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KEVEN ROGER ALVES ANDRÉ

THE ROLE OF NETWORK IN THE SIR MODEL

FORTALEZA
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Dissertação apresentada ao Programa de Pós-Graduação em Economia da Universidade Federal do Ceará, CAEN/UFC, como requisito parcial à obtenção do título de Mestre em Economia.
Área de concentração: Macroeconomia.
Orientador: Prof. Dr. Márcio Veras Corrêa
Coorientador: Prof. Dr. Marcelo Aarestrup Arbex

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BANCA EXAMINADORA

_____

Márcio Veras Corrêa (Orientador)
Universidade Federal do Ceará - UFC

_____

Marcelo Aarestrup Arbex (Coorientador)
Universidade de Windsor

_____

Sidney Martins Caetano
Universidade Federal de Juiz de Fora - UJFJ

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Resumo

Modelos matemáticos têm sido amplamente utilizados para simular a dinâmica da proliferação de doenças infecciosas, bem como para estudar propostas de políticas públicas de contenção. O objetivo desse trabalho é estudar como uma estrutura de network pode determinar a evolução de uma epidemia. Para tal, será usado o modelo macroeconômico Suscetível-Infectado-Recuperado (SIR) na presência de um ambiente de network. Em nosso modelo epidemiológico, a estrutura de network é um importante motor da proliferação do vírus. Intuitivamente, pessoas mais conectadas no círculo social são os principais vetores do virus. Por outro lado, aquelas pessoas com poucas conexões estão menos expostas à doença. Será estudado o comportamento da pandemia para diferentes tipos de network, desde uma pouco conectada até outra muito conectada. É encontrada exatamente a relação esperada: como economias mais conectadas (economias com um número médio maior de links) espalham o vírus mais rapidamente, elas experimentam consequências mais duras em um cenário pandêmico, tais como uma maior queda do consumo e horas trabalhadas agregados devido tanto ao maior número de mortes quanto ao maior esforço dos agentes suscetíveis para ficar em casa e evitar contatos físicos. Os agentes suscetíveis são mais cautelosos em relação à decisão de seu nível de consumo e de horas trabalhadas à medida que a economia é mais socialmente conectada, pois as consequências de sair de casa para consumir ou trabalhar são maiores em economias mais conectadas devido ao seu maior número de pessoas infectadas.

Palavras-chave: Epidemia, COVID-19, recessões.

Abstract

Mathematical models have been often used to simulate the dynamics of the spread of infectious disease, as well as to test containment public policy proposals. The goal of this work is to study how a network structure can determine the evolution of an epidemic. For that, we use a Susceptible-Infected-Recovered (SIR) macroeconomic model in the presence of a network environment. Network models have been important in the job search discussion. In our epidemiological model, the network structure is an important cause of the spread of the disease. Intuitively, more connected people in the social circle are the main vector of the virus. On the other hand, those people with few connections should be less exposed to the disease. We study the behavior of the pandemic for different types of network, from a low connected one to a high connected one. We find exactly the expected relationship: because more connected economies (economies with a higher average number of links) spread the virus faster, they face harder consequences in a pandemic scenario, such as a greater fall on aggregate consumption and hours worked due to both the higher number of deaths and the susceptible agents' higher attempt to stay at home and avoid physical contacts. Susceptible agents are more cautious in regard to the decision of their level of consumption and hours worked as the economy becomes more socially connected, once the consequences of leaving home to consume or to work are harder in the higher connected economy because of its higher number of infected people.

Keywords: Epidemic, COVID-19, recessions.

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1 INTRODUCTION

In this paper, we extend the SIR-macro model proposed by Eichenbaum et al. [2020] to study the implications and the role of the social network on the evolution of a pandemic, and the changes caused by the strengthening and the weakening of such social network. Discovering the role of the social network in the behavior of the pandemic is quite important for the choice of political measures to contain the spread of the virus.

In the economic field, the COVID-19 pandemic represents the worst economic crisis since the Great Depression (Gopinath [2020]). Given the rapid spread of COVID-19, countries across the World have adopted several public health measures intended to prevent its spread, including social distancing. The virus is the same, but its effects, frequently devastating, has been quite heterogeneous between countries and regions by reasons that, in the moment, are not clear enough. In the economic field, the effects are also heterogeneous, but in this case the reasons may be easier to be identified. How should countries with different fiscal situation, or different productivity, react?

Not only we must observe the economic features of a country, but also its level of social connection. A contact network is acknowledged to play a key role at the dynamics of infectious diseases and other transmission phenomena. The same question arises: how should a highly (socially) connected country react? Does it change when the country is lowly connected? These questions suggest that, despite the universality of the virus, the macroeconomics of an epidemic depends on local characteristics.

As also found by Eichenbaum et al. [2020], in our model, people's decisions to cut back on consumption and work reduce the severity of the pandemic, as measured by total deaths. These same decisions exacerbate the size of the recession caused by the pandemic. Another similarity between their result and ours is that a pandemic has both aggregate demand and aggregate supply effects. The supply effect arises because the epidemic exposes workers to the virus. Workers react to that risk by reducing their labor supply. The demand effect arises because the epidemic exposes consumers to the virus. Consumers react to that risk by reducing consumption. The supply and demand effects work together to generate a large, persistent recession.

Our main contribution to the discussion of the macroeconomics of epidemics is the implications of the social network on the spread of the virus. In particular, we simulate the model for different social networks with respect to its average size, from a less connected one to a more connected one. Results show that the economies that present more connections in the social network face a more severe pandemic, with more deaths. Meanwhile,

the less connected economy experimented a less severe pandemic, with lesss deaths.

The SIR model will help us answer those questions. That is a model widely used by epidemiologists, and was initially developed by Kermack and McKendrick [1927]. It proposes three health status: susceptible, infected and recovered. Susceptible people can contract the virus through interaction with the infected ones. Infected people can transmit the virus, and also can either dye or recover. Recovered people are immune and can no longer transmit the virus. The infection, recovery e mortality rate are the main parameter of such model. The idea is that, with a sufficient time, the society acquires the ”herd immunity” as the susceptible population falls.

A feature of epidemiological models is that the transitions between the health status are exogenous with respect to the economic variables. In other words, the expected fall on consumption and hours worked are not considered in the SIR models. That is a problem, for one of the main discussions in the course of an epidemic is the trade-off lives x economy: people reduce their economic activities aiming to reduce the odds of infection. That been said, the challenge is to study the efficiency of this trade-off, *i.e.*, how to reduce the infection rate at the lowest economic costs[1].

Eichenbaum et al. [2020] discuss this question combining a general equilibrium model with the standard SIR model. In their SIR-macro model, the number of infections depends on the level of interaction between the agents when they consume and work, and on other residual ways of infection. Therefore the susceptible population can reduce the odds of infection by reducing their consumption level and hours worked. The competitive equilibrium, however, is not Pareto efficient, for the infected and recovered agents do not take into account that their actions influence the other agents' infection and mortality rates.

Bethune and Korinek [2020] focus on this type of externality. The authors develop Susceptible-Infected-Susceptible (SIS) and SIR models to quantify the externalities of infection using both decentralized and social planner approaches. They find that, in a decentralized approach, the infected agents keep engaged in economics activities in order to maximize utility, while the susceptible agents reduce their activities to reduce the odds of infection. With a social planner approach, the planner forcefully reduces the infected agents' activities to mitigate the risk of the susceptible agents.

Another point of using this models is that a fraction of the infected population could be asymptomatic, and, without realizing it, they could increase the level of infection. Berger et al. [2020] study this incomplete information using a Susceptible-Exposed-Infected-Recovered (SEIR) model based on Kermack and McKendrick [1927]. They work on the idea of increasing the tests in the susceptible population to identify asymptomatic infected patients

[1]For a complete literature review of the economics of COVID-19, see Brodeur et al. [2021].

looking to isolated that part of the population. The authors find that such directed quarantine policy softens the negative impact of the pandemic on the economy and reduces the peak of infection.

Some countries adopted the vertical isolation for the risk group. Acemoglu et al. [2020] study this question with a multi-risk SIR model (MR-SIR) and divide the population in different groups (young, middle-aged and old), with different infection, hospitalization and mortality rates. Those conditions allow the possibility of vertical quarantine policy. The heterogeneous lockdown across different groups, been more severe for the higher risk group (the old), can reduce both the number of lives lost and the economic recession when compared with horizontal lockdowns.

With respect to the Brazilian economy, Rabelo and Soares [2020] exploit the model proposed by Eichenbaum et al. [2020] calibrating the parameters with Brazilian data. They found that an optimal social containment policy causes a larger recession in the short run than in the case where no measure of social containment are taken. On the other hand, the optimal containment policy has the ability to save about 50 thousand lives.

Borelli and Góes [2020] uses the same model study the implications of COVID-19 on the states of São Paulo, Amazonas, Ceará, Rio de Janeiro and Pernambuco. Results point to great heterogeneity, which suggests that each state may require specific measures. São Paulo is the state whose infected population reaches the higher fraction of its total population in the competitive equilibrium between those five states studied. Meanwhile, the macroeconomic schocks are more severe at Ceará and Pernambuco.

Our main contribution is to incorporate irregular networks into a SIR-model of infectious disease. In particular, by acknowledging the role of heterogeneity in network connections, we provide one reason that agents are not equally likely to receive the virus through their links. This article provides a framework to analyze the dynamics of the epidemic and the optimal behaviour of the economic agents as driven by (non-observable) agent heterogeneity in social connections.

Besides this introduction, this paper is organized in three additional sections. Section 2 presents the model. In Section 3 we discuss the calibration of the parameters. In Section 4 we present the results for a calibrated version of the model and conduct counterfactual analyses. Section 5 concludes.

2 MODEL

The model proposed by this work intends to unit both an epidemiological model,

following Eichenbaum et al. [2020], and a network model, following Arbex et al. [2019].

The economy is populated by a continuum of people with unit measure. When the virus is introduced in the society, the population is divided into four groups: susceptible (people who have not yet been exposed to the disease), infected (people who contracted the disease), recovered (people who survived the disease and acquired immunity), and deceased (people who died from the disease). The fractions of people in these four groups are denoted by m_t^S, m_t^I, m_t^R, and m_t^D, respectively. The number of newly infected people is denoted by T_t.

Susceptible people can become infected in two ways. First, they can meet infected people while purchasing consumption goods. Second, susceptible and infected people can meet at work. Hence, the more they consume and work, the more risk they will take. Moreover, we allow for heterogeneity on the risk of infection according to the number of peers an agent has: Having many links is associated with a higher risk of infection.

2.1 Demography, Network and Transmission

In this model, the agents are connected to one another in a social network, whose structure is exogenous. Each agent may have peers to whom she passes the virus when infected, and from whom she may receive the virus when susceptible. A network is described by a *degree distribution* $\{D_z\}_{z=1}^{\infty}$, where D_z is the proportion of agents who have $z \in [1, \infty)$ peers, and is given by $(a-1)z^{-a}$, where the power-law exponent a determines how heavy the tail of the distribution is, that is, how common are nodes with much higher than the mean number of pears.

The number of links will be centrally important, and so to clarify notation, we will basically refer to the number of links with two names depending on the role of the agent in the network when we refer to her. When we use z to denote her number of peers, the agent is a generic one. When we denote the type as s, it will refer to the number of links belonging to a peer of one of these agents.

The probability a given peer has s links is $\psi_s = (sD_s)/\langle z \rangle$, where $\langle z \rangle = \int_{z=1}^{\infty} (zD_z)\, dz$ is the *average degree* in the network. Note that $\psi_s \neq D_s$, i.e., the probability one of your peers has s links is not equal to the proportion of the population that has s links. This is because those with more peers are more likely to be connected to the agent whose problem we solve. Each agent contacts susceptible friends with probabilities ρ_t^c via consumption, and ρ_t^n via work. The infection rate among those agents with s links is $m_{s,t}^I$.

The rate at which the virus is passed from infected agents to their susceptible peers

depends on how much they consume (c_t^I and c_t^S): $\varphi(c_t^I, c_t^S) = (m_t^S c_t^S + m_t^I c_t^I)^{1-\lambda_c}$. Similarly, such rate also depends on how much they work (n_t^I and n_t^S): $\varphi(n_t^I, n_t^S) = (m_t^S n_t^S + m_t^I n_t^I)^{1-\lambda_n}$. Here, λ_c and λ_n measure the efficacy of this technology.

For a given agent, the joint probability another agent of type s is infected, meet her susceptible friends, transmits the virus, and is a peer is $m_{s,t}^I \rho_t^c \varphi(c_t^I, c_t^S)\psi_s$. The total probability of such an event integrates over all possible s. Thus, the probability a susceptible agent receives the virus from a peer is

$$\Omega_t^c = \int_{s=1}^{\infty} m_{s,t}^I \rho_t^c \varphi(c_t^I, c_t^S)\psi_s ds = m_t^I \rho_t^c \varphi(c_t^I, c_t^S), \tag{1}$$

$$\Omega_t^n = \int_{s=1}^{\infty} m_{s,t}^I \rho_t^n \varphi(n_t^I, n_t^S)\psi_s ds = m_t^I \rho_t^n \varphi(n_t^I, n_t^S), \tag{2}$$

via consumption and via work, respectively.

Hence, the probability a susceptible agent of type z receives the virus from at least one peer is

$$p_t^c = 1 - (1 - \Omega_t^c)^z, \tag{3}$$

$$p_t^n = 1 - (1 - \Omega_t^n)^z, \tag{4}$$

via consumption and via work, respectively.

The aggregate probability susceptible agents of different types z receive the virus via their network is

$$P_t^c = \int_{z=1}^{\infty} p_t^c D_z dz, \tag{5}$$

$$P_t^n = \int_{z=1}^{\infty} p_t^n D_z dz, \tag{6}$$

via consumption and via work, respectively.

2.2 SIR model

Epidemiology models generally assume that the probabilities governing the transition between different states of health are exogenous with respect to economic decisions. Eichenbaum et al. [2020] modify the canonical SIR model proposed by Kermack and McKendrick [1927] so that these transition probabilities depend on people's economic decisions. Since purchasing consumption goods or working brings people into contact with each other, they assume that the probability of becoming infected depends on these

activities.

We modify the the SIR-macro model proposed by Eichenbaum et al. [2020] in order to account for the social network interactions. The social network plays a significant role in the evolution of an epidemic, since the transmission of the virus depend on physical connections between people.

The population is divided into four groups: susceptible (people who have not yet been exposed to the disease), infected (people who contracted the disease), recovered (people who survived the disease and acquired immunity), and deceased (people who died from the disease). The fractions of people in these four groups are denoted by m_t^S, m_t^I, m_t^R, and m_t^D, respectively. The number of newly infected people is denoted by T_t.

Susceptible people can become infected in two ways. First, they can meet infected people while purchasing consumption goods. Second, susceptible and infected people can meet at work. The equation that describes the number of new infections through consumption and work changes in our model, when compared to Eichenbaum et al. [2020], and is given by:

$$T_t = \left(m_t^S P_t^c\right)^\gamma \left(m_t^S P_t^n\right)^{(1-\gamma)} = m_t^S \left(P_t^c\right)^\gamma \left(P_t^n\right)^{(1-\gamma)}, \tag{7}$$

where γ is the relative weight of infection via consumption. The T_t variable is driven by the network environment that we presented in the subsection 2.1.The term $m_t^S P_t^c$ represents the number of newly infected people through consumption activities, while $m_t^S P_t^n$ represents the number of newly infected people through meetings at work.

The number of susceptible people in the next period, $t+1$, equals the number of susceptible people at the current time, t, discounted by the number of susceptible people who got infected at time t:

$$m_{t+1}^S = m_t^S - T_t. \tag{8}$$

The number of infected people in the next period, $t+1$, equals the number of infected people at the current time, t, added to the number of newly infected people, T_t, subtracted by the number of infected people that recovered, $\pi_R m_t^I$, and the number of infected people who died, $\pi_D m_t^I$:

$$m_{t+1}^I = (1 - \pi_R - \pi_D) m_t^I + T_t. \tag{9}$$

The parameter π_R is the rate at which infected people recover from the disease and π_D is the mortality rate.

The timing convention implicit in Equation (9) is the same as in Eichenbaum et al. [2020]. Social interactions happen in the beginning of the period (infected and susceptible people meet). Then, changes in health status unrelated to social interactions (recovery or death) occur. At the end of the period, the consequences of social interactions materialize:

T_t susceptible people become infected.

The number of recovered people in the next period, $t+1$, is the number of recovered people at the current time, t, added to the number of infected people that just recovered, $\pi_R m_t^I$:

$$m_{t+1}^R = m_t^R + \pi_R m_t^I. \tag{10}$$

The number of deaths in the next period, $t+1$, is the number of deceased people at the current time, t, added to the number of new deaths, $\pi_D m_t^I$:

$$m_{t+1}^D = m_t^D + \pi_D m_t^I. \tag{11}$$

Total population in the next period, Pop_{t+1}, equals the total population at the current time, Pop_t, minus the number of new deaths:

$$Pop_{t+1} = Pop_t - \pi_D m_t^I, \tag{12}$$

with $Pop_0 = 1$.

Like Eichenbaum et al. [2020], we assume that, at time zero, a fraction ε of susceptible people is infected by a virus through zoonotic exposure, that is, the virus is directly transmitted from animals to humans:

$$m_0^I = \varepsilon,$$

$$m_0^S = 1 - \varepsilon.$$

Everybody is aware of the initial infection and understands the laws of motion governing population health dynamics.

2.3 Behavior of the economic agents

Now, we describe the optimization problem of different types of agents in the economy. The variable V_t^J denotes the time-t value function of a type-J person (J=S, I, R). The budget constraint a type-J agent faces is given by

$$(1+\mu_{ct})c_t^J = (1-\mu_{nt})w_t^J n_t^J + \Gamma_t, \tag{13}$$

where c_t^J and n_t^J denote the consumption and hours worked of a type-J person, respec-

tively. Moreover, w_t^J denotes the real wage of the type-J person, μ_{ct} and μ_{nt} are Pigouvian taxes rates on consumption and work[2], respectively, and Γ_t denotes lump-sum transfers from the government.

To simplify things, we assume that the instantaneous utility of a type-J agent is given by

$$u\left(c_t^J, n_t^J\right) = lnc_t^J - \frac{\theta}{2}(n_t^J)^2. \tag{14}$$

The value function of the susceptible agent is given by

$$V_t^S = \max_{c_t^S, n_t^S, \tau_t^S} \left\{ u\left(c_t^S, n_t^S\right) + \beta[(1-\tau_t)V_{t+1}^S + \tau_t V_{t+1}^I] \right\}, \tag{15}$$

where the variable τ_t is the probability a susceptible agent becomes infected:

$$\tau_t = \frac{T_t}{m_t^S} = \left(P_t^c\right)^{\gamma} \left(P_t^n\right)^{(1-\gamma)}. \tag{16}$$

This means that the susceptible agent internalizes the fact that she can reduce the probability of getting infected by consuming less and working less.

The first order conditions for consumption, hours worked and τ_t are, respectively,

$$u_1(c_t^S, n_t^S) - \lambda_{bt}^S(1+\mu_{ct}) + \lambda_{rt}\gamma \left(\frac{P_t^n}{P_t^c}\right)^{1-\gamma} \frac{\partial P_t^c}{\partial c_t^S} = 0, \tag{17}$$

$$u_2(c_t^S, n_t^s) + \lambda_{bt}^S w_t^S(1-\mu_{nt}) + \lambda_{rt}(1-\gamma) \left(\frac{P_t^c}{P_t^n}\right)^{\gamma} \frac{\partial P_t^n}{\partial n_t^S} = 0, \tag{18}$$

$$\beta(V_{t+1}^I - V_{t+1}^S) - \lambda_{rt} = 0, \tag{19}$$

where λ_{bt}^S and λ_{rt} are the Lagrange multipliers associated with constraints (13) and (16), respectively.

In a non-epidemic economy, the third term of both equations (17) and (18) would not exist. In our model, those terms are the mechanism with which the agents internalize the risks of becoming infected that they take by adopting a given level of consumption and hours worked. Therefore, equations (17) and (18) represent the trade-off between the utility the agents acquire directly by consuming and indirectly by working and the risks they take of becoming infected by consuming and working.

A third term, in this sense, yet with another shape, also appears in Eichenbaum et al. [2020]. What differs ours from theirs is that, in this model, the social network is internalized.

[2]This is a subtle difference from Eichenbaum et al. [2020], since they do not account for a tax on work.

The value function of the infected agent is

$$V_t^I = \max_{c_t^I, n_t^I} \{u\left(c_t^I, n_t^I\right) + \beta\left[\left(1 - \pi_R - \pi_D\right) V_{t+1}^I + \pi_R V_{t+1}^R\right]\}. \tag{20}$$

The expression for V_t^I embodies a common assumption in macro and health economics that the cost of death is the forgone utility of life.

The first order conditions for consumption and hours worked are, respectively,

$$u_1(c_t^I, n_t^I) - \lambda_{bt}^I(1 + \mu_{ct}) = 0, \tag{21}$$

$$u_2(c_t^I, n_t^I) + \lambda_{bt}^I w_t^I(1 - \mu_{nt}) = 0, \tag{22}$$

where λ_{bt}^I is the Lagrange multiplier associated with constraint (6).

Note that the infected agent does not have the third term in their first order conditions like the susceptible agent does. That is because infected people do not take any risk when they consume or work, once they are already infected. Therefore, in this model, infected people do not internalize the risks of consuming and working simply because they take no risks at all. They same applies to the recovered agent. In the results section, we show that the behavior of this agents corresponds to this analysis.

The value function of the recovered agent is

$$V_t^R = \max_{c_t^R, n_t^R} \{u\left(c_t^R, n_t^R\right) + \beta V_{t+1}^R\}. \tag{23}$$

The first order conditions for consumption and hours worked are, respectively,

$$u_1(c_t^R, n_t^R) - \lambda_{bt}^R(1 + \mu_{ct}) = 0, \tag{24}$$

$$u_2(c_t^R, n_t^R) + \lambda_{bt}^R w_t^R(1 - \mu_{nt}) = 0, \tag{25}$$

where λ_{bt}^I is the Lagrange multiplier associated with constraint (6).

There is a continuum of competitive representative firms of unit measure that produce consumption goods (C_t) using hours worked (N_t) according to the production function:

$$C_t = AN_t.$$

The aggregate hours worked of this economy is defined as

$$N_t = N_t^S \phi^S + N_t^I \phi^I + N_t^R \phi^R,$$

with $N_t^S = m_t^S n_t^S$, $N_t^I = m_t^I n_t^I$ e $N_t^R = m_t^R n_t^R$. Moreover, ϕ^S, ϕ^I and ϕ^R denote the labor productivity of susceptible, infected and recovered agents, respectively. It is equal to one for susceptible and recovered people $(\phi^S = \phi^R = 1)$ and less than one for infected people $(\phi^I < 1)$.

In order to maximize its time-t profits, the firm chooses hours worked:

$$\Pi_t = AN_t - w_t^S N_t^S - w_t^I N_t^I - w_t^R N_t^R.$$

The first order condition is

$$A\phi^J = w_t^J.$$

The government's budget constraint is given by

$$\mu_{ct}\left(m_t^S c_t^S + m_t^I c_t^I + m_t^R c_t^R\right) + \mu_{nt}\left(m_t^S w_t^S n_t^S + m_t^I w_t^I n_t^I + m_t^R w_t^R n_t^R\right) = \Gamma_t\left(m_t^S + m_t^I + m_t^R\right).$$

In equilibrium, the government constraint is satisfied and each agent solves her maximization problem. In addition, both the goods market and labor market clear:

$$m_t^S c_t^S + m_t^I c_t^I + m_t^R c_t^R = AN_t.$$

$$m_t^S n_t^S + m_t^I n_t^I + m_t^R n_t^R = N_t.$$

We describe the algorithm for computing the equilibrium in the A appendix.

3 PARAMETERIZATION

In this section, we present how we assign values to model parameters. For parameter that are specific to the Brazilian economy, we use Brazilian data or rely on the Brazilian literature. For parameters that are not specific to the Brazilian economy - *e.g.*, the productivity of the infected agent (ϕ^i) -, we rely on the international literature. The parameter values are shown by Table 1.

First, each period of the model corresponds to a week. Moreover, we follow Atkeson [2020] in assuming that it takes 18 days to either recover or to die from the disease. This means that the daily probability to recover or to die, given by $\pi^R + \pi^D$, should equal $1/18$. But, because our model is weekly, we will set $\pi^R + \pi^D = 7/18$.

We do acknowledge the existence of heterogeneity in life expectancy and efficiency of health systems between different countries. Therefore, we must consider probabilities

of dying and recovering from the disease that are adapted to the Brazilian context. For the value of π^D, we follow Rabelo and Soares [2020], that weighted Brazilian population age groups by the correspondent mortality rate in South Korea. They dropped the population aged more than 70 years, once their job market participation is relatively low, to finally get a (daily) mortality rate of 0,3%. Converting to weekly rate, we have $\pi^D = 7 \times 0.003/18$. π^R is given residually: $\pi^R = 7/18 - \pi^D$.

The technological parameters A and θ were chosen to match, at the pre-epidemic steady state, the number of weekly hours worked in Brazil at 2020 (39.1 hours per week) and the Brazilian weekly income *per capita* of 2020 (BRL $1,380.00/4$). For that, we use the average hours worked per week of people aged 14 years old or older and the real average monthly income *per capita*. We obtain the average number of hours worked from SIDRA[3], from IBGE (Brazilian Institute of Geography and Statistics) [4] and the weekly *per capita* income at 2020 from the National Household Sample Survey (PNAD), from IBGE.

Just like Eichenbaum et al. [2020], we calibrate the value of the parameter that controls the relative productivity of the infected population, ϕ^I, to 0.8. This value is consistent with the idea that symptomatic people do not work and with the hypothesis that 80% of the infected population is asymptomatic, according to the Chine Center for Disease Control and Prevention. Therefore, just like Borelli and Góes [2020], we do not adapt such parameter to the Brazilian context.

In contrast, all the previously calibrated parameters, π^R, π^D, A and θ, reflect the Brazilian characteristics. Also, we assign the value of $\beta = 0.966^{\frac{1}{52}}$ in order to get a value of life of BRL 2.9 million. This value of life is based on recent estimates for Brazil (Ferrari et al. [2019] and Rocha et al. [2019]). For the initial infected population, ε, we consider a fraction 0.001 of the total population.

We assume the network search effort is highly inefficient by setting λ_c and λ_n to 0.95, just like in Arbex et al. [2019]. The value of the parameter a is chosen such that $\langle z \rangle$ is equal to 5, again, just as in Arbex et al. [2019]. Without enough foundation, the value of the remaining parameter γ, the relative weight of infection via consumption, is set to the median value of 0.5. However, we will change its value in the next section in order to study its implications to the evolution of the pandemic and the agents' decisions on consumption and hours worked. The variables ρ_t^c and ρ_t^n are modeled in the following way: $\rho_t^c = \rho_t^c = m_t^S$.

[3] IBGE's Automatic Recovery System.
[4] Table 6373.

Tabela 1: Calibration and description of parameters.

Parameter	Description	Value
π_D	Weekly mortality rate	$(7 \times 0.003)/18$
π_R	Weekly recovery rate	$7/18 - \pi_D$
ε	Initial infected population	0.001
A	Labor augmenting technology	8.82352941
θ	Desutility of labor	0.0006541
β	Weekly discount factor	$0.966^{\frac{1}{52}}$
ϕ^S	Productivity of susceptible people	1
ϕ^I	Productivity of infected people	0.8
ϕ^R	Productivity of recovered people	1
a	Parameter of the *degree distribution* D_z	1.01015
λ_c	Inefficacy of infection via consumption	0.95
λ_n	Inefficacy of infection via working	0.95
γ	Relative weight of infection via consumption	0.5

Fonte: Elaboração própria.

4 RESULTS

In this section, we discuss the properties of the competitive equilibrium through a series of numerical simulations. In the first subsection, we analyze how to economy responds to a pandemic in the SIR model with network. In the second subsection, we compare the results for different number of average links in the economy - different $\langle z \rangle$ - by changing the parameter a. The third subsection presents robustness checks over the parameter γ.

4.1 Competitive equilibrium

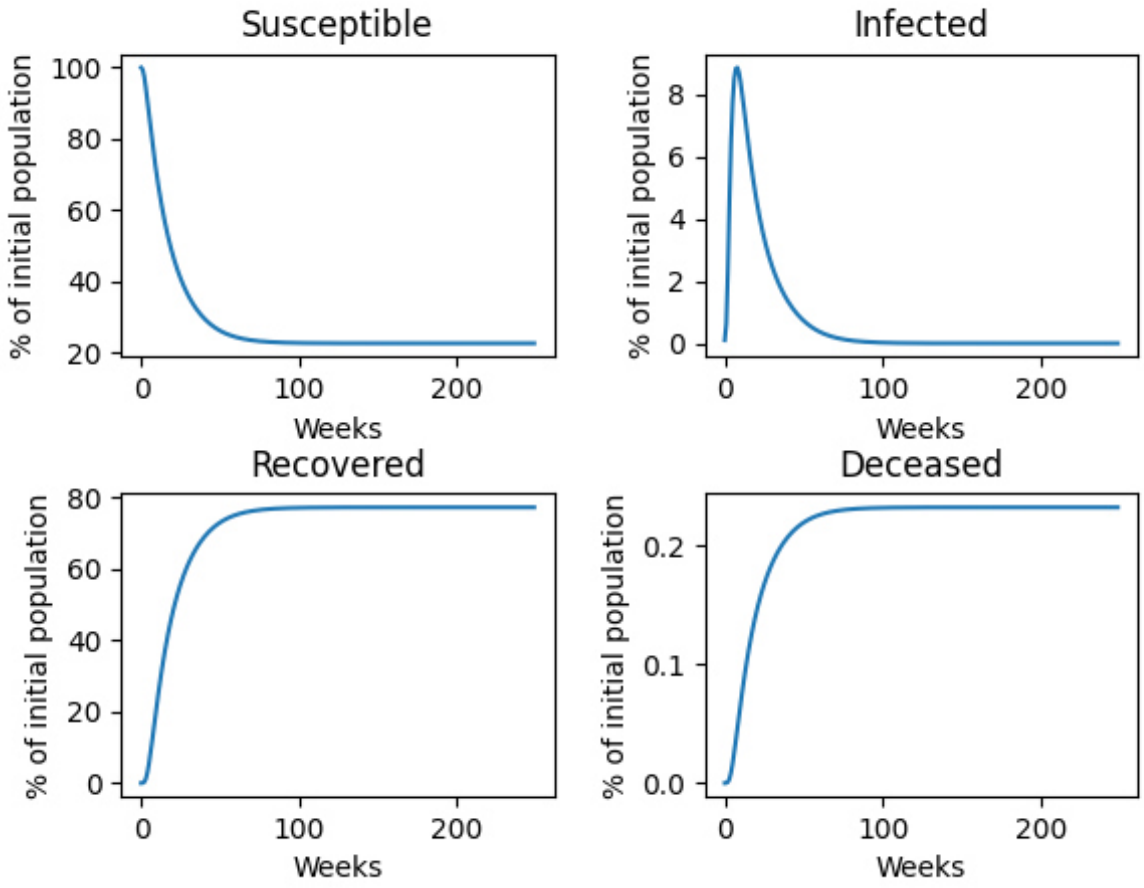
In general, the dynamics of the pandemic happens the following way: from a unit population, a fraction ε gets infected by the virus at the time 0. The fraction of susceptible people is, therefore, $1 - \varepsilon$. At first, for $\varepsilon = 0.001$, the susceptible population is relatively high. This makes the new weekly infections, given by T_t, relatively high as well, what, thus, increases both the infected population and the new value of $T - 1$.

Such process continues until the number of infected people reaches a peak at some

given time, from where the number of new infections stops increasing because of the reduction of the susceptible population. In our numerical exercise, the infected population peaks at the eighth week, representing 8.82% of the total population. Thus, the previous process reverses, and the number of infected people falls as the number of new infections falls. In the end of the pandemic, nearly 86.71% of initial population will eventually have been infected.

At this point, the number of recovered people has reached a significant fraction of the total population, and its growth is, in the absence of treatments and vaccines, the only way of ending the pandemic, which is called the ”herd immunity”. In our simulation, the long run recovered population represents 77.15% of the total population, while 22.61% remains susceptible. Total deaths represent 0.23% of the initial population. Figure 1 shows this dynamics.

Figura 1 - Dynamics of the populations.



Fonte: Elaboração própria.

From the macroeconomics perspective, the dynamics of the epidemic induce recessions. The aggregate consumption falls for two reasons. The first one is due to the low productivity of the small fraction of the population that remains infected. The second, and more important, is due to the number of deaths during the pandemic, that, in turn, also leads to a permanent reduction of the workforce.

In this first analysis, we present the results for the competitive equilibrium, where the government does not interfere in the economy to control the evolution of the pandemic. The dynamics of the pandemic is affected only by the decisions of the economic agents, who has the freedom to reduce their own probabilities of infection by reducing consumption and hours worked.

Similar to what Eichenbaum et al. [2020] found, the results point to a scenario that

only susceptible people are concerned about reducing the infection rate by consuming and working less. Figure 2 shows this pattern. The recovered people are indifferent to the pandemic, for they can no longer get infected, and, thus, act according to what they would act if there was no pandemic.

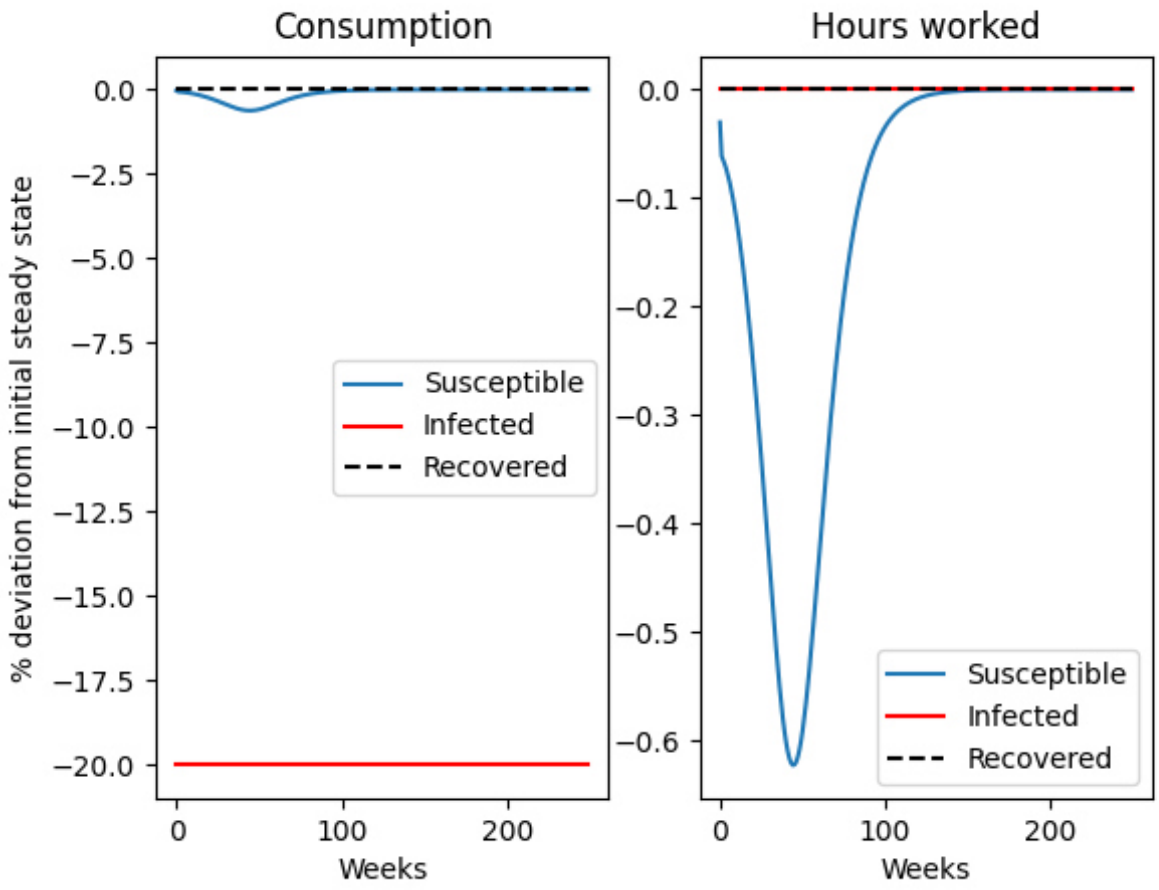
On the other hand, the infected people reduce consumption, not to control the pandemic though, but because, given their lower labor productivity, their income is also lower, and, thus, they face a higher budget constraint. They are obligated to consume less than the recovered agent. More precisely, they consume approximately 20% less then their pre-pandemic steady state consumption, while the recovered agent consumes exactly what they would consume at the pre-pandemic steady state.

However, their hours worked do not differ from the steady state: because they are already infected, they do not have anything to lose by working - similar to the recovered people. But, more precisely, the infected agent's hours worked equals the recovered agent's because the desutility of labor, θ, is the same for all agents. But, say, if we set a higher level of such parameter to the infected agent, then her hours worked would shift down just like her consumption did.

The susceptible agent's behavior on consumption and hours tends to imitate the trajectory of the infected population in its upside down form. As already said, the susceptible agent seeks to avoid infection by reducing contact with other people through consumption and hours worked. As the number of infected people grow, so do grow the probability of becoming infected. Therefore, these agents must cut even more on consumption and hours worked. This process continues until the susceptible agent consumes and works 0.62% less then her steady state levels.

When the infected population falls, the opposite occurs: the probability of becoming infected also falls, and, then, the susceptible agent can gradually increase her consumption and hours worked until she returns to her pre-pandemic steady state level.

Figura 2 - Agents' consumption and hours worked.



Fonte: Elaboração própria.

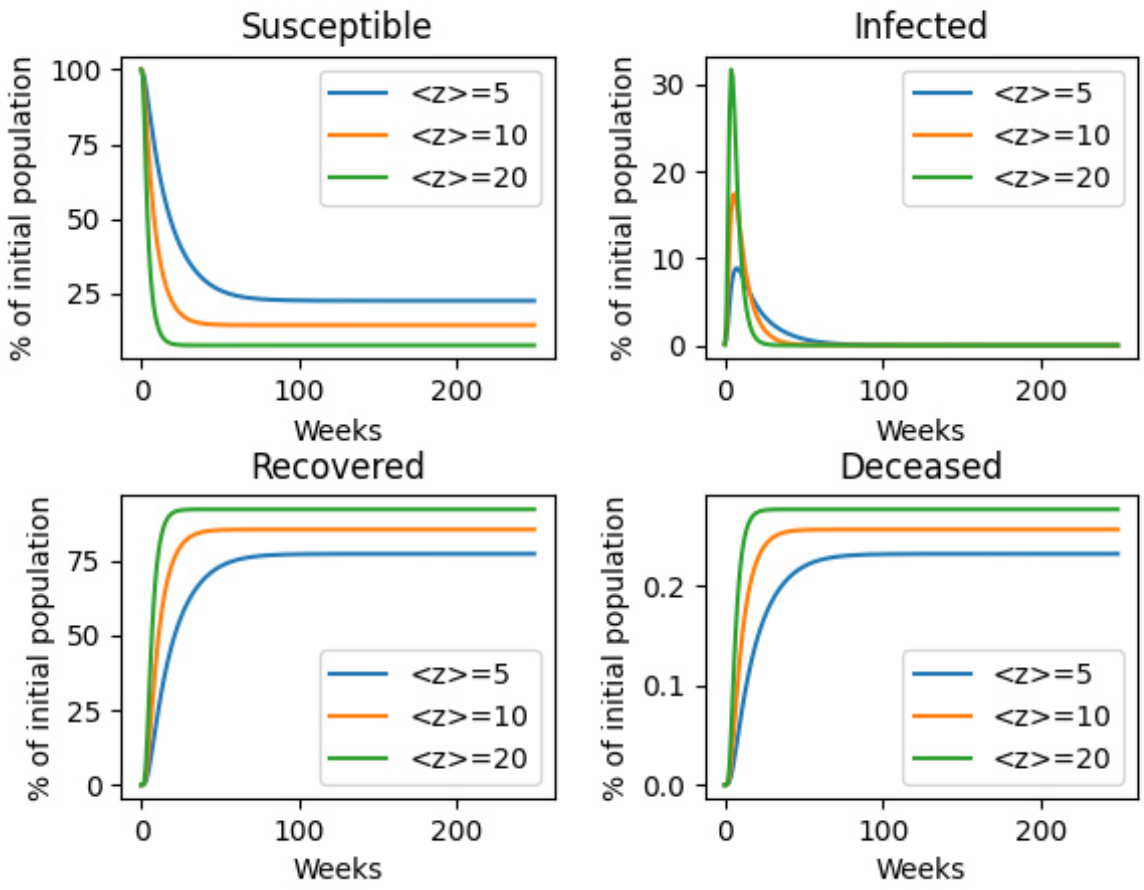
4.2 Comparison between economies with different average number of links

Now we shall look at what happens when we increase the average number of peers of the economy, given by $\langle z \rangle$. We do this by reducing the value of the parameter a. In the baseline calibration, $\langle z \rangle = 5$ corresponds to $a = 1.01015$, while $\langle z \rangle = 10$ corresponds to $a = 1.02155$, and $\langle z \rangle = 20$ corresponds to $a = 1.05$.

The figures below present the results. Figure 3 shows the dynamics of the populations, while Figure 4 shows the consumption and work trajectory for the susceptible agent alone, because the infected and recovered agents would maintain their behavior constant,

just like in Figure 2.

Figura 3 - Populations dynamics for different $\langle z \rangle$.



Fonte: Elaboração própria.

Intuitively, one could expect that more connected economies would produce more infected people and more deaths during a pandemic. And that is confirmed by the results. The populations dynamics, shown by Figure 1, says that more connected economies are associated with more infected people and more deaths. The blue, orange and green lines indicate an economy where the average number of peers equals 5, 10 and 20, respectively.

When $\langle z \rangle = 10$, the infected population reaches the peak a week sooner, at the seventh weak. In that week, infected represent 17.39% of the total population, which is a little growth compared to the baseline calibration. In addition, 85.26% of the total population are recovered in the long run, which is also higher, because more people becomes infected

and, thus, recover from the disease. Moreover, 14.47% remain susceptible, which is less than the baseline calibration, and the deaths represent 0.25% of the initial population, which is also a growth.

If we set $\langle z \rangle = 20$, these numbers get quite worse. Infected population peaks, now, three weeks sooner than the baseline calibration: at the fifth week, reaching 31.67% of the total population. In the long run, the recovered population increase to 92.00%, because the large increase in the number of infected people. Consequently, the susceptible population falls to 7.72%, and total deaths increase to 0.27% of the initial population as expected.

Intuitively, that is the expected result. Because, on average, the number of peers is relatively high, more connected societies allow a faster spread of the virus through their social network. That will cause more infections, thus, more deaths, and, consequently, less susceptible and more recovered people.

In the model, the increase of $\langle z \rangle$ is first introduced, at the first period, only in equations (5) and (6). Note that the probability a susceptible agent receives the virus from a given peer, given by (1) and (2), does not change in the first period with the increase of $\langle z \rangle$.

Analytically, the answer is obvious: both Ω_t^c and Ω_t^n do not depend on the parameter a or $\langle z \rangle$. Intuitively, we must note that, in the first period, the probability a susceptible agent receives the virus from a given peer must not change because there was no time yet for the virus to spread through the social network: the virus is introduced by an external source. The same applies to the probability a susceptible agent of type z receives the virus from at least one peer, given by (3) and (4).

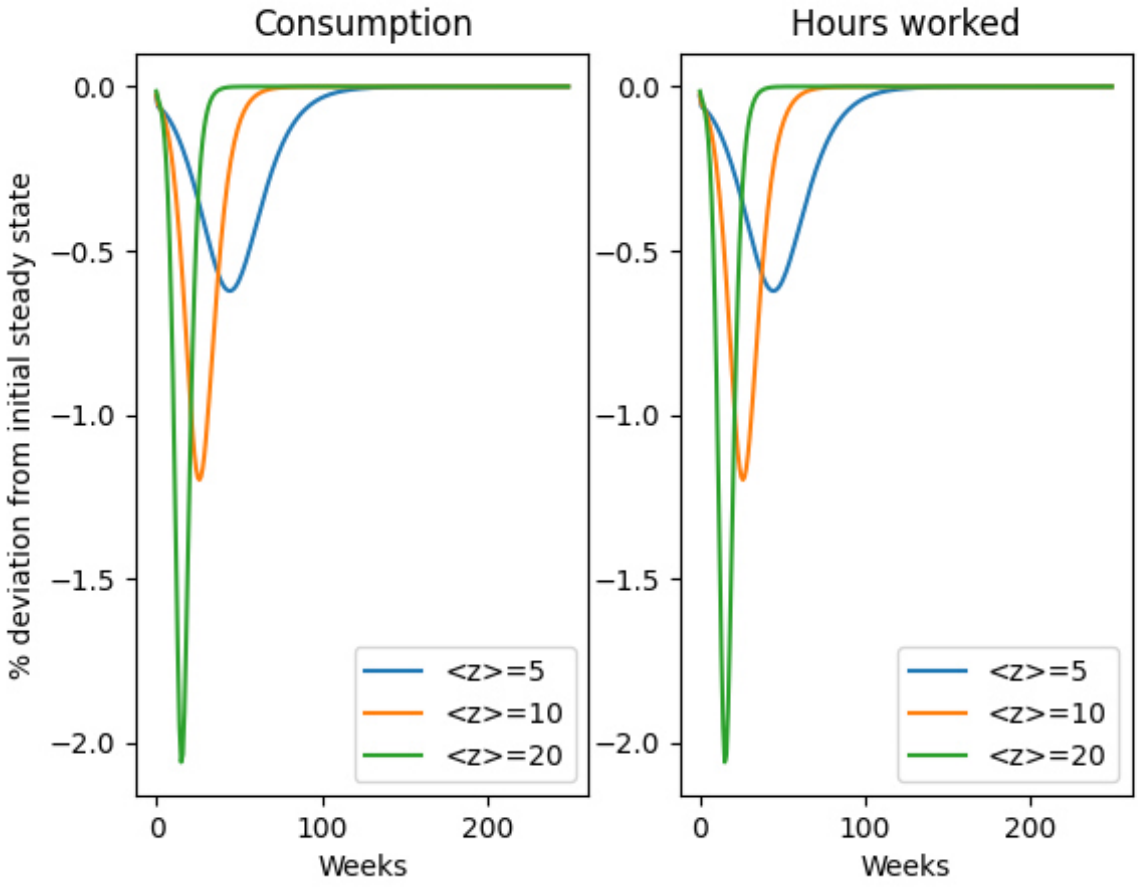
Differently from the previous equations, equations (5) and (6) introduce the effects of the increase of $\langle z \rangle$ right from period 0. Analytically, it is easy to note that both equations integrate the term D_z, which is given by $(a-1)z^{-a}$, and the parameter a appears. The intuition behind it is the following: equations (5) and (6) represent the probability the average susceptible agent receives the virus via her network. In this case, there is a crucial difference compared with the previous equations: the average susceptible agent has changed.

The average susceptible person has $\langle z \rangle$ peers. But in the baseline calibration, that number is 5, while in the other calibrations, that number increases to 10 and 20. Therefore, even at the first period, the average person that has 10 links is more likely to receive the virus through her social network than if she had 5 links. It is true that equations (1) and (2) also refer to the average person. However, they do not account for receiving the virus from her social network. It accounts only for receiving the virus from a given peer.

Figure 4 shows the trajectories of the susceptible agent consumption and hours

worked for different levels of $\langle z \rangle$. We focus only on the susceptible agent because both recovered and infected agents do not change their behavior when we change $\langle z \rangle$, for they are not worried about getting infected. Susceptible people, on the other hand, do care about the number of $\langle z \rangle$, because it will define the evolution of the pandemic, shown by Figure 3, and, thus, influence the probability of becoming infected.

Figura 4 - Consumption and hours worked by susceptible agent for different $\langle z \rangle$.



Fonte: Elaboração própria.

When, on average, people have 5 peers, the susceptible agent cuts less on consumption and work, than when the average number of links is 10 and 20. At the bottom of the curve, as already said, these agents consume and work approximately 0.62% of their steady state level. When the average number of peers is 10, the agents cut less on their economic activities, until the consumption and the hours worked reach a fall of about

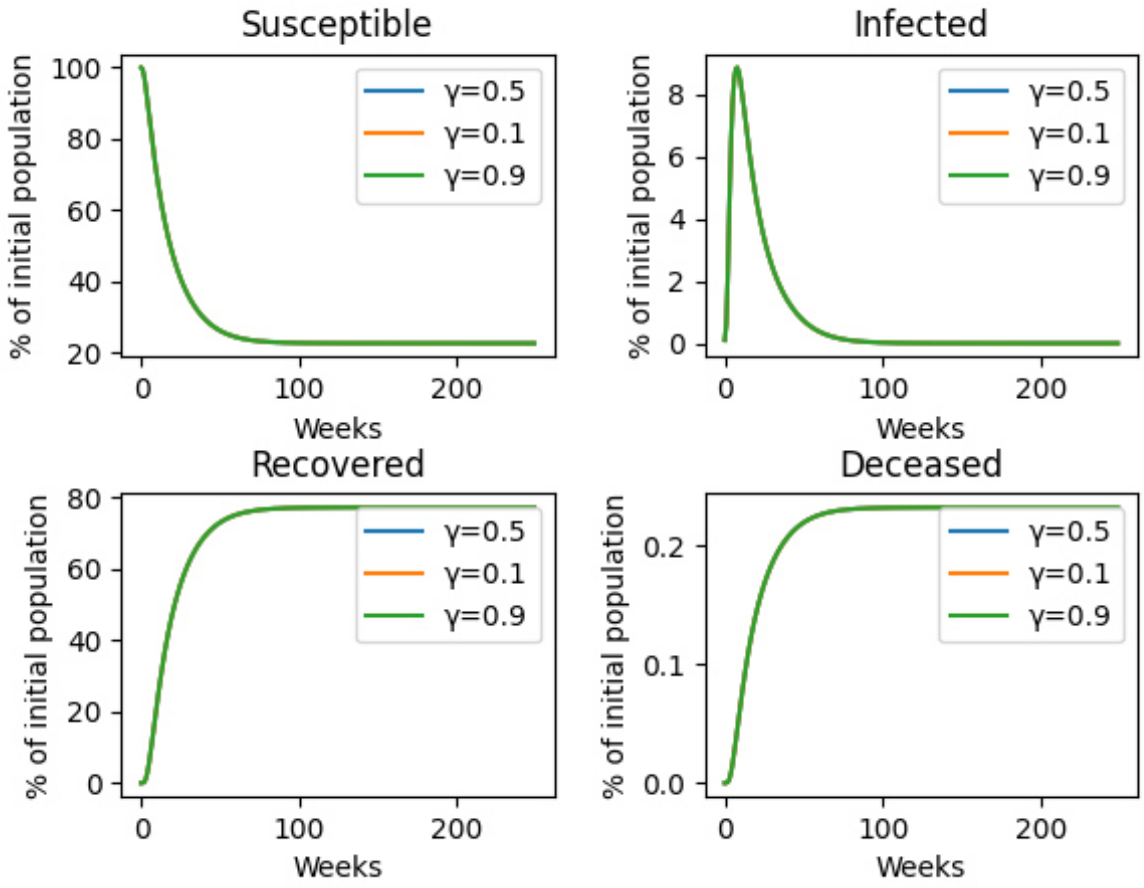
1.19% of the their steady state levels. In an economy where $\langle z \rangle = 20$, it is the extreme case: the susceptible agents decrease their consumption only until they consume and work approximately 2.5% less than their steady state levels.

4.3 Robustness

In this subsection, we do robustness check on the only parameter we do not have a sufficiently reliable source, which is γ, the parameter governing the relative weight of infection via consumption. In the baseline, it was set $\gamma = 0.5$.

Figure 5 shows the dynamics of the populations for different values of γ. By running the model with extreme values - 0.1 and 0.9 -, we get the same result as the one of the baseline. The blue line, corresponding to the baseline calibration, was overlapped by the orange one, that represents the scenario where $\gamma = 0.1$, and the orange line was overlapped by the green line, that represents $\gamma = 0.9$. Therefore, we can state that the results will change little if we change the value of γ. Thus, we maintain our guess that $\gamma = 0.5$ without great concern.

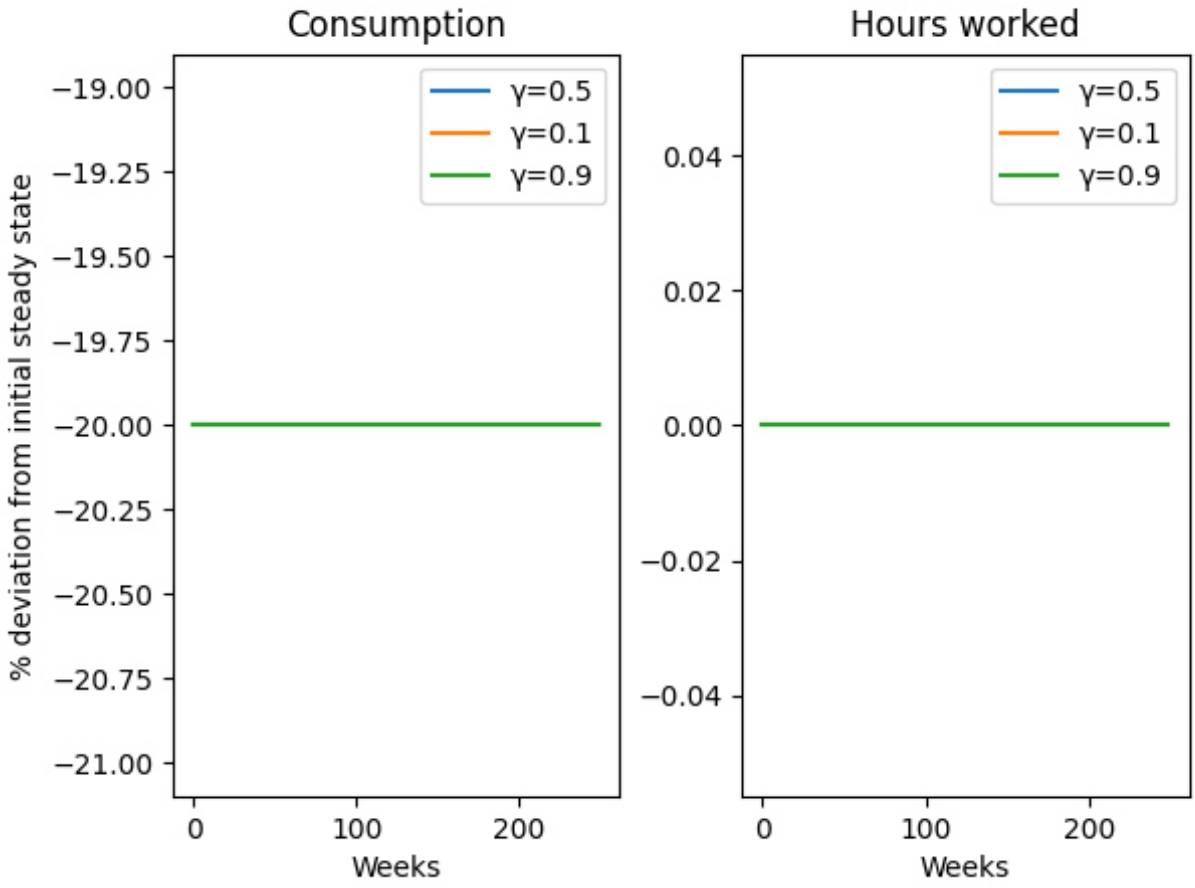
Figura 5 - Populations dynamics for different γ.



Fonte: Elaboração própria.

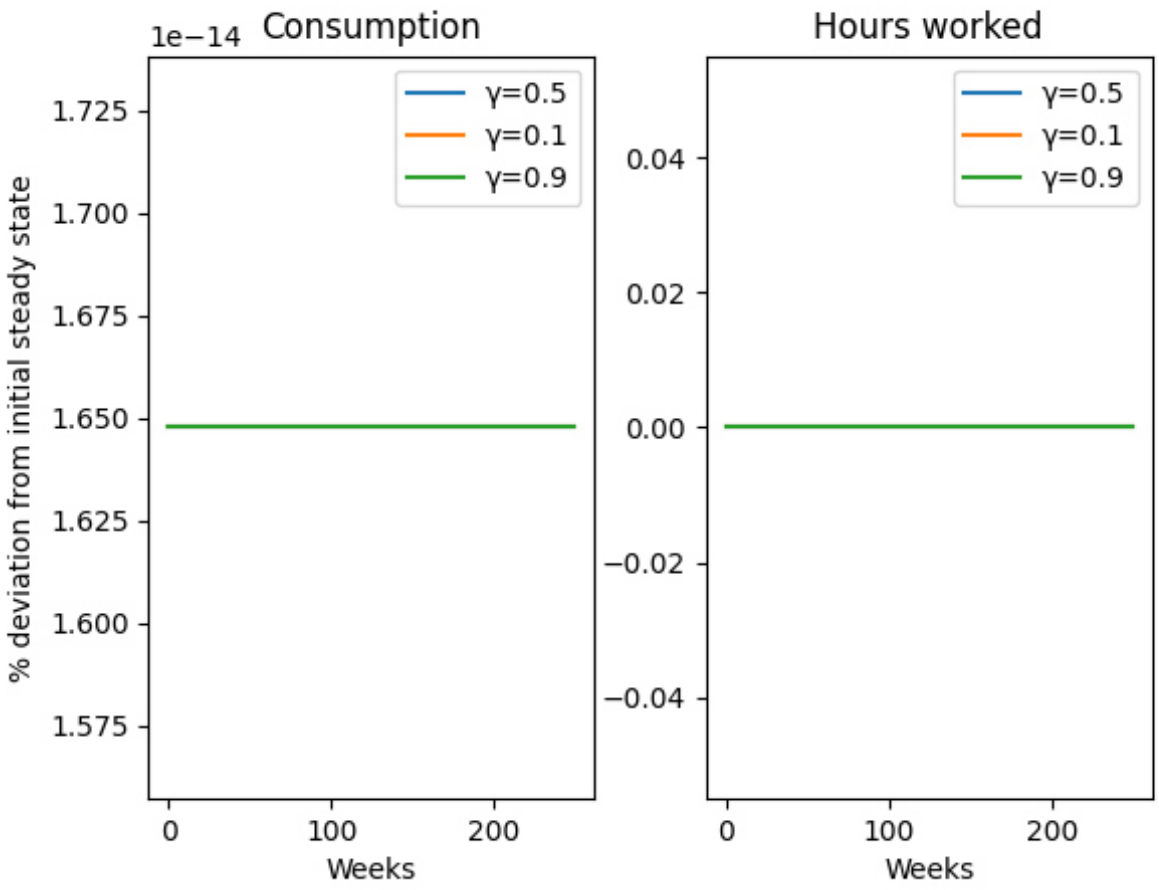
Now we analyze the behavior of the consumption and hours worked of each type of agent as γ changes. Figures 6 and 7 show that behavior for the infected and recovered agents.

Figura 6 - Infected agent's consumption and hours worked for different γ.



Fonte: Elaboração própria.

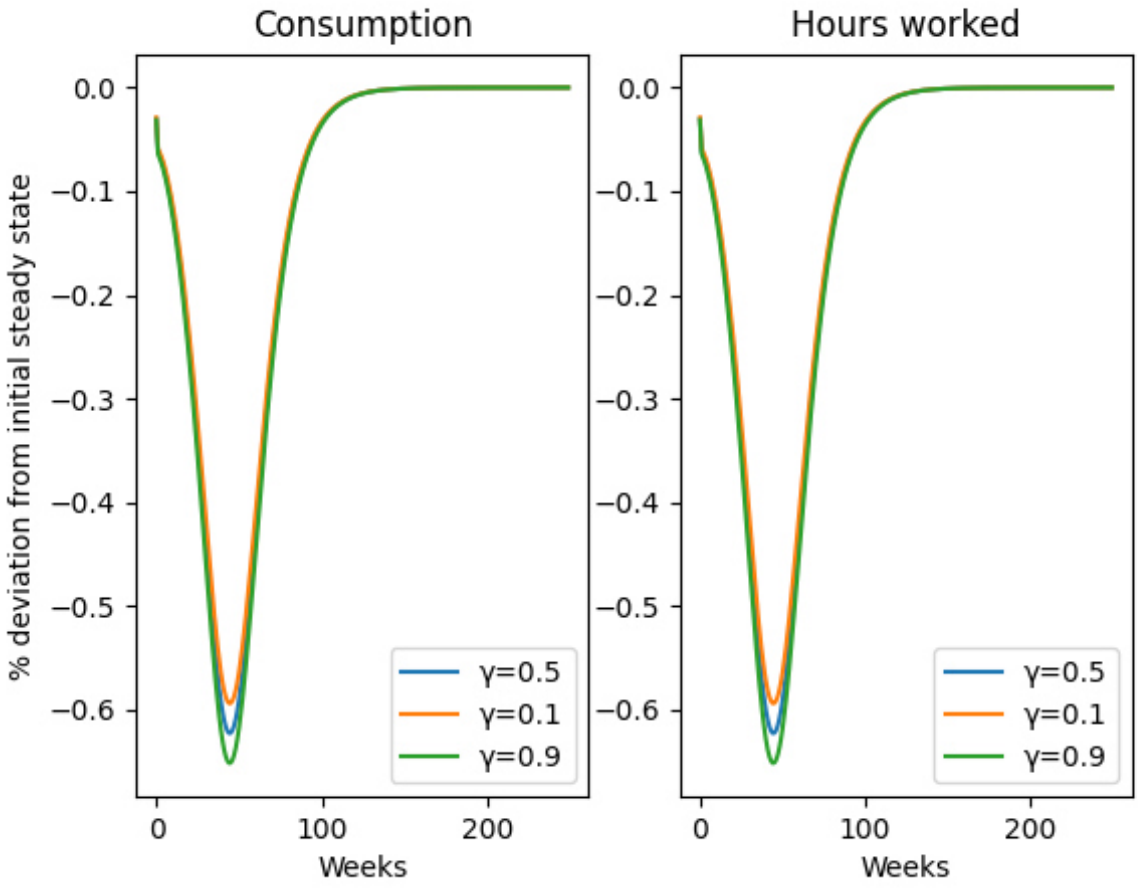
Figura 7 - Recovered agent's consumption and hours worked for different γ.



Fonte: Elaboração própria.

The same result we found at the populations dynamics is confirmed here: the results do not change with variations of γ. The blue line was overlapped by the orange line, which was also overlapped by the green line. Thus, infected and recovered agents do not change their behavior according to changes in γ. But susceptible agents do change their behavior, as shown by Figure 8.

Figura 8 - Susceptible agent's consumption and hours worked for different γ.



Fonte: Elaboração própria.

When $\gamma = 0.1$, the susceptible agent's consumption and hours worked minimum represents a 0.59% fall of their steady state levels. It is higher than the minimum achieved in the baseline calibration, which is a fall of 0.62%.

Although γ is the relative weight of infection via consumption, the higher it is, the lower is the relative weight, because P_t^c, its base (see equation (7)) is between 0 and 1, for it is a probability. Note also that, according to equation (7), 1-γ is the relative weight of infection via work. Now, a higher γ means a higher weight. In this specific case, when we set $\gamma = 0.1$, we are decreasing its value compared to the baseline calibration - $\gamma = 05$ -, and, thus, increasing the relative weight of infection via consumption and decreasing the relative weight of infection via work.

According to our algorithm, the agents first decide how much to work, and, based on

such decision, they consume. Because $\gamma = 1$ represents a decline of the relative weight of infection via work, the susceptible agent would like to work more when compared to the baseline calibration. Thus, the susceptible agent's hours worked has the "U" shaped format, because of the pandemic, but it slightly shifts up.

The opposite occurs when we increase γ to 0.9. Because the relative weight of infection via work has increased, the susceptible agent would like to work less compared to the baseline calibration: the "U" shaped trajectory of hours worked shifts down. At its minimum level, the susceptible agents works 0.65% of the steady state hours worked. Based on such trajectory, her consumption decision is also similar, reaching a 0.65% fall of steady state consumption at its minimum.

Although we find important and intuitive changes on the susceptible agent's decision on consumption and hours worked, changes on the parameter γ are change the main results very little. The trajectories of the populations and the infected and recovered agents' decisions on consumption and hours worked does not change, and the changes found on the susceptible agent's consumption and hours worked are relatively small - about 0.3%. Therefore, we are confident to state our main results with the baseline calibration of $\gamma = 0.5$.

5 CONCLUSION

In this paper, we analyze the specific effects of the COVID-19 pandemic on the Brazilian economy, given the characteristics of its economy and social network. In addition, we study the sensibility of the dynamics of the pandemic on the social network features. For that, we use a Susceptible-Infected-Recovered (SIR) model adapted to a network environment.

Like Eichenbaum et al. [2020] found, the results show the already known behavior of susceptible people reducing consumption and hours worked in order to reduce the probability of becoming infected. With respect to the network environment, we find the expected result. In our model, a person with a higher number of links will be more likely to get infected through her network. Therefore, the higher the average number of links in the society, the virus will spread faster, leading to more infected people and more deaths.

The decision of the agents' consumption and hours worked was also expected. Recovered people maintain their level of pre-pandemic state steady consumption and hours worked because they no longer can be infected by the disease. Infected people also cannot be infected again by the disease, and thus, they also act as if there was no pandemic, but the difference is that, because these people are less productive, they make less money,

and, thus, consume less than they did at the pre-pandemic steady state.

On the other hand, susceptible people, during the course of the pandemic, decrease their consumption and hours worked in order to avoid contact with other people, and, thus, avoid acquiring the disease. With respect to the social network, in more connected economies, the number of infected people is higher. With this, the susceptible agents reduce their consumption and hours worked even more, because the chances of getting infected increase. The opposite is also true: In less connected economies, with less infected people, the susceptible agents' consumption and hours worked, despite reducing because of the presence of infected people, such reduction is quite slighter.

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A APPENDIX: ALGORITHM FOR COMPUTING THE EQUILIBRIUM

The algorithm used to compute the equilibrium is similar to the one that was used by Eichenbaum et al. [2020]. The difference is that we adjust for the network environment. For a given sequence of containment rates, $\{\mu_{ct}\}_{t=0}^{H-1}$, for some large horizon, H, guess the sequences for $\left\{n_t^S, n_t^I, n_t^R\right\}_{t=0}^{H-1}$. Similarly, I solve the model for $H = 250$ weeks. Compute the sequence of the remaining unknowns variables in each of the following equilibrium equations:

$$\theta n_t^R = A\lambda_{bt}^R(1-\mu_{nt}),$$

$$\left(c_t^R\right)^{-1} = (1+\mu_{ct})\,\lambda_{bt}^R,$$

$$u\left(c_t^R, n_t^R\right) = lnc_t^R - \frac{\theta}{2}(n_t^R)^2,$$

$$(1+\mu_{ct})\,c_t^R = (1-\mu_{nt})An_t^R + \Gamma_t,$$

$$\theta n_t^I = \phi^I(1-\mu_{nt})A\lambda_{bt}^I,$$

$$\left(c_t^R\right)^{-1} = (1+\mu_{ct})\,\lambda_{bt}^I,$$

$$u\left(c_t^I, n_t^I\right) = lnc_t^I - \frac{\theta}{2}(n_t^I)^2,$$

$$(1+\mu_{ct})\,c_t^S = (1-\mu_{nt})An_t^S + \Gamma_t,$$

$$u\left(c_t^S, n_t^S\right) = lnc_t^S - \frac{\theta}{2}(n_t^S)^2,$$

$$D_z = (a-1)z^{-a},$$

$$\rho_t^c = m_t^s,$$

$$\rho_t^n = m_t^s,$$

$$\Omega_t^c = \rho_t^c\varphi(c_t^I, c_t^S)m_t^I,$$

$$\Omega_t^n = \rho_t^n\varphi(n_t^I, n_t^S)m_t^I,$$

$$p_t^n = 1-(1-\Omega_t^c)^z\,,$$

$$p_t^n = 1-(1-\Omega_t^n)^z\,,$$

$$P_t^c = \int_{z=1}^{\infty} p_t^c D_z dz = (a-1)\int_{z=1}^{\infty}\left[1-(1-\Omega_t^c)^z\right]z^{-a}dz,$$

$$P_t^n = \int_{z=1}^{\infty} p_t^n D_z dz = (a-1) \int_{z=1}^{\infty} \left[1 - (1-\Omega_t^n)^z\right] z^{-a} dz.$$

Given initial values for Pop_0, S_0, I_0, R_0 and D_0, iterate forward using the following six equations for $t = 0, ..., H-1$:

$$T_t = m_t^S \left(P_t^c\right)^{\gamma} \left(P_t^n\right)^{(1-\gamma)},$$

$$Pop_{t+1} = Pop_t - \pi_D m_t^I,$$

$$m_{t+1}^S = m_t^S - T_t,$$

$$m_{t+1}^I = (1 - \pi_R - \pi_D) m_t^I + T_t,$$

$$m_{t+1}^R = m_t^R + \pi_R m_t^I,$$

$$m_{t+1}^D = m_t^D + \pi_D m_t^I.$$

Iterate backwards from the post-epidemic steady-state value of V_t^S, V_t^I and V_t^R:

$$V_t^R = \max_{c_t^R, n_t^R} \left\{ u\left(c_t^R, n_t^R\right) + \beta V_{t+1}^R \right\},$$

$$V_t^I = \max_{c_t^I, n_t^I} \left\{ u\left(c_t^I, n_t^I\right) + \beta \left[\left(1 - \pi_R - \pi_D\right) V_{t+1}^I + \beta \pi_R V_{t+1}^R \right] \right\},$$

$$\tau_t = \frac{T_t}{m_t^S} = \left(P_t^c\right)^{\gamma} \left(P_t^n\right)^{(1-\gamma)},$$

$$V_t^S = \max_{c_t^S, n_t^S, \tau_t^S} \left\{ u\left(c_t^S, n_t^S\right) + \beta[(1-\tau_t) V_{t+1}^S + \tau_t V_{t+1}^I] \right\}.$$

Calculate the sequence of the remaining unknowns in the following equations:

$$\beta(V_{t+1}^I - V_{t+1}^S) - \lambda_{rt} = 0,$$

$$\left(c_t^S\right)^{-1} - \lambda_{bt}^S (1 + \mu_{ct}) - \lambda_{rt} \gamma \left(\frac{P_t^n}{P_t^c}\right)^{1-\gamma} \frac{\rho_t^c m_t^I \left(1 - \lambda_c\right) m_t^S}{\left(m_t^S c_t^S + m_t^I c_t^I\right)^{\lambda_c}} \int_{z=1}^{\infty} (1 - \Omega_t^c)^{z-1} z^{1-a} dz = 0,$$

$$\left(1 + \mu_{ct}\right) c_t^I = A n_t^I + \Gamma_t,$$

$$m_t^S c_t^S + m_t^I c_t^I + m_t^R c_t^R = A N_t,$$

$$-\theta n_t^S + (1 - \mu_{nt}) \lambda_{bt}^S w_t^S - \lambda_{rt} (1 - \gamma) \left(\frac{P_t^c}{P_t^n}\right)^{\gamma} \frac{\rho_t^n m_t^I \left(1 - \lambda_n\right) m_t^S}{\left(m_t^S n_t^S + m_t^I n_t^I\right)^{\lambda_n}} \int_{z=1}^{\infty} (1 - \Omega_t^n)^{z-1} z^{1-a} dz = 0.$$

B APPENDIX: DERIVATION OF SUSCEPTIBLE AGENT'S MAXIMIZATION

The susceptible agent seeks to maximize the following function:

$$\max_{c_t^S, n_t^S, \tau_t} V_0^S + \sum_{t=0}^{H-1} \left\{ \lambda_{bt}^S \left[(1+\mu_{ct}) c_t^S - w_t^S (1+\mu_{ct}) n_t^S - \Gamma_t \right] - \lambda_{rt} \left[\tau_t - (P_t^c)^{\gamma} (P_t^n)^{(1-\gamma)} \right] \right\}.$$

The first order conditions for c_t^S and n_t^S are given by (15) and (16), respectively.

Calculating $\frac{\partial P_t^c}{\partial c_t^S}$:

$$\frac{\partial P_t^c}{\partial c_t^S} = \frac{\partial \int_{z=1}^{\infty} p_t^c D_z dz}{\partial c_t^S}.$$

D_z is given by $(a-1)z^{-a}$. Thus, and according to (3):

$$P_t^c = \int_{z=1}^{\infty} \left[1 - (1-\Omega_t^c)^z \right] (a-1) z^{-a} dz,$$

$$P_t^c = (a-1) \left[\int_{z=1}^{\infty} z^{-a} dz - \int_{z=1}^{\infty} (1-\Omega_t^c)^z z^{-a} dz \right].$$

Therefore:

$$\frac{\partial P_t^c}{\partial c_t^S} = (1-a) \frac{\partial \int_{z=1}^{\infty} (1-\Omega_t^c)^z z^{-a} dz}{\partial c_t^S},$$

$$\frac{\partial P_t^c}{\partial c_t^S} = (a-1) \int_{z=1}^{\infty} (1-\Omega_t^c)^{z-1} z^{1-a} \left(\frac{\partial \Omega_t^c}{\partial c_t^S} \right) dz.$$

Solving $\frac{\partial \Omega_t^c}{\partial c_t^S}$ separately:

$$\frac{\partial \Omega_t^c}{\partial c_t^S} = \frac{\partial \left[\rho_t^c (m_t^S c_t^S + m_t^I c_t^I)^{1-\lambda_c} m_t^I \right]}{\partial c_t^S},$$

$$\frac{\partial \Omega_t^c}{\partial c_t^S} = \rho_t^c m_t^I \frac{\partial (m_t^S c_t^S + m_t^I c_t^I)^{1-\lambda_c}}{\partial c_t^S},$$

$$\frac{\partial \Omega_t^c}{\partial c_t^S} = \frac{\rho_t^c m_t^I \left(1-\lambda_c\right) m_t^S}{(m_t^S c_t^S + m_t^I c_t^I)^{\lambda_c}}.$$

Thefore:

$$\frac{\partial P_t^c}{\partial c_t^S} = \frac{(a-1)\rho_t^c m_t^I \left(1-\lambda_c\right) m_t^S}{(m_t^S c_t^S + m_t^I c_t^I)^{\lambda_c}} \int_{z=1}^{\infty} (1-\Omega_t^c)^{z-1} z^{1-a} dz.$$

Similarly, doing the same steps:

$$\frac{\partial P_t^n}{\partial n_t^S} = \frac{(a-1)\rho_t^n m_t^I \left(1-\lambda_n\right) m_t^S}{(m_t^S n_t^S + m_t^I n_t^I)^{\lambda_n}} \int_{z=1}^{\infty} (1-\Omega_t^n)^{z-1} z^{1-a} dz.$$