

Incorporating Social Determinants of Health into Modelling of COVID-19 and other Infectious Diseases: A Baseline Socio-economic Compartmental Model

Giorgos Galanis and Adam Hanieh

Abstract

The role of socio-economic conditions has been largely implicit in mathematical epidemiological models. However, measures to address the current pandemic, specifically the relevant interventions proposing physical distancing, have highlighted how social determinants affect contagion and mortality dynamics of COVID-19. For the most part, these social determinants are not present in either policy discussions or in relevant epidemiological models. We argue for the importance of incorporating relevant social determinants of health into the modelling dynamics of COVID-19, and show how *global* variation of these conditions may be integrated into relevant models. In doing so, we also highlight a key political economy aspect of reproduction dynamics in epidemics.

Keywords: epidemiology, COVID-19, inequality, uneven development, compartmental models, contagion, social determinants of health

1. Introduction

The extensive discussions around predictive modelling of SARS-CoV-2 (COVID-19) is commonly based on the use of mathematical epidemiological models. These largely originate in the work of Kermack and McKendrick (1927), and study the transition of individuals between different compartments that correspond to relevant health conditions. The simplest and most popular of these is the *SIR* model, which assumes that a given population is split between three compartments: Susceptible (*S*), Infected (*I*), and Recovered (*R*), where at every point in time some of the susceptible individuals become infected and some of the infected individuals recover. In the baseline version of the *SIR* model, immunity of the recovered is assumed, and as more people get infected the number of *S* diminishes, which eventually leads the contagion to die out.

Models using this framework have been extended in different directions by dividing the population into additional compartments, adding stochasticity in the different transition probabilities and/or using an agent-based framework. For example, Prem et al. (2020) study the effects of physical distancing measures in Wuhan, China using an age-structured susceptible-exposed-infected-removed (*SEIR*) model that includes the addition of an “exposed” compartment and the division of the population into 16 age categories. Kucharski et al. (2020) use a stochastic *SEIR* model to study the contagion dynamics in Wuhan and the transmission potential outside Wuhan. Klöh et al. (2020) use an agent-based model to assess interventions in

Rio de Janeiro, Brazil and Chang et al (2021) introduce mobility networks to an SEIR model and analyse the contagion dynamics across social groups. In all cases of mathematical epidemiological models, the key focus remains the transition probabilities between different compartments, which are then used to analyse the contagion dynamics of a particular epidemic.

Compartmental models have been the basis of a large part of COVID-19-related epidemiological research. In general, these models attempt to capture the combined interaction of a given viral epidemiology, the effects of relevant non-pharmaceutical interventions (NPIs), and the compliance of the public with these measures. They have guided much of the international policy response to COVID-19 (see McBryde et al. 2020 and Thomson, 2020) and have even garnered wider public media attention beyond epidemiological research circles. In this respect, the main NPIs under discussion have related to *physical distancing* between individuals. These types of policies aim to reduce the average number of contacts between individuals in close spatial proximity, which in turn, will lower on average the rate through which individuals get infected and hence the epidemic's effective reproduction number (Paeng and Lee, 2017; Milner and Zhao, 2008; for example see Funk et al., 2010; Manfredi and D'Onofrio, 2013; Verelst et al., 2016; Weston et al. 2018; Hauck, 2018; Di Guilmi et. al. 2020).

It is our contention, however, that standard compartmental epidemiological models do not adequately consider the various social determinants of health that have a direct impact on the inequalities of health outcomes and the ability of populations to effectively comply with NPIs (McCartney et al 2019). Incorporating these social determinants into compartmental models can improve the predictive power and accuracy of modelling both with regards to contagion

dynamics and the severity of the relevant epidemic. Moreover, these can help us understand the impact of different social welfare regimes on the efficacy of a given set of NPIs.

In what follows, we introduce a set of social determinants (SDs) that can be linked to relevant variables and used to assess the utility of various policy options. The focus of our discussion is on the global inequalities between high income and low-medium income countries (LMICs) although, as we note in the conclusion, the argument could be extended to examine inequalities within a single country or community. It is important to examine the situation of LMICs, due to the significant obstacles these countries face in implementing standard containment and mitigation policies (Ahmed et al. 2020).

Our argument builds upon the Social Determinants of Health (SDH) literature, which emphasises the necessity of viewing health outcomes as a social phenomenon that demand a broad range of intersectoral initiatives (Solar and Irwin 2010; Krieger 2017). Social determinants of health are understood as the wider conditions in which people are born, live, and age. They encompass a variety of factors such as socioeconomic conditions, access to education and employment, the quality of urban and physical environments, robustness of social support networks, and access to health care (Adler and Stewart 2010; Gottlieb et al 2019). The insights of the SDH literature have had a demonstrable impact on the development of health policy at the global level. By and large, however, the various social determinants identified in this literature have not been incorporated into epidemiological modelling. This remains a significant gap in both understanding and addressing the differential vulnerabilities associated with pandemics such as COVID-19.

In this paper we address this gap, proposing a framework within the broad field of social epidemiology (Berkman and Kawachi 2000). We analyse how three SDs - conditions of employment, conditions of housing, and access and quality of health infrastructures - might affect the different transition rates between the various compartments in compartmental epidemic models. Our starting point is a version of the Susceptible Infected Recovered Deceased (SIRD) model, which we extend to allow the aforementioned SDs to influence the transition probabilities from Susceptible to Infected and from Infected to Deceased respectively. We have chosen this model as it is the simplest framework which includes the relevant compartments for the three SDs mentioned. However, we should note that our analysis could be easily extended to models which include more compartments and other characteristics, for example, different age cohorts as in Prem et al. (2020).

Our choice of SDs draws upon key factors that have been highlighted in the SDH literature (Rolfe et al 2020; Braveman and Gottlieb 2014; Solar and Irwin 2010) but should be understood as merely illustrative of our broader argument. We recognise that a different range of SDs could be utilised, and also that social determinants of health are significantly co-constituted and mutually-reinforce one another (see the discussion of Solar and Irwin 2010, pp. 20-42). As our focus is on the social aspects that can be included in a mathematical epidemiological model, our starting point is the simplest possible model where we can add the social variables. We should note however, that the same intuition can hold even if complexity is added through the inclusion of more compartments, age cohorts and/or stochastic dynamics. Our contribution is twofold. By directly relating compartmental transition rates to quantifiable SDs, we are able to improve the

estimations of contagion dynamics of COVID-19. Second, we provide the basic structure of a framework that can be used to analyse the effects of SDs on epidemiological modelling for other infectious diseases.

2. An SIRD Model

For completeness we first present an SIRD model which will be extended in the next section.

Assume a population of N individuals who at any point in time t , are split between: Susceptible (S_t), Infected (I_t), Recovered (R_t), and Deceased (D_t) with

$$S_t + I_t + R_t + D_t = N.$$

Here we assume that all infected individuals are also infectious during the time that they are infected. While infected can be split into exposed but not infectious, on one hand, and infectious, on the other (this extension corresponds to the SEIRD model), this would not provide any additional insights with regards to our argument. This is because SDs would only influence the transition from susceptible to exposed and not affect the transition from exposed to infectious.

The key dynamics of the model follow the idea that as individuals get infected the number of susceptible people decreases, and also that the more that people are infected at any point in time the easier it will be for the average susceptible individual to get infected. We also allow for a fraction ε of the recovered individuals to become susceptible per time step. This is captured by the following equation:

$$S_{t+1} = S_t + \varepsilon R_t - \beta(S_t + \varepsilon R_t)I_t / N, \quad (1)$$

Where β captures the average number of contacts per individual at each time step, times the probability of getting infected if meeting an infected individual and is known as the infection rate. Note that $0 \leq \varepsilon \leq 1$ such that if $\varepsilon = 0$ it is equivalent to assuming that all recovered individuals become immune, which means that (1) would become

$$S_{t+1} = S_t - \beta S_t I_t / N.$$

While the latter special case follows the assumption of the recent models for COVID-19 (for example see Prem et al., 2020 or Kucharski et al., 2020), it is known that immunity may last for only a short period (Iwasaki, 2020). Furthermore, while our motivation is the current pandemic, our contribution aims to be more general. For these two reasons we use a more general form of the standard SIRD model.

The simple framework that we use as our starting point here, assumes random mixing between the individuals within the population. This means that the probability of getting infected if susceptible ($\beta(S_t + \varepsilon R_t)I_t$) is the product of β with the number of susceptible and infected individuals and is the same across susceptible individuals. Physical distancing measures will have an effect of reducing the contact between susceptible and infected individuals hence reducing β .

Equation (2) shows that the number of infected individuals in a given period is equal to the number that were infected the previous period plus the newly infected, minus a fraction of infected who have either recovered or become deceased:

$$I_{t+1} = I_t + \beta(S_t + \varepsilon R_t)I_t / N - \gamma I_t, \quad (2)$$

where γ captures the fraction of infected individuals who, per time step change compartment and move to recovered or deceased, hence γ can be understood as the inverse infection period. Put differently, $1/\gamma$ captures the average period that individuals stay infected for some time before “moving” to the next compartment, thus, higher γ corresponds to a shorter infection period. The initial severity of the spread of the disease is given by $R_0 = \frac{\beta}{\gamma}$ also known as the basic reproduction number. The basic reproduction number refers to the time before any measures are imposed; and gives the information of how many individuals are expected to be infected by a single infected person. After measures have been imposed, we refer to the *effective* reproduction number which we denote by R_e .

Let δ capture the case fatality ratio or CFR (the share of infected individuals which become deceased) and $1 - \delta$ the share that recover after being infected, such that the evolution of the number of recovered and deceased individuals is given by the following equations:

$$R_{t+1} = (1 - \varepsilon)R_t + \gamma(1 - \delta)I_t, \quad (3)$$

$$D_{t+1} = D_t + \gamma\delta I_t. \quad (4)$$

Equation (3) states that in every period the number of recovered is increased by the number infected who recovered but is decreased by a fraction ε of recovered individuals who become susceptible again. Equation (4) captures the evolution of the number of deceased individuals.

The system of equations (1)-(4) capture the dynamics of COVID for given values of β , γ , δ and ε . However, as we demonstrate in the next section, both the speed of contagion of COVID-19 and its mortality rate will be significantly influenced by SDs. This means that β and δ can be

understood as functions of different SDs that can be quantified and explicitly included in future analysis. While we acknowledge that the same could be true for γ , which, as we have mentioned, in this framework captures the inverse of the period being infected and infectious, we treat it here as in the standard epidemiological modelling literature.

3. Social Determinants

As noted, a large number of social determinants have been identified and explored in the literature as important factors in shaping health outcomes. It has further been observed that these factors are directly influenced by patterns of social stratification and social hierarchy, including class, income status, gender, and race/ethnicity (Solar and Irwin 2010; Braveman and Gottlieb 2014). Nonetheless, while there exists a wide range of possible SDH that could be incorporated into epidemiological modelling, we have selected three of the most commonly discussed with a clear and explicit bearing on the infection rate and on the CFR. We briefly discuss these SDs, their global variability, and their potential impact on the pandemic.

SD1: Conditions of employment. Unemployment and levels of precarious, low-paid and temporary work are widely recognised in the SDH literature as an important determinant of health outcomes (Stringhini et al 2010; Bartley et al 2015), and public debates around how best to respond to COVID-19 have illustrated the mutually-reinforcing relationship between effective NPIs and conditions of employment. Calls for people to self-isolate when symptomatic – or the enforcement of longer periods of mandatory lockdowns – are economically challenging for those who cannot easily shift their work online, service sector employees who work in zero-hour

contracts, or those engaged in other kinds of short-term employment contracts. Availability of sick pay, childcare, and other forms of employment support also impact the ability of people to comply with NPIs (Chin et al 2020). As a result, conditions of employment bear directly on the degree of social contact between people (hence affecting the infection rate β) as well as the CFR. Recognising the fundamental consequences of these work patterns for public health, many European governments have announced far reaching financial packages to support those made unemployed or forced to stay at home during this crisis (HM Government 2020).

However, in contrast to high income countries, most states around the world do not have the capacity to provide similar levels of financial support. According to the OECD (2019), around 70% of all employment in developing and emerging countries takes place in the informal sector, where labour is unregulated, intermittent, and poorly remunerated. There is substantial variation in informality across regions, ranging from 86% in Africa to around 68% in the Middle East, Asia and the Pacific, and 40% in the Americas (OECD 2019). In these conditions, it is very difficult to implement effective physical distancing through longer periods of lockdown and social isolation because the majority of the population depends upon immediate daily wages for survival and lacks any savings. Indeed, as part of their COVID-19 strategy for developing countries, the International Labour Organization (ILO) has acknowledged that ‘physical distancing measures’ are an ‘impossible choice for informal economy workers’ (ILO April 2020). These problems are compounded by the fact that there will almost certainly be very large increases in the numbers of ‘working poor’ as a direct result of the pandemic – creating a mutually-reinforcing feedback cycle between the pandemic and deleterious conditions of employment (Dimarco et al 2020). All of these features of LMIC labour markets have an impact

on β and δ and, as we demonstrate below, can be incorporated into epidemiological modelling of the virus.

SD2: Conditions of Housing. Conditions of housing encompass issues such as overcrowding and sub-standard buildings, lack of access to safe drinking water and other utilities, ineffective waste disposal, poor quality sanitation infrastructure and inadequate provision of affordable and quality housing stock. The SDH literature has illustrated a clear relationship between housing and deleterious social health outcomes (Krieger and Higgins 2002; Rolfe et al 2020, Kang et al 2020). This is a global issue, but it is one that particularly impacts the estimated 1-1.6 billion people who live in slums and informal housing, a figure that represents around one-quarter of the world's urban population (Habitat for Humanity 2020). Indeed, for some cities in the developing world, the number of people living in slums can reach up to 80% of the total population (WHO 2020).

Moreover, as with conditions of employment, poor housing conditions present severe obstacles for populations attempting to physically distance or undertake quarantine or self-isolation (UN Habitat 2020). Overcrowded and informal housing typically consists of multiple families sharing single dwellings, and intergenerational family units that can bring vulnerable populations into close contact with potential sources of infection. Shared and substandard infrastructures including water, sewage and sanitation, present further potential vectors of infection, a potential that is exacerbated due to high population density and the poor quality of this infrastructure. As a result, conditions of housing can directly impact both the infection rate (β) and the CFR (δ).

SD3: Access to and quality of health infrastructure One of the key concerns raised in the global response to COVID-19 has been access to and overall quality of public health systems, including the availability of hospital beds, adequately trained nurses and doctors, equipment (such as ventilators and oxygen), and the ability of hospitals to scale up critical care capacity. The question of critical care capacity has been particularly prominent in the case of COVID-19, due to the rapid and very large surge in serious and critical cases that typically require hospital admission as a result of the virus. Addressing these capacity issues in order to alleviate potential pressure on hospital infrastructure has been a central facet of strategies to ‘flatten the curve’ (Ferguson et al., 2020).

Discussions around these issues have largely concentrated on the health systems of high-income countries. It is essential to note, however, that health systems in much of the rest of the world have significantly less capacity than developed countries (Murthy et al 2015; Martinez-Alvarez et al 2020; Sousa et al 2020). These intra-country inequalities in health systems directly bear on the ability of states to provide effective treatment support for hospitalised COVID-19 patients (for example, through adequate access to drugs or ICU capacity). The pressures on health systems in LMICs are further compounded by the significant disease burden related to other ‘comorbidities of poverty’ in many of the world’s poorest countries (IPN 2004; Hansen and Painsil 2016; Yang et al 2020; Shiau et al 2020). Moreover, in situations where hospital and primary care settings lack effective PPE or the financial and administrative capacity to implement adequate physical distancing measures, health systems can also become important sites for increased exposure and thus infection. Taken together, these factors indicate how access and quality of health infrastructure can significantly determine both the infection rate and the CFR.

In summary, the three SDs discussed here all have a direct bearing on the CRF and infection rate variables. They are also significantly connected to patterns of social hierarchy and degrees of social marginalisation and exclusion observed across different parts of the world. In this sense, incorporating these (and potentially other) SDs into epidemiological modelling can help elucidate the interaction between health outcomes and other forms of social policy, including the health implications of various social welfare regimes.

4. A Baseline Socio-economic Compartmental Model

Integrating employment conditions into a compartmental epidemiological model can show that higher levels of employment insecurity, informality, and poverty will directly impact the efficacy of physical distancing measures. Below, we formally express the effects of each of the SDs to β and δ through and we also introduce relevant functional forms which allow us to incorporate the SDs into the SIRD model of section 3.

4.1 Integration of Social Determinants

Let the variable c_1 capture SD1, such that higher values of c_1 correspond to higher levels of informal work, poverty, inequality etc., c_2 capture SD2, such that higher values of c_2 correspond to *worse* conditions of housing and c_3 capture SD3 such that higher values of c_3 mean better access to health and better quality of health infrastructure. Then based on the analysis of the previous section both the infection rate β is and the CFR δ are functions of c_1, c_2 and c_3 , with

$$\frac{\partial \beta}{\partial c_i} > 0 \text{ and } \frac{\partial \delta}{\partial c_i} > 0, \text{ for } i=1,2$$

and

$$\frac{\partial \beta}{\partial c_3} < 0 \text{ and } \frac{\partial \delta}{\partial c_3} < 0.$$

The conditions state that that the higher the value for c_1 and c_2 the higher the infection rate and CFR. The opposite is true for c_3 , which has a negative effect on both the infection rate and CFR.

4.2 Total effects

The previous assumptions capture the key elements of the relationship between SDs and the probabilities of moving between different compartments within the SIRD framework presented in the previous section. While the specific functional form of the two parameters with respect to c_1 , c_2 and c_3 would be different across countries and is an empirical question outside the scope of this study, we can assume for simplicity a linear form which satisfies the previous assumptions:

$$\beta = \beta_0 + a_1 c_1 + a_2 c_2 - a_3 c_3, \quad (5)$$

and

$$\delta = \delta_0 + b_1 c_1 + b_2 c_2 - b_3 c_3, \quad (6)$$

where $a_1, a_2, a_3, b_1, b_2, b_3 > 0$ capture the relative importance of SD1, SD2 and SD3 on β and δ respectively; and could be empirically estimated for different countries using different indicators (e.g. labour force engaged in informal work for SD1, proportion of the population living in slums or informal dwellings and/or average housing density for SD2 and ICU beds per capita for SD3) The parameters β_0 and δ_0 capture the rest of the factors which influence the two rates and are not (directly) influenced by SDs. While for δ_0 it is natural to assume that it depends

mainly on the epidemiological characteristics of the disease, β_0 also depends on the NPIs in place.

We should highlight at this point that the linear form of equations (5) and (6) is a reasonable assumption given the focus of the paper on inequalities across countries. However, if the focus would have been within country inequalities, it would have been important to also allow for network effects related to heterogeneity in transmission (for example see Chang et al, 2021, Hébert-Dufresne et al, 2021) which may also make superspreading events more likely in particular communities.

Equations (1)-(6) define a baseline (simple) version of what we can call a socio-economic compartmental (SEC) model.

Given equation (5) the first implication of our baseline SEC model is that the basic reproduction number will be

$$R_0 = \frac{\beta_0 + a_1 c_1 + a_2 c_2 - a_3 c_3}{\gamma}. \quad (7)$$

This shows the effect of two of the SDs to the main variable related to the transmissibility of the virus. Equation (7) also shows that improving SDs will require *ceteris paribus* lower levels of NPIs to reduce the effective reproduction number R_e , such that $R_e < 1$. This means that improving SDs will not only have a direct effect on people's wellbeing but will also have an overall positive effect through leading to a lower R_0 and R_e . Furthermore, given the high economic costs of NPIs, an improvement of SIs will also have economic benefits. Again here,

there might also be indirect network effects which may or may not influence the reproduction number (Hébert-Dufresne et al, 2021).

5. Conclusion

We have shown how three SDs potentially impact the transmission dynamics and severity of COVID-19 across different countries and indicated how these conditions could be integrated into standard SIR-type modelling of the disease.

The approach presented here, can be extended in at least five broad directions.

First, using available data regarding relevant SDs and also the data regarding the contagion dynamics of COVID-19 across countries, it is possible to more closely quantify the effects of the various SDs. This would contribute towards: (i) having a better understanding of the importance of SDs with regards to infections and deaths (ii) estimating the economic benefits of improving relevant SDs and (iii) running policy simulations using the SEC model to analyse the health effects of different policies.

Second, our particular selection of SDs should not be understood as the only possible relevant factors that could be included in compartmental models. Even though our preceding analysis has been largely focused on differences in socio-economic conditions between poorer and richer countries; the same approach could also be used to model the impact of social differentiation within individual countries. Issues such as gender and racial inequalities, educational disparities,

levels of government expenditure on social services, etc. all bear directly on β and δ ; and could also be incorporated into a social compartmental model. Furthermore, as heterogeneity in transmission as a result of SD may also make superspreading events more likely in particular communities, the study of social inequalities while incorporating network effects across social groups along the lines of Chang et al (2021) and Hébert-Dufresne et al (2021) is a particularly interesting research avenue within this theme.

Third, in line with the SDH literature, our argument can help model the potential effect of different social welfare regimes on the progress and consequences of pandemics such as COVID-19. At a policy level, we can better understand the effect of various efforts to tackle inequality and social exclusion across the world, including, for example, policies aimed at reducing employment precarity (SD1), better provision of public housing (SD2), and improving access to public health (SD3).

Fourth, a key contribution here has been to highlight the social components of the models typically used to analyse the epidemic, as well as introduce the basic structure of what we have classified as a global social compartmental model. Given the focus of this paper, we have used one of the most basic compartmental models, namely the SIRD, as the basis of our analysis. Our approach can be extended into other compartmental models that include more compartments and/or other structures like age cohorts (Prem et al., 2020) and/or social groups (Albi et al, 2020a, b) in order to take into account other relevant characteristics of an epidemic and be able to also analyse in detail within country and other intra-group inequalities.

Finally, in this baseline framework, we have assumed that the duration of the period when individuals are infected and infectious is not influenced by SDs. However, we understand that this may not always be the case, hence another research direction can be to investigate the effects of SDs both with regards to the infection period of individuals and the period that they are infectious.

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