i,t}+M^I_{j,t})}{N_{i,t}}}_{\text{Home Exposure}}\right)^{\mathscr{C}^h_{i,t}}}_{\text{Effectiveness of home isolation in reducing home-infection exposure }(-)}\right] \leq 0$$

where the inequality arises by inspection of the contact rates (exponents). When $\frac{\partial P^I_i}{\partial h} = 0$ home isolation is ineffective in reducing the likelihood of getting infected, and we are back in the environment of Proposition 1. For the rest of this analysis, let us focus on the interesting case where $\frac{\partial P^I_i}{\partial h} < 0$.

In the absence of direct utility benefits from travels, the first order and Kuhn-Tucker conditions for the susceptibles are

$$-\frac{du_i}{dc}w\bar{l} + \delta(U^{I'}_i - U^{S'}_i)\frac{\partial P^I_i}{\partial m} + \mu^{lo}_m - \mu^{hi}_m = 0, \tag{27}$$

$$-\frac{du_i}{dc}w\bar{l} + \delta(U^{I'}_i - U^{S'}_i)\frac{\partial P^I_i}{\partial h} + \mu^{lo}_h - \mu^{hi}_h = 0, \tag{28}$$

$$m\mu^{lo}_m = 0, \tag{29}$$

$$(\bar{m}-m)\mu^{hi}_m = 0, \tag{30}$$

$$h\mu^{lo}_h = 0, \tag{31}$$

$$(\bar{h}-h)\mu^{hi}_h = 0, \tag{32}$$

$$m, h, \mu^{lo}_m, \mu^{hi}_m, \mu^{lo}_h, \mu^{hi}_h \geq 0. \tag{33}$$

Assume an interior solution exists for both $h \in (0, \bar{h})$ and $m \in (0, \bar{m})$. By complementarity slackness conditions, $\mu_h^{hi} = \mu_h^{lo} = \mu_m^{hi} = \mu_h^{lo} = 0$. Then by combining (27) and (28) we get $\partial P_i^I / \partial m = \partial P_i^I / \partial h < 0$. In this edge case, there is a continuum of solutions, so assume without loss of generality that the agent chooses $m > 0$ and $h = 0$.

Let us now consider the case where $\partial P_i^I / \partial m < \partial P_i^I / \partial h < 0$. Simple manipulations yield

$$\frac{\partial P_i^I}{\partial m} - \frac{\partial P_i^I}{\partial h} = \frac{1}{1-\theta_i} \left[\left(1 - \tau \cdot \frac{(I_{i,t} - M_{i,t}^I + M_{j,t}^I)}{N_{i,t}} \right)^{\mathscr{C}_{i,t}^h} - \left(1 - \tau \cdot \frac{(I_{j,t} - M_{j,t}^I + M_{i,t}^I)}{N_{j,t}} \right)^{\mathscr{C}_{i,t}^m} \right] < 0,$$

By combining (27) and (28) we have:

$$\delta \underbrace{(U_i^{I'} - U_i^{S'})}_{\leq 0} \left(\frac{\partial P_i^I}{\partial m} - \frac{\partial P_i^I}{\partial h} \right) = \mu_m^{hi} + \mu_h^{lo} - \mu_h^{hi} - \mu_m^{lo}.$$

Since $U_i^{I'} < U_i^{S'}$ (Lemma 1) and $\partial P_i^I / \partial m < \partial P_i^I / \partial h < 0$, the left hand side is positive. Then we have the following cases:

- $h \in (0, \bar{h}]$ and $m = \bar{m} > 0$: i.e. $\mu_h^{lo} = 0$, $\mu_h^{hi} \geq 0$, $\mu_m^{lo} = 0$, $\mu_m^{hi} > 0$; $\mu_m^{hi} > \mu_h^{hi}$;
- $h = 0$ and $m \in (0, \bar{m}]$: i.e. $\mu_h^{lo} > 0$, $\mu_h^{hi} = 0$, $\mu_m^{lo} = 0$, $\mu_m^{hi} \geq 0$;
- $h = 0$ and $m = 0$: i.e. $\mu_h^{lo} > 0$, $\mu_m^{lo} \geq 0$, $\mu_h^{lo} > \mu_m^{lo}$;

which identify situations in which susceptibles weakly prefer travel to isolation as an infection avoidance mechanism. A similar reasoning can be applied to show that the reverse is true when $\partial P_i^I / \partial m < \partial P_i^I / \partial h < 0$.

Next we prove Proposition 2.(b). The proposition explores conditions under which the lagrange multiplier associated with the lower bound of home isolation is strictly positive ($\mu_h^{lo} > 0$), and, accordingly, agents do not isolate for infection avoidance purposes. First, independently of the presence of direct utility benefits

from travels, inspection of equation (28),

$$\mu_h^{lo} = \frac{du_i}{dc} w\bar{l} + \frac{\delta(U_i^{S'} - U_i^{I'})}{1-\theta_i} \Bigg[\underbrace{\left(1 - \tau \cdot \frac{(I_{i,t} - M_{i,t}^I + M_{j,t}^I)}{N_{i,t}}\right)^{\mathscr{C}_{i,t}^l} - \left(1 - \tau \cdot \frac{(I_{i,t} - M_{i,t}^I + M_{j,t}^I)}{N_{i,t}}\right)^{\mathscr{C}_{i,t}^h}}_{\to \quad 0 \quad \text{as home infection rates go to zero}} \Bigg]$$

shows that as infection rate in the home region goes to zero, the term in brackets on the right-hand-size goes to zero, and the multiplier converges to the marginal utility benefits of working, which is strictly positive.

Second, we explore how the relative benefits of travel choices affect the attractiveness of home isolation. To do so, in presence of direct utility benefits of travel we replace (27) with

$$\frac{du_i}{dm} - \frac{du_i}{dc} w\bar{l} + \delta(U_i^{I'} - U_i^{S'}) \frac{\partial P_i^I}{\partial m} + \mu_m^{lo} - \mu_m^{hi} = 0$$

Subtracting this expression from (28), and manipulating yields

$$\mu_h^{lo} = \frac{du_i}{dm} - \mu_m^{hi} + \frac{\delta(U_i^{S'} - U_i^{I'})}{1-\theta} \Bigg[\underbrace{\left(1 - \tau \cdot \frac{(I_{j,t} - M_{j,t}^I + M_{i,t}^I)}{N_{j,t}}\right)^{\mathscr{C}_{i,t}^m} - \left(1 - \tau \cdot \frac{(I_{i,t} - M_{i,t}^I + M_{j,t}^I)}{N_{i,t}}\right)^{\mathscr{C}_{i,t}^h}}_{\text{Effectiveness of Travel relative to Isolation in reducing infection exposure}} \Bigg]$$

Suppose: *(i)* home and foreign infection rates are sufficiently small (term in squared brackets goes to zero) or travel is more effective than home isolation in reducing the likelihood of infection (term in squared brackets is positive); *(ii)* marginal utility benefits of travel are sufficiently large, $\partial u / \partial m$; and *(iii)* the shadow value of relaxing the upper bound constraint on travel, μ_m^{hi}, is not too large. Under *(i)-(iii)* the lagrange multiplier μ_h^{lo} is positive, agents do not isolate and prefer travel for infection avoidance purposes.

A.4 Proof of Proposition 3

Pre-pandemic problem. The solution to the pre-pandemic agents' problem (5) satisfies the following FOC:

$$\frac{\beta_i}{m_{i,t}^{pre}} = \frac{(1-\beta_i)}{1-m_{i,t}^{pre}},$$

which implies $m_{i,t}^{pre} = \beta_i$.

Infectious and recovered problems. Likewise, the infectious and recovered FOCs are, respectively,

$$\frac{\beta_i^I}{m_{i,t}^I} = \frac{(1-\beta_i^I)}{(1-\theta_i)-m_{i,t}^I},$$
$$\frac{\beta_i}{m_{i,t}^R} = \frac{(1-\beta_i)}{(1-\theta_i)-m_{i,t}^R},$$

which imply $m_{i,t}^I = \beta_i^I(1-\theta_i)$ and $m_{i,t}^R = \beta_i(1-\theta_i)$.

Susceptible Problem. The FOC for the susceptible is:

$$\frac{\beta_i}{m_{i,t}^S} + \delta(U_{i,t+1}^I - U_{i,t+1}^S)\frac{\partial P_{i,t}^I}{\partial m_{i,t}^S} = \frac{(1-\beta_i)}{(1-\theta_i)-m_{i,t}^S},$$

where $\frac{\partial P_{i,t}^I}{\partial m}$ is given by (18).

Let $B \equiv \delta(U_{i,t+1}^S - U_{i,t+1}^I)\frac{\partial P_{i,t}^I}{\partial m}$, and rearrange the susceptibles' first order condition.

$$\frac{\beta_i}{m_{i,t}^S} = \frac{(1-\beta_i)+B(1-\theta_i)-Bm_{i,t}^S}{(1-\theta_i)-m_{i,t}^S},$$

The marginal benefits of travel in the left-hand-side is an hyperbolic function with a vertical asymptote at 0, strictly decreasing in the feasible set $m_{i,t}^S \in [0,\bar{m}]$. The right-hand-side is an hyperbolic function with vertical asymptote at $(1-\theta_i)$ and strictly increasing in the feasible set $m_{i,t}^S \in [0,\min\{\bar{m},1-\theta_i\}]$. Accordingly, given $B \equiv \delta(U_{i,t+1}^S - U_{i,t+1}^I)\frac{\partial P_{i,t}^I}{\partial m}$, there exists a unique real interior solution to the susceptible problem for $m \in [0,1-\theta]$. (Note, the upper-bound $\bar{m}$ may still be binding.)

Characterization of the solution. The solution to the susceptible problem consists of one of the roots $(\hat{m}^S_+, \hat{m}^S_-)$ that solve the quadratic equation

$$(m^S_{it})^2 - \left(\frac{1}{B} + (1-\theta_i)\right) m^S_{it} + \frac{\beta_i(1-\theta_i)}{B} = 0, \tag{34}$$

where

$$\hat{m}^S_+ \equiv \frac{1}{2}\left((1/B + (1-\theta_i)) + \sqrt{(1/B + (1-\theta_i))^2 - 4\beta_i(1-\theta_i)/B}\right),$$
$$\hat{m}^S_- \equiv \frac{1}{2}\left((1/B + (1-\theta_i)) - \sqrt{(1/B + (1-\theta_i))^2 - 4\beta_i(1-\theta_i)/B}\right),$$

From Proposition 1 we know that the sign of $\frac{\partial P^I_{i,t}}{\partial m}$ distinguishes the two cases of travel as infection avoidance asset (< 0) or liability (> 0), and in turn determins whether agents travel more or less than recovered, $m^R_{i,t} = (1-\theta_i)\beta_i$.

When $\frac{\partial P^I_{i,t}}{\partial m} < 0$, we have that $-4\beta_i(1-\theta_i)/B > 0$. Accordingly,

$$\sqrt{(1/B + (1-\theta_i))^2 - 4\beta_i(1-\theta_i)/B} > \sqrt{(1/B + (1-\theta_i))^2} = |\frac{1}{B} + (1-\theta_i)| \geq \frac{1}{B} + (1-\theta_i)$$

Accordingly, $\hat{m}^S_-$ is negative and not feasible. The unique solution in the case of travel as infectious avoidance asset is therefore $\hat{m}^S_+ > 0$

Let us now consider the case of travel as infectious avoidance liability, $\frac{\partial P^I_{i,t}}{\partial m} > 0$. From 1 we know that for any B the solution is lower than $m^R_{i,t} = (1-\theta_i)\beta_i$. By contradiction, let us suppose that the solution is the $\hat{m}^S_+$ root. Then

$$\left((1/B + (1-\theta_i)) + \sqrt{(1/B + (1-\theta_i))^2 - 4\beta_i(1-\theta_i)/B}\right) \leq 2(1-\theta_i)\beta_i$$

We also know that traveling decreases in B. Accordingly, let us focus on the case where $B \to \infty$, which gives the minimum of the left-hand-side for $B \in \mathbb{R}_+$ (travel as

infectious avoidance liability),

$$(1-\theta_i)+\sqrt{((1-\theta_i))^2} \leq 2(1-\theta_i)\beta_i$$
$$2(1-\theta_i) \leq 2(1-\theta_i)\beta_i$$

which reaches a contradiction, since $\beta_i < 1$ ($\perp$).

Since a unique solution exists, it follows that when $\frac{\partial P^I_{i,t}}{\partial m} > 0$, the susceptible optimal travel choice is the left-root,

$$\hat{m}^S_- \equiv \frac{1}{2}\left((1/B+(1-\theta_i)) - \sqrt{(1/B+(1-\theta_i))^2 - 4\beta_i(1-\theta_i)/B}\right) \leq m^R_{i,t}.$$

Accordingly, under Assumption 1, absent isolation choices, susceptible choose to travel more when traveling reduces the likelihood of becoming infectious $\left(\frac{\partial P^I_{i,t}}{\partial m^S_{i,t}} < 0\right)$ and less otherwise,

$$m^S_{i,t} = \begin{cases} \frac{(1+B(1-\theta_i))+\sqrt{(1+B(1-\theta_i))^2-4B\beta_i(1-\theta_i)}}{2B}, & \frac{\partial P^I_{i,t}}{\partial m^S_{i,t}} < 0 \quad \text{Inf. Avoidance Asset} \\ \frac{(1+B(1-\theta_i))-\sqrt{(1+B(1-\theta_i))^2-4B\beta_i(1-\theta_i)}}{2B}, & \frac{\partial P^I_{i,t}}{\partial m^S_{i,t}} > 0 \quad \text{Inf. Avoidance Liability} \end{cases}$$

Finally, we show the equality in (21). Define $\tilde{B} \equiv B(1-\theta_i)$. From (34) we have

$$B(m^S_{it})^2 - \left(1+(1-\theta_i)B\right)m^S_{it} + \beta_i(1-\theta_i) = 0.$$

This implies,

$$\begin{aligned}
m^S_{i,t} &= \frac{\left(1+(1-\theta_i)B\right) \pm \sqrt{\left(1+(1-\theta_i)B\right)^2 - 4B\beta_i(1-\theta_i)}}{2B} \\
&= \frac{1}{2B} + \frac{(1-\theta_i)}{2} \pm \frac{\sqrt{\left(1+(1-\theta_i)B\right)^2 - 4B\beta_i(1-\theta_i)}}{2B} \\
&= \frac{(1-\theta_i)}{2}\left(1+\frac{1}{\tilde{B}}\right) \pm \frac{(1-\theta_i)}{2\tilde{B}}\sqrt{\left(1+\tilde{B}\right)^2 - 4\beta_i\tilde{B}} \\
&= \frac{m^R_{i,t}}{2\beta_i}\left(1+\frac{1}{\tilde{B}}\right) \pm \frac{m^R_{i,t}}{2\tilde{B}\beta_i}\sqrt{1+\tilde{B}(\tilde{B}+2(1-2\beta_i))} \\
&= m^R_{i,t}\left[\frac{1}{2\beta_i} + \frac{1}{2\beta_i\tilde{B}}\left(1 \pm \sqrt{1+\tilde{B}(\tilde{B}+2(1-2\beta_i))}\right)\right].
\end{aligned}$$

Thus,

$$\begin{aligned}
\frac{m^S_{i,t}}{m^R_{i,t}} &= \frac{1}{2\beta_i} + \frac{1}{2\beta_i\tilde{B}}\left(1 \pm \sqrt{1+\tilde{B}(\tilde{B}+2(1-2\beta_i))}\right) \\
&= \frac{1}{2\beta_i} + \frac{2\beta_i - 1}{2\beta_i} + \left[\frac{1-2\beta_i}{2\beta_i} + \frac{1}{2\beta_i\tilde{B}}\left(1 \pm \sqrt{1+\tilde{B}(\tilde{B}+2(1-2\beta_i))}\right)\right] \\
&= 1 + \left[\frac{1-2\beta_i}{2\beta_i} + \frac{1}{2\beta_i\tilde{B}}\left(1 \pm \sqrt{1+\tilde{B}(\tilde{B}+2(1-2\beta_i))}\right)\right].
\end{aligned}$$

Now, to prove (22), note that for an interior solution with $h^S_{i,t} > 0$, the susceptible agent's FOCs imply

$$\frac{\beta_i}{m^S_{i,t}} + \delta(U^I_{i,t+1} - U^S_{i,t+1})\frac{\partial P^I_{i,t}}{\partial m^S_{i,t}} = \delta(U^I_{i,t+1} - U^S_{i,t+1})\frac{\partial P^I_{i,t}}{\partial h^S_{i,t}}.$$

Thus,

$$
\begin{aligned}
m^S_{i,t} &= \frac{\beta_i}{\delta(U^I_{i,t+1} - U^S_{i,t+1})(\frac{\partial P^I_{i,t}}{\partial h^S_{i,t}} - \frac{\partial P^I_{i,t}}{\partial m^S_{i,t}})} \\
&= \frac{(1-\theta_i)\beta_i}{\delta(U^I_{i,t+1} - U^S_{i,t+1})\left(\left(1-\tau\cdot\frac{(I_{j,t}-M^I_{j,t}+M^I_{i,t})}{N_{j,t}}\right)^{\mathscr{C}^m_{i,t}} - \left(1-\tau\cdot\frac{(I_{i,t}-M^I_{i,t}+M^I_{j,t})}{N_{i,t}}\right)^{\mathscr{C}^h_{i,t}}\right)} \\
&= \frac{m^R_{i,t}}{\delta(U^S_{i,t+1} - U^I_{i,t+1})\left(\left(1-\tau\cdot\frac{(I_{i,t}-M^I_{i,t}+M^I_{j,t})}{N_{i,t}}\right)^{\mathscr{C}^h_{i,t}} - \left(1-\tau\cdot\frac{(I_{j,t}-M^I_{j,t}+M^I_{i,t})}{N_{j,t}}\right)^{\mathscr{C}^m_{i,t}}\right)}.
\end{aligned}
$$

B Solution Algorithm

We introduce a solution algorithm to recover the sequence of probabilities that satisfy (10). The algorithm iterates between the epidemiological and economic model until the two are consistent.[47] The algorithm follows these steps:

1. **Initialize the Aggregate Disease State**: Start with an initial aggregate disease state of the economy, $\mathscr{T}_{i,0}$, where a small portion of the population is infectious to initiate the pandemic.

2. **Guess Infection Probabilities and Travel Choices**: Guess the region-specific time series of infection probabilities, $\{\mathscr{P}^{SI}_{i,t}\}_{t=1}^{T}$, and aggregate susceptibles' travels and consumption, $\{\hat{M}^{S}_{i,t}, \hat{C}^{S}_{i,t}\}_{t=1}^{T}$

3. **Simulate the Pandemic Evolution Forward**: Given $\{\mathscr{P}^{SI}_{i,t}\}_{t=1}^{T}$, simulate the evolution of the pandemic according to equations (1) through (4). Collect the aggregate disease state for each period, $\mathscr{T}_{i,t} = (S_{i,t}, I_{i,t}, R_{i,t}, D_{i,t})$.

4. **Compute Regional Populations**: Determine the population in each region and period using the sequence of aggregate disease states and equation (12).

5. **Solve the Agents Problem Backward**: Given $\{\mathscr{T}_{i,t}\}$, solve the susceptible agent's problem described in (6). Solve backward from the last period, $t = T$, to the beginning of the pandemic, $t = 0$.[48] Collect choices of infectious and recovered individuals, which are independent of the aggregate disease state, $\mathscr{T}_{i,t}$.

6. **Re-Simulate the Pandemic Forward**: Simulate the pandemic forward from $t = 0$ to T using the endogenous choices together with (11) to compute the share of susceptible which become infectious in each period $\{\tilde{P}^{I}_{i,t}\}_{t=1}^{T}$.

[47] In Appendix E.3, we verify the accuracy of the algorithm by comparing to another approach which uses the Fischer-Burmeister function to solve the equilibrium equations directly.

[48] We assume the pandemic ends with certainty at time T (e.g., due to the introduction of a vaccine). Thus, at time $T+1$, the remaining S, I, and R types become identical, all policy restrictions are lifted, and the continuation value for each type at time T is derived from the solution to (5).

7. **Compare and Update Guesses**: Compare the aggregate disease states generated from the initial guesses $\{\hat{P}^I_{i,t}\}^T_{t=1}$ with those derived from the endogenous choices $\{\tilde{P}^I_{i,t}\}^T_{t=1}$. If the sequences are sufficiently far, update the guesses and repeat steps 2-7 until convergence.

C More on Quantitative Analysis

C.1 Endogenous Travel and Isolation Choices

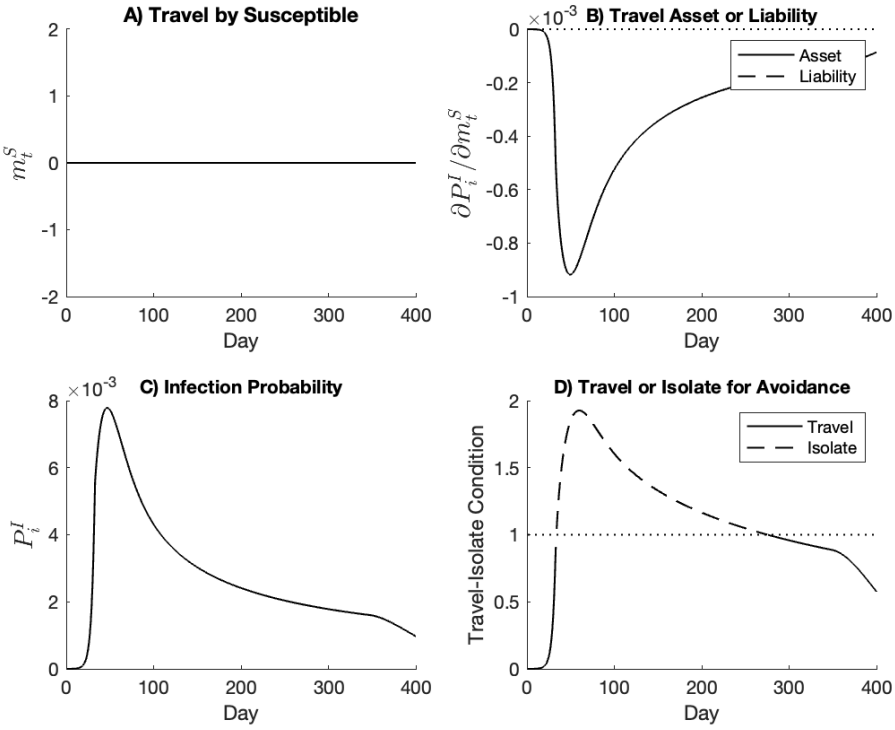

Figure 4: Symmetric regions with no utility from travel.

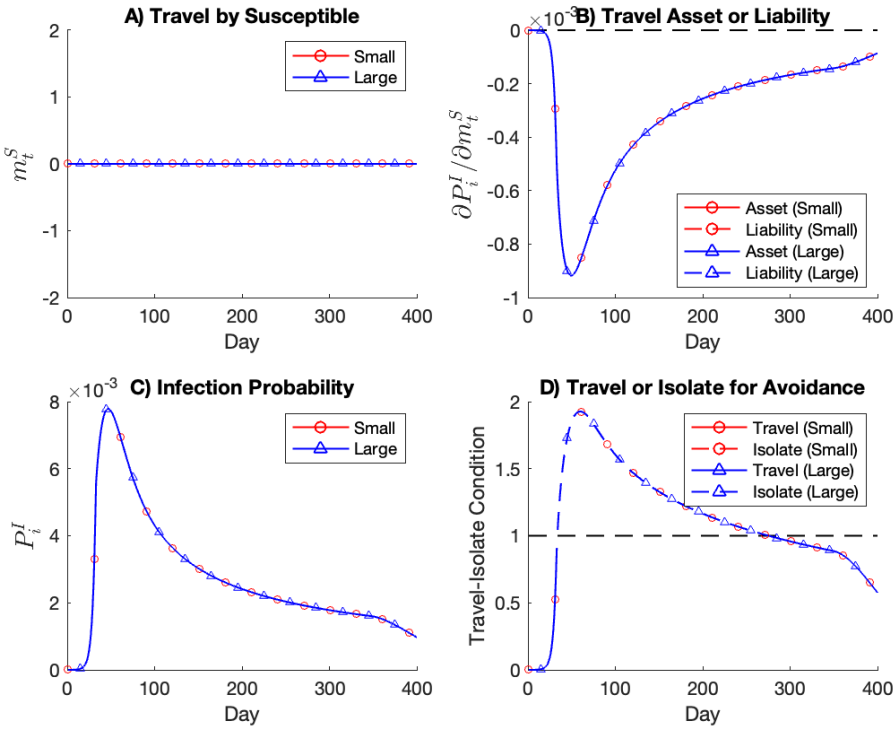

Figure 5: Asymmetric size regions with no utility from travel.

C.2 Disease Dynamics and Behavioral Responses

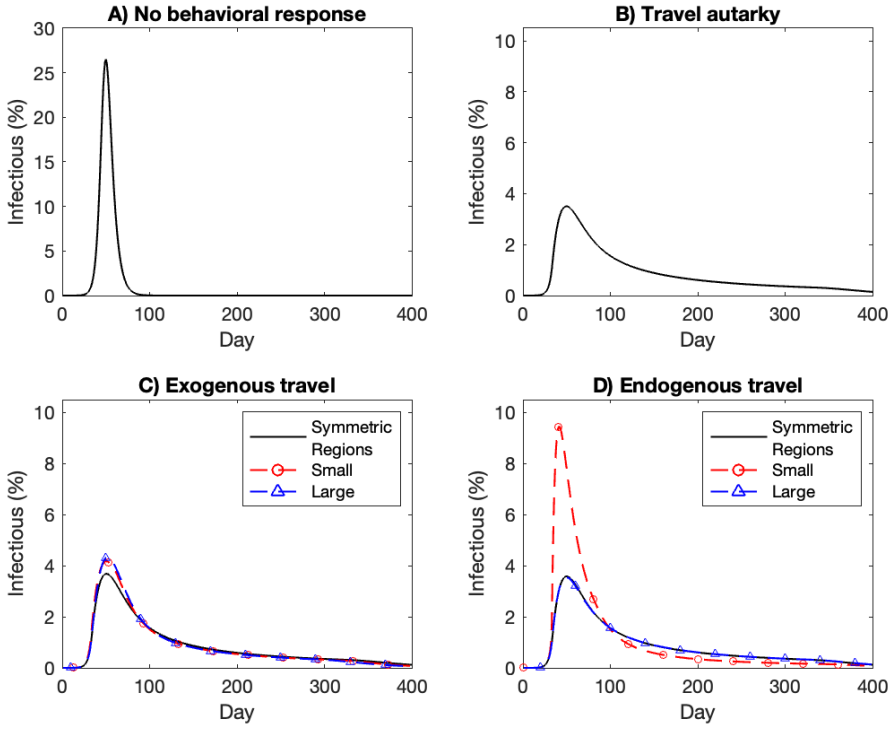

Figure 6: Infectious under no behavioral response, home isolation autarky, exogenous, and endogenous travel (benchmark) model specifications with identical home (small) and foreign (large) infectious seeds.

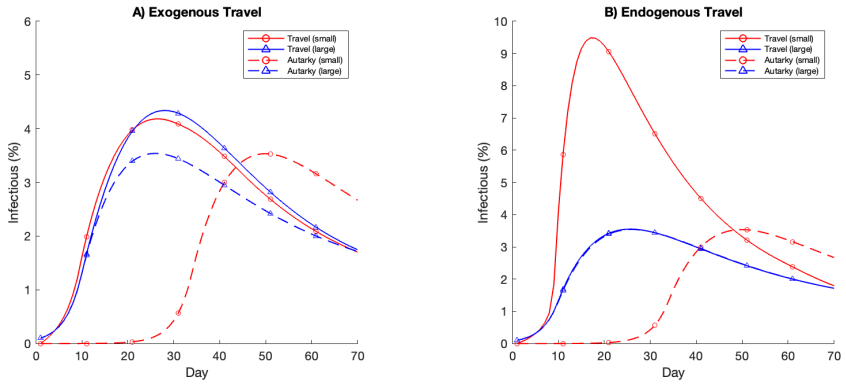

Figure 7: Infectious under alternate model specifications, contrasting home isolation autarky with exogenous (Panel A) or endogenous (Panel B) travel. In all cases, home (small) infectious seed is less than foreign (large) infectious seed $I_{0,H} < I_{0,F}$.

C.2.1 Asymmetric Autarky Economies

Table 4: Peak Infectious and Days Between Peaks under Travel Autarky

	Peak Infectious (%)		Days Between Peaks	
	$I_{0,H} = I_{0,F}$	$I_{0,H} < I_{0,F}$	$I_{0,H} = I_{0,F}$	$I_{0,H} < I_{0,F}$
Symmetric Size				
No Behavioral Response	26.52	26.52	0	24
Home Isolation	3.51	3.51	0	24
Asymmetric Size				
No Behavioral Response	26.46	26.46	0	24
Home Isolation	3.53	3.53	0	24

C.3 Public Policy under Pandemic Federalism

C.3.1 Welfare Calculation

We report welfare effects in consumption equivalent units. The value for an economy with no pandemic is

$$U^{\text{no pandemic}} = \sum_{t=0}^{\infty} \delta^t \sum_i \lambda_i \left(u(c_{i,t}, m_{i,t}) + P^{D^*} \Omega_i \right), \tag{35}$$

where $c_{i,t}, m_{i,t}$ solve (5). The value for the economy that experiences the pandemic is

$$\begin{aligned} U^{\text{pandemic}} = &\sum_{t=0}^{T} \delta^t \sum_i \left(\sum_{z \in \{S,I,R\}} z_{i,t} u^z(c^z_{i,t}, m^z_{i,t}) + D^*_{i,t} \Omega_i \right) \\ &+ \sum_{t=T+1}^{\infty} \delta^t \sum_i \lambda_i^{post} \left(u(c_{i,t}, m_{i,t}) + P^{D^*} \Omega_i \right), \end{aligned} \tag{36}$$

where $D^*_{i,t}$ are new deaths from either the virus or other causes and λ_i^{post} is the population share of i post-pandemic (i.e. the population share pre-pandemic less those that die from the virus). Define $U(\xi)$ welfare under no pandemic when consumption is adjusted by ξ,

$$U(\xi) \equiv \sum_{t=0}^{\infty} \delta^t \sum_i \lambda_i \left(u\left(c_{i,t}(1+\xi), m_{i,t}\right) + P^{D^*} \Omega_i \right) \tag{37}$$

Under each pandemic scenario, we report the value ξ (in percentage points) that makes welfare in the absence of a pandemic equal to that under the pandemic, $U(\xi) = U^{\text{pandemic}}$. A negative ξ indicates a welfare *loss* relative to the no-pandemic economy.

C.3.2 Exogenous Social Distancing Policies

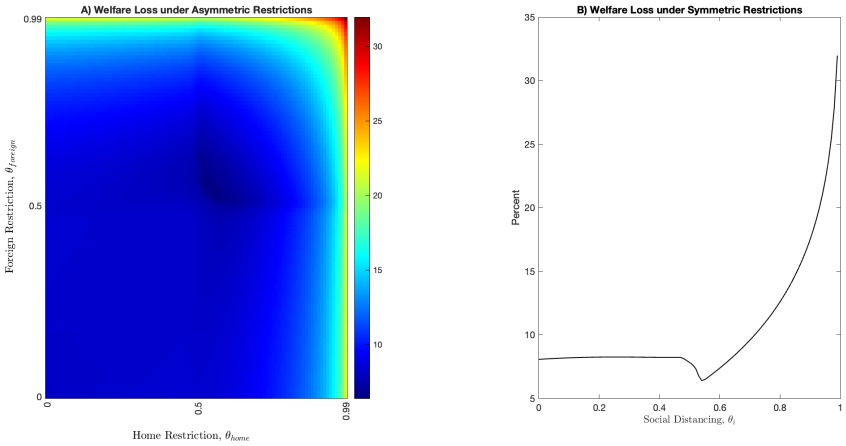

Figure 8: Welfare under different social distancing requirements - Symmetric Economy

C.3.3 Exogenous Travel Bans

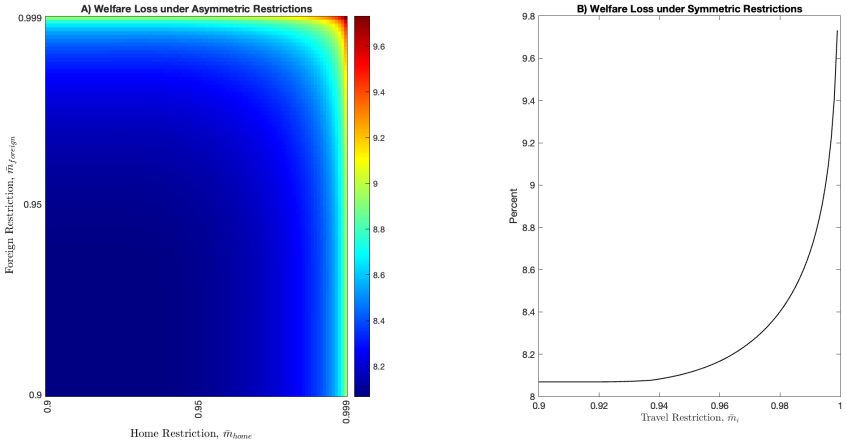

Figure 9: Welfare under different travel restrictions - Symmetric Economy

C.3.4 Nash Solution to Optimal Public Policies

Travel restriction game. We study the Nash equilibrium of the travel restriction game with a fixed policy rule when the foreign region begins with a higher initial

infectious share, $I_{foreign,0} = 0.1\%$, $I_{home,0} = 0.0001\%$. In all cases, the prisoners dilemma outcome with $\bar{m}_{home} = \bar{m}_{foreign} = 99\%$ is the unique effective Nash equilibrium. Table 5 displays equilibrium outcomes.

Table 5: Nash equilibrium outcomes under alternate infection seeding

	Peak Infectious	Welfare
Symmetric Size	3.59	-23.35
Asymmetric Size - Small	4.22	-5.03
Asymmetric Size - Large	3.53	-21.54

Social distancing game. Consider a game where local policymakers choose social distancing requirements for their own region θ_i, taking the policy of the other region as given. We allow policymakers to choose from the grid $\theta_i \in \{0, 0.25, 0.5, 0.75, 0.99\}$. That is, a reduction of time spent on economic activities of 25%, 50%, 75%, or 99%, or no restriction. In all cases, the unique Nash equilibrium is that with *no* social distancing restrictions, $\theta_i = 0$, which lead to identical outcomes to those presented in Section 5.2 under endogenous travel.

D Surrogate Models

Consider a vector $\underline{\mathbf{x}} \in \mathbb{X} \subset \mathbb{R}^n$ which summarizes the state of the economy; this vector may include the aggregate disease states (S, I, R, D) as well as model parameters (or "pseudo-states"). Now, let $y \in \mathbb{R}^m$ be a vector of outcomes of our epi-econ model associated with a particular input vector $\underline{\mathbf{x}}$; for example, these outcomes could be the cumulative welfare losses reported in the last column of Table 3. The key intuition behind the surrogate model approach is to find an estimate,

$$\hat{f} : \mathbb{X} \to \mathbb{R}^m$$

of the unknown mapping $f : \mathbb{X} \to \mathbb{R}^m$ between the input space $\mathbb{X}$ and epi-econ model outcomes $y = f(\underline{\mathbf{x}})$.

To do so, we proceed as follows. As it is not feasible to evaluate the unknown function f over the entire domain $\mathbb{X}$, we solve the epi-econ model at finitely many points, $\underline{\mathbf{x}} \in \mathbb{X}$, to construct a rich training sample of $(f(\underline{\mathbf{x}}), \underline{\mathbf{x}})$ (Subsection D.1). We then use this training sample to estimate our surrogate model, $\hat{f} : \mathbb{X} \to \mathbb{R}^m$, that is an estimator of the epi-econ vector-valued outcomes $f(\mathbb{X})$ (Subsection D.2). For our purposes, we choose different specification of our surrogate models for each outcome (Subsection D.3). In our quantitative analysis, we then leverage the estimated (trained) surrogate model, to generate predictions and conduct sensitivity analysis without the need to solve the epi-econ model additional times.

D.1 Generating the Training Sample

We study the following model parameters: infectious productivity ϕ, reproductive number R_0, duration of infectiousness d, case fatality rate μ, time 0 infectious seeds $I_{home,0}, I_{foreign,0}$, and the home population share λ_{home}.

First we use estimates of disease characteristics from the epidemiological literature collected in Table 6 to determine the boundaries of our grids. For R_0, we choose a lower bound of 1.01 ($\mathrm{R}_0 > 1$ is necessary to start a pandemic), and an upper bound of 18, corresponding to estimates for the highly infectious measles. We set the duration of infectiousness, d, within a range of 5 days (COVID-19)

Table 6: Estimates of epidemiological parameters across diseases

Disease	R_0	d	μ
Ebola	1.51-2.53 (Michigan)	6 (CDC)	0.5 (WHO)
H1N1	1.46-1.48 (Michigan)	∼8 (De Serres et al., 2010)	0.0002 (CDC)
Seasonal flu	0.9-2.1 (Michigan)	8 (CDC)	0.0001 (CDC)
Measles	12-18 (Michigan)	8 (WHO)	0.0003 (CDC)
MERS	1 (Michigan)	10+ (CA Public Health)	<0.35 (WHO)
Polio	5-7 (Michigan)	20 (ECDC)	0.05-0.15 (ECDC)
SARS	<1-2.75 (Michigan)	10+ (IL Public Health)	0.03 (WHO)
Smallpox	5-7 (Michigan)	21 (CDC)	0.3 (WHO)

Table 7: Parameter ranges for surrogate model training sample

	ϕ	R_0	d	μ	$I_{i,0}$	λ_{home}
Lower bound	0.29	1.01	5	0.0001	1e-9	1e-5
Upper bound	1	18	21	0.5	1e-5	0.5

to 21 days (smallpox). The case fatality rate μ varies from 0.0001 (seasonal flu) to 0.5 (Ebola). As noted by Ash et al., 2022, the infectious productivity ϕ must lie in the range $[d_a/d_h, 1]$, where d_a is the duration of asymptomatic and d_h is the average duration of infection conditional on being hospitalized.[49] We use Ebola, which has particularly severe symptoms, to construct the lower bound for ϕ. This implies $\phi \in [0.29, 1]$.[50] Finally, we set the bounds for the initial infectious shares to $I_{i,0} \in [1e-9, 1e-5]$ ($i \in \{home, foreign\}$), and the home population share to $\lambda_{home} \in [1e-5, 0.5]$.

Second, we use a Sobol sequence (Sobol', 1967), a low-discrepancy, quasi-random sequence, to generate the training sample on the parameter space $\mathbb{X}$ defined by Table 7.[51] Our fast solution algorithm enables us to solve the 1,000,000 in a

[49] Infectious productivity is estimated as a weighted average of the (population) share of time spent in different degrees of disease severity (asymptomatic, minor sufferers, hospitalized). The restriction $\phi \in [d_a/d_h, 1]$ ensures the relevant population shares are non-negative. See Supporting Information 2.1.1 of Ash et al., 2022 for a discussion.

[50] Per the CDC, Ebola symptoms appear 2 - 21 days after exposure. Qureshi et al., 2015 report a mean hospitalization duration for Ebola patients of 7 days. From these, we assign a lower bound for ϕ of 2/7.

[51] Low-discrepancy sequences fill the sample space more evenly than does drawing points from a uniform distribution, and have been shown to work well in high-dimensional problems (see, e.g., Kucherenko and Sytsko, 2005). In addition, they improve over a grid-based approach by providing

reasonable timeframe—approximately 24 hours for the laissez-faire economies—by parallelizing across 64 (x86_64 AMD Milan) CPU cores on the CU Boulder Alpine Supercomputer.[52]

For each model economy (parameter vector), we collect results for both the laissez-faire equilibrium (Section 5.1) and the Nash equilibrium outcome to the travel restriction game (Section 5.3).

D.2 Training of the Surrogate Models

We train surrogate models to estimate time-invariant equilibrium outcomes, such as the peak infection in region i. The surrogate models are trained as function of the seven parameters $\underline{\mathbf{x}}^{params} = [\phi, R_0, d, \mu, I_{home,0}, I_{foreign,0}, \lambda_{home}]$, and are used to make predictions for the time-invariant outcomes,

$$\widehat{\text{Time-invariant Outcome}}_i = \hat{f}_i(\underline{\mathbf{x}}^{params}).$$

D.3 Surrogate Models Specifications

Following the approach in Chen, Didisheim, and Scheidegger (2023), we employ "deep surrogates" (i.e. surrogates constructed using deep neural networks). In the construction of any neural network, the user must specify the network "hyper-parameters" such as the number of hidden layers and nodes per hidden layer. We use K-fold cross-validation to select the model architecture.[53] We compare performance across networks with two or three hidden layers, and 10, 50, or 100 nodes per layer. We also choose the regularization parameter (L2 regularization) from the set

the surrogate information on the behavior of outcomes over the entire parameter space, rather than only on specific hyperplanes.

[52] We choose the full set of 1,000,000 parameter combinations before training the surrogate model. An alternate approach involves estimating the surrogate on an initial training sample, and subsequently choosing additional sample points in regions of the parameter space where the surrogate performs poorly (see e.g. Chen, Didisheim, and Scheidegger, 2023).

[53] K-fold cross-validation proceeds as follows. For each hyperparmeter combination under consideration, the data is split into K subsets (i.e. folds), with the model trained on $K-1$ folds, and evaluated on the remaining fold. This process is repeated K times for each hyperparameter set. Those hyperparameters which minimize the (average) loss during cross-validation are chosen.

$\{1e-1, 1e-2, ..., 1e-6\}$. The ReLU activation function is used to introduce non-linearity to the network, and we train using mini-batch stochastic gradient descent (Robbins and Monro, 1951; Bengio, 2012; Azinovic, Gaegauf, and Scheidegger, 2022) and the Adam optimization algorithm (Kingma, 2014).[54]

D.4 Travel as an Effective Asset

We define travel to be an effective asset when it reduces the infection probability and is preferred to isolation as an avoidance mechanism. Formally, travel is an effective asset if:

(a) $\partial P^I_{i,t}/\partial m^S_{i,t} < 0$ and $h^S_{i,t} = 0$ OR

(b) $\partial P^I_{i,t}/\partial m^S_{i,t} < 0$ and $h^S_{i,t} > 0$ and the denominator of (22) is less than one,

$$\delta(U^S_{i,t+1} - U^I_{i,t+1}) \left[\left(1 - \tau \cdot \frac{(I_{i,t} - M^I_{i,t} + M^I_{j,t})}{N_{i,t}} \right)^{\mathscr{C}^h_{i,t}} - \left(1 - \tau \cdot \frac{(I_{j,t} - M^I_{j,t} + M^I_{i,t})}{N_{j,t}} \right)^{\mathscr{C}^m_{i,t}} \right] < 1.$$

We report the average share of time for which travel is an effective asset, conditional on global infection rates being at least one in one million (the infection seed used in Section 5). For J economies, the statistic is given by

$$X^{\text{eff. asset}} \equiv \frac{1}{J} \sum_{j=1}^{J} \frac{1}{T^*_j} \sum_{t=1}^{T} \mathbb{I}(m^S_{home,t,j} \text{ is effective}) \cdot \mathbb{I} \left(\sum_i I_{i,t} \geq 1e-6 \right),$$

where $T^*_j \leq T$ denotes the number of periods with infection rates above one in one million for economy j, the first indicator function is equal to one when travel is an effective asset as defined by conditions (a) and (b) above, and the second indicator function is equal to one if global infection rates are above the cutoff.

[54] We use mini-batches of size 512 and a learning rate of 10^{-3}. We use early stopping which withholds a subset of the training data (5%) for validation during each epoch, and ends the training process when the model performance on the validation set stops improving. This helps to prevent overfitting.

We also report the average share of time for which travel is effective, conditional on global infection rates being at least one in one million and no more than 1%. The statistic is calculated

$$X^{\text{eff. asset low inf.}} \equiv \frac{1}{J}\sum_{j=1}^{J}\frac{1}{T_j^{**}}\sum_{t=1}^{T}\left[\mathbb{I}(m^S_{home,t,j} \text{ is effective})\cdot\mathbb{I}\left(\sum_i I_{i,t}\geq 1e-6\right)\right.$$
$$\left.\cdot\mathbb{I}\left(\sum_i I_{i,t}\leq 0.01\right)\right],$$

where $T_j^{**}\leq T$ denotes the number of periods with infection rates above one in one million and below 1% for economy j.

D.5 Permutation Feature Importance

The permutation feature importance is a methodology used to evaluate the relative importance of model parameters. This model inspection technique proceeds as follows. For each of the n parameters (features) $\underline{\mathbf{x}}\in\mathbb{X}\subset\mathbb{R}^n$, the column of the dataset corresponding to parameter (feature) d is randomly shuffled. Predictions are then made using the corrupted data. The permutation importance is then

$$\text{Permutation feature importance}(d) = S(f(\underline{\mathbf{x}}),\hat{f}(\underline{\mathbf{x}})) + \left[\underbrace{-\frac{1}{K}\sum_{k=1}^{K}S(f(\underline{\mathbf{x}}),\hat{f}(\underline{\mathbf{x}}_{k,d}))}_{\text{Permutations}}\right],$$

where $\underline{\mathbf{x}}_{k,d}$ is the corrupted data and the score $S(\cdot)$ denotes the prediction accuracy of the surrogate model (higher values of the score are associated with better accuracy of $\hat{f}(\underline{\mathbf{x}})$). We select as score function the negative of the mean absolute error, the same score function used to train our surrogate model. The first term reports the score before the shuffling. The second term (in squared brackets) reports the average score across the K permutations of the d parameter (feature). If a parameter d is irrelevant (for the model outcome), its permutations do not affect its score; in this case, the Permutation feature invariance gets close to 0. Conversely, permutations of

a relevant parameter would reduce the accuracy of the surrogate model fit, reducing (on average) its score, generating a positive second term in brackets. Accordingly, a more relevant parameter is associated with larger values of its Permutation feature importance.[55]

For comparability, Table 8 lists the permutation feature importance for each outcome in Figure 3, after normalizing each row to sum to one.

Table 8: Permutation Feature Importance

	ϕ	R_0	d	μ	$I_{home,0}$	$I_{foreign,0}$	λ_{home}
Endo. Travel and Isolation							
Travel Effective	0.15	0.36	0.06	0.2	0.04	0.03	0.16
Travel Effective ($I > 1\%$)	0.13	0.37	0.07	0.21	0.04	0.04	0.15
Disease Dynamics and Behavior							
Peak Sync. - Exogenous	0.1	0.25	0.13	0.18	0.11	0.11	0.11
Peak Sync. - Endogenous	0.18	0.32	0.07	0.2	0.01	0.02	0.2
Peak Inf. - Exogenous	0.09	0.41	0.06	0.16	0.02	0.03	0.22
Peak Inf. - Endogenous	0.27	0.26	0.05	0.14	0.05	0.04	0.18
Public Policy							
Peak Infection - Nash	0.04	0.45	0.02	0.05	0.0	0.0	0.44
Welfare Loss - Nash	0.19	0.38	0.02	0.21	0.02	0.02	0.17

D.6 Partial Dependence Plot

Partial dependence plots are used to illustrate the effect of an input (epi-econ parameter) on some output generated by a "black box" algorithm (Hastie et al., 2009). Let $\underline{\mathbf{x}} \in \mathbb{X} = \mathbb{X}_1 \times \mathbb{X}_2 \times \ldots \times \mathbb{X}_d \times \ldots \times \mathbb{X}_n \subset \mathbb{R}^n$. The Partial dependence plot reports for an arbitrary level of the parameter $x_d \in \mathbb{X}_d \subset \mathbb{R}$ (horizontal axis) an estimate of

[55] Permutation feature importance may take negative values when a model is overfit to noise or the model permutations accidentally disrupt misleading patterns learned by the model. Large negative values are therefore symptoms of problems in the surrogate model estimation. For irrelevant parameters, permutation feature importance may be negative due to numerical rounding factors. When Permutation feature importance$(d) < 0$ and $|\text{Permutation feature importance}(d)| < 1e-6 \cdot \sum_k |\text{Permutation feature importance}(k)|$, we set the permutation feature importance for parameter d to zero for computing the normalized values in Table 8.

the partial dependence of the model outcomes $f(x_d, \underline{\mathbf{x}}^C)$ on x_d,

$$\text{Partial dependence}(x_d) \equiv E\left[f(x_d, \underline{\mathbf{x}}^C)\right],$$

where the expectation is taken with respect to the complement parameter inputs, $\underline{\mathbf{x}}^C$, $\underline{\mathbf{x}}^C \equiv [x_1, ..., x_{d-1}, x_{d+1}, ..., x_n]$. This expectation is estimated for each value of x_d by

$$\widehat{\text{Partial dependence}}(x_d) \equiv \frac{1}{J}\sum_{j=1}^{J} \hat{f}(x_d, \underline{\mathbf{x}}_j^C),$$

where J is the number of economies, $\hat{f}(x_d, \underline{\mathbf{x}}_j^C)$ is the value of the estimated surrogate model at (an arbitrary) $x_d \in \mathbb{X}_d$ and $\underline{\mathbf{x}}_j^C$, where j indexes all occurrences of $\underline{\mathbf{x}}^C$ in the training data. The Partial dependence then measures the effect of parameter x_d on the model outcome, taking into account the average effect of all other parameters, $\underline{\mathbf{x}}^C$.

E Further Robustness

This section provides further robustness tests.

E.1 Exposed disease state

We consider an extension of the model by adding a fifth disease state, "exposed," to the SIRD model. These exposed agents are pre-symptomatic and do not realize they have become exposed. They therefore make decisions as though they were susceptible. Following an incubation period, exposed individuals will then develop symptoms and become infectious. The SEIRD system governing the transitions

across disease states becomes:

$$
\begin{aligned}
S_{i,t+1} &= \left(1 - \mathscr{P}_i^{SI}(\mathscr{A}_t, \mathscr{T}_t) - P^{D^*}\right) S_{i,t} + \gamma\left(S_{i,t} + E_{i,t} + I_{i,t} + R_{i,t}\right), \\
E_{i,t+1} &= \left(1 - P^{D^*} - P^I\right) E_{i,t} + \mathscr{P}_i^{SI}(\mathscr{A}_t, \mathscr{T}_t) S_{i,t}, \\
I_{i,t+1} &= \left(1 - P^{D^*} - P^R - P^D\right) I_{i,t} + P^I E_{i,t}, \\
R_{i,t+1} &= \left(1 - P^{D^*}\right) R_{i,t} + P^R I_{i,t}, \\
D_{i,t+1} &= D_{i,t} + P^D I_{i,t} + P^{D^*}\left(S_{i,t} + E_{i,t} + I_{i,t} + R_{i,t}\right),
\end{aligned}
$$

where P^I is the share of exposed agents who become infectious each period, given by $P^I = 1/d_a$, and d_a is the average duration an agent spends pre-symptomatic. Following Ash et al., 2022, we set $d_a = 5.2$.

The presence of exposed agents creates a delay in the timing of peak infections and lowers the peak infection rate, though the qualitative results are otherwise similar. To illustrate, we recreate Table 2 for a model including the exposed disease state. Results are shown in Table 9.

Table 9: Peak Infectious and Days Between Peaks for Models with Exposed Disease State

	Peak Infectious - Home (%)		Days Between Peaks	
	$I_{0,H} = I_{0,F}$	$I_{0,H} < I_{0,F}$	$I_{0,H} = I_{0,F}$	$I_{0,H} < I_{0,F}$
Autarky				
No Behavioral Response	12.95	12.95	0	58
Home Isolation	3.14	3.14	0	58
Symmetric Size				
Exogenous Travel	3.24	3.28	0	4
Endogenous Travel	3.19	3.18	0	8
Asymmetric Size				
Exogenous Travel	3.60	3.61	4	4
Endogenous Travel	8.35	8.39	11	11

E.2 Alternative home exposure risk

We consider an alternative formulation for the exposure probability under which susceptible agents do not meet tourists when isolating. Under the alternate specification, (11) is replaced by

$$P_i^E(m_t^S, l_t^S, h_t^S; \mathscr{T}_t) \equiv 1 - \Bigg[\underbrace{\left(\frac{l_t^S}{1-\theta_i}\right)}_{\text{Work share}} \left(1 - \underbrace{\tau \cdot \frac{(I_{i,t} - M_{i,t}^I + M_{j,t}^I)}{N_{i,t}}}_{\text{Home Exposure}} \right)^{\mathscr{C}_{i,t}^l}$$

$$+ \underbrace{\left(\frac{m_t^S}{1-\theta_i}\right)}_{\text{Travel share}} \left(1 - \underbrace{\tau \cdot \frac{(I_{j,t} - M_{j,t}^I + M_{i,t}^I)}{N_{j,t}}}_{\text{Foreign Exposure}} \right)^{\mathscr{C}_{i,t}^m} + \underbrace{\left(\frac{h_t^S}{1-\theta_i}\right)}_{\text{Isolation share}} \left(1 - \underbrace{\tau \cdot \frac{(I_{i,t} - M_{i,t}^I)}{N_{i,t}^*}}_{\text{Home Exposure}} \right)^{\mathscr{C}_{i,t}^h} \Bigg].$$

The relevant i populations become

$$N_{i,t} = \sum_{Z \in \{S,I,R\}} Z_{i,t} - M_{i,t}^Z - H_{i,t}^Z + M_{j,t}^Z, \tag{38}$$

$$N_{i,t}^* = \sum_{Z \in \{S,I,R\}} Z_{i,t} - M_{i,t}^Z, \tag{39}$$

where $H_{i,t}^Z = h_{i,t}^Z Z_{i,t}$ denotes the population isolating. Finally, average consumption (14) becomes

$$C_{i,t}^* \equiv \sum_{Z \in \{S,I,R\}} \left(\underbrace{c_{i,t}^Z \frac{Z_{i,t} - M_{i,t}^Z - H_{i,t}^Z}{N_{i,t}}}_{\text{Consumption by } i \text{ agents}} + \underbrace{\kappa \frac{M_{j,t}^Z}{N_{i,t}}}_{\text{Consumption by } j \text{ agents}} \right).$$

Table 10 recreates the results presented in Section 5.2 under the alternative home exposure risk.

Table 10: Peak Infection and Days Between Peaks for Models with Alternative Home Exposure Risk

	Peak Infectious - Home (%)		Days Between Peaks	
	$I_{0,H} = I_{0,F}$	$I_{0,H} < I_{0,F}$	$I_{0,H} = I_{0,F}$	$I_{0,H} < I_{0,F}$
Autarky				
No Behavioral Response	26.52	26.52	0	24
Home Isolation	7.47	7.47	0	24
Symmetric Size				
Exogenous Travel	9.21	9.36	0	0
Endogenous Travel	8.26	6.94	0	7
Asymmetric Size				
Exogenous Travel	6.93	6.97	2	2
Endogenous Travel	8.45	8.49	7	7

E.3 Solving with the Fischer-Burmeister function

The first order and Kuhn-Tucker conditions for the susceptible problem are

$$\frac{-(1-\beta_i)}{1-\theta_i-m-h} + \frac{\beta_i}{m} + \delta(U^{I'} - U^{S'})\frac{\partial P_i^I}{\partial m} - \mu_m^{hi} = 0, \tag{40}$$

$$\frac{-(1-\beta_i)}{1-\theta_i-m-h} + \delta(U^{I'} - U^{S'})\frac{\partial P_i^I}{\partial h} + \mu_h^{lo} - \mu_h^{hi} = 0, \tag{41}$$

$$\mu_m^{hi}(\bar{m} - m) = 0, \tag{42}$$

$$\mu_h^{lo} h = 0, \tag{43}$$

$$\mu_h^{hi}(\bar{h} - h) = 0. \tag{44}$$

Our solution algorithm solves for the endogenous choices directly using the FOCs. To verify the solution accuracy (especially close to the constraints), we implement an alternate routine which solves the above system of equations and compare results. In particular, we replace (42)-(44) with their equivalent representations using

the Fischer-Burmeister function (see e.g. Maliar, Maliar, and Winant, 2021):

$$\left(\mu_m^{hi} + (\bar{m} - m) - \sqrt{(\mu_m^{hi})^2 + (\bar{m} - m)^2}\right)^2 = 0,$$
$$\left(\mu_h^{lo} + h - \sqrt{(\mu_h^{lo})^2 + h^2}\right)^2 = 0,$$
$$\left(\mu_h^{hi} + (\bar{h} - h) - \sqrt{(\mu_h^{hi})^2 + (\bar{h} - h)^2}\right)^2 = 0.$$

The latter have an advantage in that they are smooth approximations of the Kuhn-Tucker conditions, useful when using a nonlinear solver (especially one which does not allow constraints).

We solve the modified system of equations using the Matlab function fsolve. The two algorithms obtain a maximum difference in the aggregate disease states (share of the population) of 0.0000003.

E.4 Restricted Range on R_0

We confirm the robustness of our results in Section 6 for a restricted range of the reproductive number, R_0. Specifically, we generate one million new parameter combinations (training sample), with $R_0 \in [1.01, 6]$ (the other parameter ranges are left according to the bounds in Table 7). We then train surrogate models on the new (restricted) data. Figure 10 uses these trained surrogates to recreate Figure 3 in the main text under the restricted range on R_0.

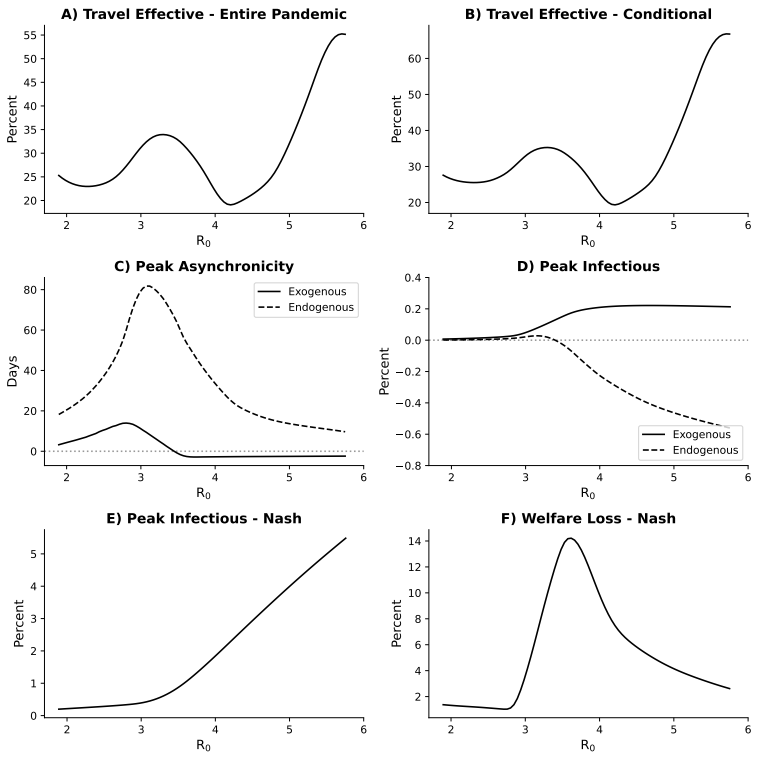

Figure 10: Partial dependence plot of model outcomes (vertical axis) on R_0 (horizontal axis) for $R_0 \in [1.01, 6]$. A) Average percent of days travel is an effective avoidance asset. B) Average percent of days travel is an effective avoidance asset when the infectious population share is above 1%. C) Change in the time between home and foreign peaks from a home isolation autarky model to models with exogenous (solid line) or endogenous (dashed line) travel. D) Percentage point change in peak infectious (percent of population) from a home isolation autarky model to models with exogenous (solid line) or endogenous (dashed line) travel. E) Peak infectious under the aggregate-welfare-minimizing Prisoner's Dilemma. F) Change in welfare (consumption equivalent units) from the aggregate-welfare-maximizing coordinated solution to the aggregate-welfare-minimizing Prisoner's Dilemma.