The effects of heterogeneous transmission in a COVID-19-like regime

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Abstract

Minimizing infections and deaths in an epidemic are not the same thing. While society has some control on the final number of infected individuals through intervention and mitigation strategies, we may have much greater control over the age-profile of the final cohort of infected individuals. Mitigations which ignore this distinction and focus on minimizing transmission rates equally among the entire population could increase deaths among all age groups compared to carefully crafted alternatives.

We argue for the *heterogeneous transmission thesis* in the context of a infectious disease with a COVID-19-like parameter regime. In the response to a highly transmittable infectious disease with highly age-variable mortality rates, death rates (for all age groups) may be minimized by mitigation strategies which selectively reduce transmission rates in at-risk populations, while maintaining closer-to-normal transmission rates in low-risk populations.

1 Introduction

The basic idea of the heterogeneous transmission thesis is simple: at the end of the COVID-19 outbreak, a significant fraction of the world's population (e.g., at least perhaps 5%-20%) will have been infected. Mitigation strategies can affect this final number of total infections, but perhaps only by a small multiplicative factor (for example, halving them). On the other hand, mitigation strategies also have the potential to shift the age distribution among eventually infected population. Because the mortality rate from COVID-19 between age groups varies by several orders of magnitude (rather than a small multiplicative factor), this can be a much more powerful approach to reducing overall mortality. In particular, this suggests that strategies which are intended to shift the final age distribution—by reducing the transmission rates among older populations more than among younger populations—could have the potential to save the greatest numbers of lives.

The goal of this paper is to argue that heterogeneous transmission could reduce fatalities from a COVID-19-like epidemic under certain assumptions, namely:

Assumption 1. **It is possible to significantly modify the transmissibility of the infection**. In particular, we assume it is possible to to effect different aggregate changes in infection transmissibility for two sub-populations.

Assumption 2. **Worldwide short-term containment is not possible**. We assume that the epidemic will not end without a reduction in the susceptible population large enough to affect overall transmission rates. In particular, we assume that mitigation strategies must be effective even in the face of reintroduction of infection.

Assumption 3. **Near-normal interaction levels will eventually return**. We assume that mitigation efforts to reduce transmission rates cannot continue indefinitely. In particular, in our principal model profile we assume a gradual return to normal transmission levels from the 6 month to 1 year mark. We also consider a regime where normal transmission levels are gradually resumed over the 6 month period from the 18-month to 2-year marks.u

Assumption 4. **Mortality from COVID-19 is dramatically higher in older populations**. Current estimates place the mortality rate for COVID-19 at roughly 50 times higher for people over 70 than those under 50.

Our assumptions depend on political, social, economic, and geopolitical factors, and they may or may not hold in the current COVID-19 fight faced by various governments. Our goal is to demonstrate that these four assumptions are sufficient imply that heterogeneous mitigation strategies may minimize mortalities:

The heterogeneous transmission thesis: Given Assumptions 1-4, heterogeneous mitigation strategies, which target different transmission rates in low- and high-risk population, can reduce mortalities more than homogeneous mitigation strategies which affect transmission rates equally among all subpopulations.

We support the heterogeneous transmission thesis with results from a simple extension of the SIR model which accounts for two sub-populations, where different transmission rates can be achieved in the two sub-populations[1].

The model

Our model is a modification of the basic SIR (**S**usceptible, **I**nfected, **R**ecovered / Removed) model. The basic model describes the dynamics of individuals transitioning across these three states, that is $S \to I \to R$. The transition to the I state is proportional to the interaction between infected and susceptible individuals, that is $I \cdot S$. The infected individuals (I) transition to the R state that represents either the recovered or the removed individuals. In the basic SIR model these two end states are not distinguished since this has no impact

[1] We simplistically refer to these populations as under-65 vs over-65. In reality, mitigation strategies targeting heterogeneous transmission rates would have to recognize other at-risk sub-populations.

on global model dynamics such as the outbreak duration, timing, and peak infections. In our model we are interested in keeping track of mortalities and thus we will have two explicit end states: R for recovered, and M for mortality.

We define the following functions:

$$\begin{array}{rlrl} S(t): & \text{over 65 susceptible} & s(t): & \text{under 65 susceptible} \\ I(t): & \text{over 65 infected} & s(t): & \text{under 65 infected} \\ R(t): & \text{over 65 recovered} & r(t): & \text{under 65 recovered} \\ M(t): & \text{over 65 mortality} & m(t): & \text{under 65 mortality} \end{array} \tag{1}$$

and the following constants:

$$\begin{array}{rlrl} \beta_{uu}: & \text{under 65/under 65 infection rate} & \beta_{uo}: & \text{under 65/over 65 infection rate} \\ \beta_{oo}: & \text{over 65-over 65 infection rate} & & \\ \gamma_u: & \text{under 65 recovery rate} & \gamma_o: & \text{over 65 recovery rate} \\ \delta_u: & \text{under 65 mortality rate} & \delta_o: & \text{over 65 mortality rate} \end{array} \tag{2}$$

and

$$\alpha_u := \gamma_u + \delta_u \tag{3}$$

$$\alpha_o := \gamma_o + \delta_o \tag{4}$$

$$\tag{5}$$

We let S_f the fraction of the total population which is susceptible to infection; N_u, N_o, and N are the under-65, over-65, and total populations, respectively.

2 Model dynamics

Our model follows the SIR model for two populations. The populations interact only via the infected individuals. An infected individual from one population can infect a susceptible individual from another.

$$\frac{dI}{dt} = \beta_{uo} \cdot S(t) \cdot i(t)/N + \beta_{oo} \cdot S(t)I(t)/N - \alpha_o I(t) \tag{6}$$
$$\frac{dS}{dt} = -\beta_{uo} \cdot S(t) \cdot i(t)/N - \beta_{oo} \cdot S(t)I(t)/N \tag{7}$$
$$\frac{dR}{dt} = \gamma_o I(t) \tag{8}$$
$$\frac{dM}{dt} = \delta_o I(t) \tag{9}$$
$$\frac{di}{dt} = \beta_{uu} \cdot s(t) \cdot i(t)/N + \beta_{uo} \cdot s(t)I(t)/N - \alpha_u i(t) \tag{10}$$
$$\frac{ds}{dt} = -\beta_{uu} \cdot s(t) \cdot i(t)/N - \beta_{uo} \cdot s(t)I(t)/N \tag{11}$$
$$\frac{dr}{dt} = \gamma_u i(t) \tag{12}$$
$$\frac{dm}{dt} = \delta_u i(t) \tag{13}$$
$$\tag{14}$$

Explanation: Consider the change $\frac{dI}{dt}$ in the infected over-65 population $I(t)$. It has two terms corresponding to positive changes, let us consider the first. $i(t)/N$ captures the rate at which encounters with random members of the overall population are actually encounters with younger infected individuals. β_{uo} is the constant which translates this rate to the transmission rate to a single over-65 individual, and $S(t)$ is the total susceptible population. The other differential equations have analogous simple interpretations.

3 Correspondence of R_0 with $\beta_{uu}, \beta_{uo}, \beta_{oo}$

To make use of our model in connection with real-world estimates of the transmissibility of COVID-19 it is necessary to make choices of $\beta_{uu}, \beta_{uo}, \beta_{oo}$ which correspond to known ranges of the R_0 value of COVID-19.

In the simple SIR model, there is only one transmission parameter β, one recovery/removal parameter α, and R_0 is the ratio β/α, which is the expected number of new infections which would occur from one initially infected individual in a completely susceptible population.

Our extension of this model collapses to the SIR model in the case that $\beta_{uo} = \beta_{uu} = \beta_{oo}$ and $\alpha_u = \alpha_o$.

Defining $\rho_o = N_o/N$ and $\rho_u = N_u/N$, we assume that at time 0 we have a single infected individual, which corresponds to having $I(0) = \rho_o$ and $i(0) = \rho_u$, while $S(0) = N_o$ and $s(0) = N_u$. That is if 85% of the population is under 65 the n at time 0 we have .85 infected individuals under 65 and .15 infected individuals over 65. In this case we write $\beta := \beta_{uu} = \beta_{uo} = \beta_{oo}$, and we have at $t = 0$ that

$$\frac{dI}{dt}(0) + \frac{di}{dt}(0) = \beta_{oo}\rho_o^2 + 2\beta_{uo}\rho_u\rho_o + \beta_{uu}\rho_u^2 - \alpha_o\rho_o - \alpha_u\rho_u$$
$$= \beta(\rho_o + \rho_u)^2 - \alpha_o\rho_o - \alpha_u\rho_u = \beta - (\alpha_o\rho_o + \alpha_u\rho_u). \quad (15)$$

In particular, the correspondence between $\beta = \beta_{uu} = \beta_{uo} = \beta_{oo}$, $\alpha = \alpha_u\rho_u + \alpha_o\rho_o$ and R_0 are the same for our model as for the SIR model. Thus given, e.g., a target R_{0uu} for the R_0 value we wish to set for transmission within the under-65 population, we translate this to the $\beta_u u$ transmission coefficient via

$$R_{0uu} = \frac{\beta_{uu}}{(\alpha_o\rho_o + \alpha_u\rho_u)}, \quad (16)$$

and similarly for the the coefficients β_{uo} and β_{oo}. In particular, **we translate R_0 values into each transmission coefficient by setting them at the value which would achieve the given R_0 value in the population as a whole if all transmission coefficients were set to that value, reproducing the same R_0 value in the SIR model.**

Correction for S_f

Our model includes the parameter S_f, which is the fraction of the initial population which is susceptible to infection. (As discussed in our main document, our findings about the relative merits of mitigation strategies are not sensitive to this parameter, though it affects total mortality estimates.)

Using $S_f = 1$ corresponds to assuming an initial population which is entirely susceptible to infection. This is the value used when estimating the R_0 value for COVID-19 from observed infection rates. Thus if we assume that in fact S_f is less than 1, we must correct the given R_0 value (we use 2.8) by a factor of $\frac{1}{S_f}$.

Parameters

Our model involves several parameters which we set to the currently best available estimates. Sensitivity to the exact choices of these parameters is explored in a later section.

- **transmission rates between/within under- and over-65 populations:** These determine the rate at which encounters between infected and susceptible individuals lead to new infections. Due to Assumption 3, we assume that after 1 year, these transmission rates will return to levels that imply an R0-value of 2.8 in a completely susceptible population. The R0 value correspond to the median estimate across a several studies summarized in [1].
- **recovery and mortality rates** We assume a rate of recovery based on a 14-day average recovery time [2]. We assume the mortality rate for our

older population is 50 times greater than for our younger population [3, 4]. While the relative mortality rates are much more important for the validity of our conclusions than the absolute rates, we have chosen mortality rates for the two groups which correspond to an overall mortality rate of 0.5

- **medical system capacity** The US is estimated to have a total capacity of 728,000 hospital beds [5]. Of course not all of these beds are available for COVID-19 patients. To model the effects of overburdened medical systems, we assume that above a threshold of 500,000 infected cases, mortality increases by a factor of 2. We show in our sensitivity analysis that our model is not sensitive to these choices.

- **susceptible fraction** We have set our susceptible fraction to 0.25 based on observations of the total infected population in flu pandemics []. Of course the actual value may be much closer to 1 however since non-susceptible individuals do not interact with the other compartments in the SIR model this has no effect on the dynamics of the dynamics, only the absolute numbers.

Results

We consider 5 different model scenarios, each with different management of the transmission rate within age groups. In each model, we assume that after 9 months, transmissibility for both groups begins to return linearly over 3 months to a level which would be equivalent to an R0-value of 2.8 in a completely susceptible population. We allow ourselves to choose the transmission rate within age groups before the 9 month point for each model. In particular, for each model, at any time point, there are two transmission rates; β_{uu} for the under-65 population, and β_{oo} is the level for within the over-65 population. We assume that that the inter-population rate $\beta_{uo} = \min(\beta_{oo}, \beta_{uu})$. In the discussion here, instead of the real rate values we will use their R_0 equivalence R_{0uu} and R_{0oo}, so that 2.8 corresponds to completely unsuppressed transmission.

In Scenario 0 we will let R_{0uu} and R_{0oo} both be 2.8 for the entire simulation. This corresponds to no mitigation strategies being taken. Among all scenarios we consider, this results in the greatest number of fatalities.

In Scenario 1 we will let R_{0uu} and R_{0oo} begin very low (.9) for 9 months, before returning linearly to normal levels. This corresponds to extreme homogeneous measures being taken on the 9-month time scale. This also results in a very large number of fatalities because this scenario simply delays the epidemic dynamics

In Scenario 2 we let R_{0uu} and R_{0oo} be 1.8 for 9 months, before increasing to normal levels. 1.8 is chosen because for our parameter regime, this minimizes mortalities among all possible homogeneous mitigation strategies. Many lives are saved in this scenario, with mortalities dropping by roughly a third.

In Scenario 3 we consider extreme heterogeneous measures. R_{0oo} is controlled .6, while the younger population retains completely normal transmission rates with R_{0uu} at 2.8. Again, transmission rates return to 2.8 linearly between 9 months and a year. Among all scenarios we consider this minimizes the total number of fatalities, with more than a 60% drop from Scenario 0.

Finally, in Scenario 4, we consider a (possibly more realistic) case of heterogeneous measures, where R_{0oo} begins at 1, while R_{0uu} is slightly depressed to 2.4. In this scenario we still see far fewer mortalities than can be achieved by homogeneous measures, with roughly a 50% reduction from Scenario 0.

References

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