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Modelling COVID-19 Ripple Effect and Global Supply Chain Productivity Impacts using a Reaction-Diffusion Time-Space SIS Model

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Abstract. As the COVID pandemic shows, infection spreads widely across regions, impacting economic activity in unforeseen ways. We represent here how the geographic spread of the pandemic, by reducing the workers' participation to economic life, undermines the ability of firms and as a result the entire supply networks to satisfy customers' demands. We model the spatio-temporal dynamics of the propagation of Covid-19 infection on population, transport networks, facilities and population flows. The mathematical models will enable prospective analyses to be performed reliably. Such models will be used in what-if scenarios to simulate the impact on both populations and supply chain activities in case of future pandemics. The outcome should be useful tools for policymakers and managers. Results from this research will help in understanding the impact and the spread of a pandemic in a particular region and on supply chains. The data will be from European regions and the expected models will have validity in Europe.

Keywords: Spatial dynamics · ripple effect · propagation · disruption · graph theory · network evaluation

1 Introduction

Covid-19 is a highly contagious virus-induced communicable disease, transmitted via droplets and contaminated objects during close unprotected contact between a healthy and an infected person [6]. As such infected people move away from the location in which they were contaminated, uncontaminated locations farther and farther away become centres of infection in their own right.

The Covid-19 pandemic has had huge human and economic consequences. Thus, understanding how to reduce the spread of the disease and which specific policies to implement in order to manage the pandemic is paramount

As is well understood, the movement of infected people and the evolution of the virus are fundamental to the spread of the pandemic. The flows of populations are represented depending on the granularity of the data and the level of detail of the maps. When such movements are coupled with the incidence of infection, insights can be obtained about the evolution in time and in space, and causes may be inferred. As the level of detail increases, provided that the

corresponding data exists, further insights about the causes of the dispersion of the virus and the corresponding infection intensity in the population can be obtained. Thus, mapping how the infection spreads can shed light both on the type of countermeasures and the impact on economic activity.

The European economy is dependent on international cooperation and on the fact that goods can flow freely. Most of the firms' supply chains are highly interconnected, characterised by a high degree of complexity, long distances, and a large number of intermediaries. According to a study, 75% of European supply chains have been negatively affected by the crisis. The most important bottlenecks are the inward flow of goods from suppliers (62%), lack of insight into customer needs (60%), and the outward flow of goods to customers (50%).

We are therefore now in a largely unknown territory in relation to risk management in the supply chain. The challenge does not only include sharp fluctuations on the part of customers with unknown and highly fluctuating demand but also from supplies which can no longer be produced because specific inputs are produced at slower rates or arrive sporadically.

The purpose of the study is to be able to identify the evolution of the pandemic and model its impact on supply chains. Several models approximating the temporal spread of the pandemic [2] or of the effect of lockdown measures [8] already exist but do not help in understanding the ripple effect on supply chains.

In the following, we present the state of research on the way a pandemic's effects spreads through supply chain networks and the proposed ways for managers to control this impact on their firms' activity. We then present two epidemiological models which explain the infection spread in a homogeneous population and its extension into the spread among various populations. In section 5, we model the impact of a pandemic on the various nodes of a supply network in terms of productivity as workers get infected. We draw some conclusions and present recommendations for future research in section 7.

2 Ripple Effect visualisation for global supply chains

The phenomenon of the ripple effect has received great research interest in recent years and more and more contributions have tried to model the dynamics of the ripple effect through a supply chain network. The ripple effect occurs when a major disruptive event, such as the lock-downs initiated by Covid-19 virus, trigger a wave of simultaneous disturbances coming from several different directions [12,13,7,15]. It occurs through the propagation of Low Frequency and High Impact unforeseen disturbances [14].

Existing work attempts to understand the effects by modelling the ripple effect [13], and other related supply chain network redesign approaches [22]. Baghersad and Zobel [3] examine the effects of supply chain disruptions on firms' performance by applying a new quantitative measure of a disruption's impact adapted from the systems resilience literature. Li et al. [20] study and examine disruption propagations through simulating simple interaction rules of firms

inside the supply chain network by developing an agent-based computational model.

The literature has thus attempted to understand the effects better by ripple effect quantification and other related supply chain mapping approaches [16]. However, there is a niche for exploring other Ripple effect approaches, and our study attempts to contribute towards the area of Ripple effect visualization.

3 The Classical SIS Model

The susceptible-infected-susceptible (SIS) epidemiological model represents one of the simplest frameworks to analyze disease dynamics. The population, N , which is assumed to be constant, is composed of two groups: individuals who are infected, I_t , and individuals who are not infected but susceptible to infection, S_t . Infected individuals spontaneously recover from the disease at a speed $\delta > 0$, while susceptible individuals contract the disease at a rate $\alpha > 0$ by interacting through random matching with infected ones. Unhappily, as we now know [23], individuals who have recovered from the infection can be re-infected, that is, after a lapse of time they are susceptible again. This means that we cannot simply use a SIR model (Susceptible, Infected, Recovered) as presented in [21,11,1,5]. The probability with which matching occurs depends on the actual spread of the disease across the population [9,4,10,18,19]. The evolution of the number of infectives and susceptibles is described by the following differential equations:

$$\dot{S}_t = \delta I_t - \alpha \frac{S_t I_t}{N} \quad (1)$$

$$\dot{I}_t = \alpha \frac{S_t I_t}{N} - \delta I_t, \quad (2)$$

The above system can be simply recast in terms of the share of infectives, $i_t = \frac{I_t}{N}$, and the share of susceptibles, $s_t = \frac{S_t}{N}$, as follows:

$$\dot{s}_t = \delta i_t - \alpha i_t s_t \quad (3)$$

$$\dot{i}_t = \alpha i_t s_t - \delta i_t. \quad (4)$$

Since $1 = s_t + i_t$, essentially, the epidemic dynamics can be completely characterized by focusing on one of the two equations as follows:

$$\dot{i}_t = \alpha i_t (1 - i_t) - \delta i_t. \quad (5)$$

The above equation describes the evolution of the disease prevalence in the entire population. Note that analyzing the equilibria for the above model is rather straightforward. As discussed in the epidemiology literature, the long run outcome solely depends on the basic reproduction number, \mathcal{R}_0 , given by $\mathcal{R}_0 = \frac{\alpha}{\delta}$.

4 A Reaction-Diffusion Time-Space SIS Model

We now focus on an extension of the basic SIS model to allow for geographical heterogeneities and externalities by introducing a spatial dimension. We denote

with $S_{x,t}$ and $I_{x,t}$, respectively the susceptibles and infectives in the position $x \in \Omega$, where $\Omega \subset \mathbb{R}^2$ is a compact set at date $t \in \mathbb{R}_+$.

The epidemic dynamics cannot be fully characterized by focusing only on the evolution of the portions of infectives and susceptibles ($i_{x,t}$ and $s_{x,t}$) rather than the numbers of infectives and susceptibles ($I_{x,t}$ and $S_{x,t}$). This is due to the fact that the population is spatially distributed, $N = \int_{\Omega} N_{x,t}$ with $N_{x,t} = S_{x,t} + I_{x,t}$, and thus it is not necessarily true that the shares of infectives and susceptibles sum up to one in each location x (i.e., they do sum up to one over the whole spatial domain). In particular, the share of infectives, $i_{x,t} = \frac{I_{x,t}}{N}$, and the share of susceptibles, $s_{x,t} = \frac{S_{x,t}}{N}$, in each location x jointly determine the share of the total population residing in that specific location, $n_{x,t}$ with $n_{x,t} = s_{x,t} + i_{x,t}$, while the sum of the shares of the total population residing in all locations is one, $\int_{\Omega} n_{x,t} dx = 1$.

Therefore, we need to analyze the evolution of the share of infectives and susceptibles over time and across space, and the spatial model can be represented through a system of reaction-diffusion partial differential equations (a similar approach is used, for instance, in [17,24] to describe pollution diffusion) as follows:

$$\frac{\partial s_{x,t}}{\partial t} = d\nabla^2 s_{x,t} + \delta i_{x,t} - \alpha \int_{\Omega} s_{x',t} i_{x',t} \varphi_{x',x} dx', \quad (6)$$

$$\frac{\partial i_{x,t}}{\partial t} = d\nabla^2 i_{x,t} + \alpha \int_{\Omega} s_{x',t} i_{x',t} \varphi_{x',x} dx' - \delta i_{x,t}, \quad (7)$$

where the term $\varphi_{x,x'}$ describes the probability that infected people at the location x' could spread the infection at the location x with $x \neq x'$.

By recalling that $n_{x,t} = s_{x,t} + i_{x,t}$, it follows that $n_{x,t}$ solves the summation of equations (6) and (7), that is $\frac{\partial n_{x,t}}{\partial t} = d\nabla^2 n_{x,t}$ with Neumann boundary conditions and initial conditions directly determined from those related to $s_{x,t}$ and $i_{x,t}$. This allows us to consider $n_{x,t}$ as a known exogenous variable, which thus can be substituted in (6) and (7) by writing $s_{x,t} = n_{x,t} - i_{x,t}$.

$$\frac{\partial n_{x,t}}{\partial t} = d\nabla^2 n_{x,t} \quad (8)$$

$$\frac{\partial i_{x,t}}{\partial t} = d\nabla^2 i_{x,t} + \alpha \int_{\Omega} (n_{x',t} - i_{x',t}) i_{x',t} \varphi_{x',x} dx' - \delta i_{x,t}. \quad (9)$$

Notice that the expression of $n_{x,t}$ is known in closed-form once a specific shape of the set Ω is assumed.

In order to understand the disease dynamics, we can thus analyze the system of partial differential equations (PDEs) given by (8) and (9), which generalizes to a spatial dimension SIS model that accounts for population mobility across locations.

Let us notice that whenever the kernel function $\varphi_{x',x}$ coincides with the Dirac, the above model boils down to:

$$\frac{\partial n_{x,t}}{\partial t} = d\nabla^2 n_{x,t} \quad (10)$$

$$\frac{\partial i_{x,t}}{\partial t} = d\nabla^2 i_{x,t} + \alpha(n_{x,t} - i_{x,t})i_{x,t} - \delta i_{x,t}, \quad (11)$$

with the Neumann boundary conditions $\frac{\partial n_{x,t}}{\partial n} = 0$ and $\frac{\partial i_{x,t}}{\partial n} = 0$.

To conclude, once the configuration of the network is known, the number of infected per node (location) can be evaluated for every location and period of time. Note that this description is computed directly, contrary to [11] which requires an algorithm. When comparing this model with the estimation method in [21], we purport that this one presents numerous advantages as it does not rely on Bayesian mechanics (including having to evaluate multinomial distributions) and yet could be applied using the same Baidu-Qianxi database of population mobility.

5 Supply Chain Network and Total Productivity

The global supply chain is modelled by the means of a graph $G = (V, E)$ with M nodes, where V denotes the node set and $E \subset V \times V$ is the edge set. For simplicity we identify each node $v \in V$ of the network with its geographical coordinates x_v .

Given two nodes $v, u \in V$, $0 \leq \phi_{vu} \leq 1$ represents the degree of interaction from node v to node u , summarizing the intensity of their reciprocal trade and logistic relationships. The network is then described in compact form by the triplet $G = (V, E, \phi)$ where ϕ is a $M \times M$ weighted matrix with the property that $\phi_{uv} = \phi_{vu}$. The level of infected at each node $v \in V$, is modelled by $i_{x_v,t}$. The local evolution of the infection is depending on both the local evolution of the epidemics as well as the interaction with the adjacent locations.

If $\Gamma(x)$ describes the per capita productivity at location x , the total productivity index $\Gamma_{tot}(t)$ of the supply chain network at time t depends on the susceptible population at each node $v \in V$ of the network and is defined as

$$\Gamma_{tot}(t) = \sum_{v \in V} \Gamma(x_v) s_{x_v,t} = \sum_{v \in V} \Gamma(x_v) (n_{x_v,t} - i_{x_v,t}). \quad (12)$$

Let us notice that, in absence of epidemics,

$$\Gamma_{tot}(t) = \sum_{v \in V} n_{x_v,t} \Gamma(x_v). \quad (13)$$

The combination of the dynamics of the epidemic with the definition of the network productivity leads to the following evolution equation of total $\Gamma_{tot}(t)$:

$$\begin{aligned}
\dot{I}_{tot}(t) &= \sum_{v \in V} \Gamma(x_v) \frac{\partial s_{x_v,t}}{\partial t} \\
&= \sum_{v \in V} \Gamma(x_v) \left(d \nabla^2 (n_{x_v,t} - i_{x_v,t}) - \alpha \int_{\Omega} (n_{x',t} - i_{x',t}) i_{x',t} \varphi_{x',x_v} dx' + \delta i_{x_v,t} \right) \\
&= \sum_{v \in V} \Gamma(x_v) \left(d \nabla^2 s_{x_v,t} - \alpha \int_{\Omega} s_{x',t} i_{x',t} \varphi_{x',x_v} dx' + \delta i_{x_v,t} \right). \tag{14}
\end{aligned}$$

6 Numerical simulation

In this section we present a numerical simulation of the dynamic model:

$$\frac{\partial n_{x,t}}{\partial t} = d \nabla^2 n_{x,t} \tag{15}$$

$$\frac{\partial i_{x,t}}{\partial t} = d \nabla^2 i_{x,t} + \alpha \int_{\Omega} (n_{x',t} - i_{x',t}) i_{x',t} \varphi_{x',x} dx' - \delta i_{x,t}. \tag{16}$$

For simplicity we suppose that Ω is a 1-dimensional domain and it is normalized to $\Omega = [0, 1]$ and that $\varphi_{x',x} = \delta_x(x')$ is the Dirac at the point x . We also assume that the diffusion coefficient d is normalized to 1, the initial distribution of infected people i_0 is equal to $0.01x(1-x)$ as shown in Fig. 1, and the initial population $n_{x,0}$ is homogeneous and normalized to 1. The solution to

$$\frac{\partial n_{x,t}}{\partial t} = d \frac{\partial^2 n_{x,t}}{\partial x^2} \tag{17}$$

subject to the Neumann condition being known, is provided via the Fourier expansion by:

$$n_{x,t} = \sum_{n \geq 0} A_n e^{-(n\pi)^2 t} \cos(n\pi x), \tag{18}$$

where

$$A_0 = \int_0^1 n_{x,0} dx = 1, \tag{19}$$

$$A_n = 2 \int_0^1 n_{x,0} \cos(n\pi x) dx = 0, \tag{20}$$

which implies that $n_{x,t} = 1$ for any x and t . The above model thus boils down to

$$\frac{\partial i_{x,t}}{\partial t} = d \frac{\partial^2 i_{x,t}}{\partial x^2} + \alpha(1 - i_{x,t})i_{x,t} - \delta i_{x,t}. \tag{21}$$

In this case, for any network G , the total productivity evolves accordingly to:

$$\Gamma_{tot}(t) = \sum_{v \in V} \Gamma(x_v)(1 - i_{x_v,t}). \quad (22)$$

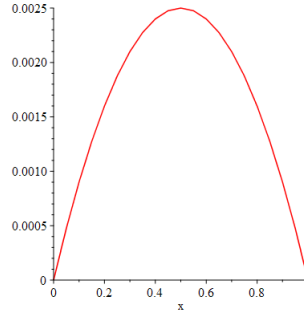


Fig. 1. Initial profile of infected people: $i_0(x) = 0.01x(x - 1)$

In the first numerical simulation we suppose that the infection rate α is equal to 0.1328 and the recovery rate $\delta = 0.0476$ (see [18]). The figure shows that after an initial transition phase of growth, the number of infected people converges to a long-run endemic and homogeneous equilibrium $i_{x,\infty}$. In this scenario, the natural recovery rate δ is not big enough to guarantee a decrease of $i_{x,t}$ over time.

In this case, let us observe that due to the presence of the pandemic, the global productivity index Γ_{tot} will converge to (see Fig. 2):

$$\lim_{t \rightarrow +\infty} \Gamma_{tot}(t) = (1 - i_{x_v,\infty}) \sum_{v \in V} \Gamma(x_v) < \sum_{v \in V} \Gamma(x_v) \quad (23)$$

In the second numerical simulation, we suppose that the infection rate α is still equal to 0.1328 but the implementation of treatment has raised the recovery rate δ to 0.1428. In this case, as Fig. 3 shows, the number of infectives decreases over time and it converges to the disease eradication in the long-run. Of course, the interesting part is for intermediate periods where the number of infectives may be non-homogeneous geographically: some locations will be more affected than others.

Let us observe that in this case, instead, the global productivity index Γ_{tot} will converge to:

$$\lim_{t \rightarrow +\infty} \Gamma_{tot}(t) = (1 - i_{x_v,\infty}) \sum_{v \in V} \Gamma(x_v) = \sum_{v \in V} \Gamma(x_v) \quad (24)$$

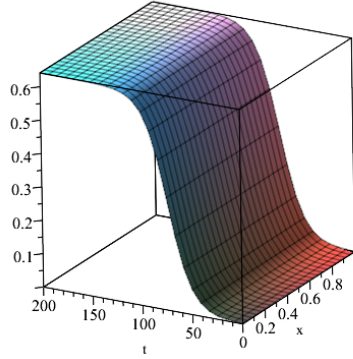


Fig. 2. Long-run behavior of the disease: $d = 1$, $\alpha = 0.1328$, $\delta = 0.0476$

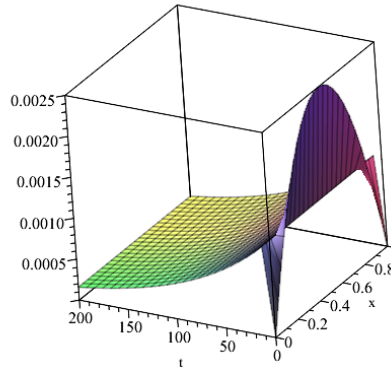


Fig. 3. Long-run behavior of the disease: $d = 1$, $\alpha = 0.1328$, $\delta = 0.1428$

7 Conclusion

In conclusion, we see how a pandemic spreads over regions, countries and continents in continuous time. We have modelled how such a pandemic infects workers and how this effect slows the production in a supply network, thus impacting the productivity of single production units and so the whole supply networks.

In this model, we have considered that the effect on production is simply additive. Of course, in reality, once an upstream partner in a supply network is impacted, all the downstream partners are also impacted. In a later refinement, we could look at a model where the effect of a pandemic is exponential in terms of the position of a node in the network. Another model might take into account the possibility that the infection rate α has different values across regions, or time.

It is easy to include this in the model described in (9) as $\alpha_{x,t}$, so accommodating variants to the original virus.

In contrast to other studies of spatial transmission of the pandemic mentioned here, the advantage of the model is to only build from well defined and understood parameters: the infection rate α , recovery rate δ , the layout of the network, and population distribution in the various locations. The model takes into account the fact that people can be re-infected (which means that SIR or SIRD models are inadequate).

In this way, once a new virus is identified, knowing the characteristics of a network, a policy maker (manager) can build a forecasting model to evaluate the spread of the infection in the regions under her purview (supply chain network). Armed with such a model, a calibrated set of measures can be implemented which might impose a lesser burden on populations in the case of public policy or improve the resilience of the supply chain network.

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