

An extended SIR model incorporating population classes

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June 6, 2020

Motivation

The aim of this model is to provide a solid basis to explore the impact of a possible outbreak in Syrian refugees' camps. The model should have sufficient complexity to bring reliable results while being sufficiently general to embrace the diversity of situations that exist in the different camps.

There are a number of considerations with respect to models developed in the context of high income countries. Firstly, the inaccessibility to any kind of health system, makes the consideration of compartments such as "hospitalized" meaningless, and will not be considered here. Also along these lines, we note that epidemiologic parameters found in the literature should be reconsidered to account from a more severe impact of the disease at all levels. Since there is no existing data a careful sensitivity analysis of the model under different scenarios must be performed. To continue with, social interactions within the camps is itself compartmentalized, segregating the activities by gender and age [APG: How true is this assertion, I show an example below], which may be considered when the frequency of contacts are modelled. Lastly, any actions intended to minimize the impact of the spread of the virus should be designed considering these limitations and the social structure. Since the most feasible actions are related to shielding strategies, the complexity of the living conditions should be considered in the models.

To address these challenges, we incorporate in the model the concept of *population class*, which is a subset of the population that we may want to distinguish either because it has different epidemiological parameters (e.g. fatality rates), significantly different frequency in the number of contacts with members that do not belong to their own class, or both. Population classes allow us to generalize previous models that considered stratification of population in classes [REF] or locations [REF], that we can implicitly model via different subsets of parameters. In this way, we can also model the impact of potential management measures like shielding a vulnerable population, or subsetting the population classes like a quarantined population in a separated area of the camp, controlling the probability of contacts with potential carers.

Description of the model

The model starts considers a generic population class i , whose dynamics is described by the model:

$$\dot{S}_i = -\lambda_i S_i \quad (1)$$

$$\dot{E}_i = \lambda_i S_i - \delta_E E_i \quad (2)$$

$$\dot{P}_i = \delta_E E_i - \delta_P P_i \quad (3)$$

$$\dot{A}_i = f_i \delta_P P_i - \gamma_A A_i \quad (4)$$

$$\dot{I}_i = (1 - f_i) \delta_P P_i - (\gamma_{I_i} + \eta_i) I_i \quad (5)$$

$$\dot{V}_i = g_i \eta_i I_i - \alpha_i V_i \quad (6)$$

$$\dot{R}_i = \gamma_A A + (1 - g_i) \gamma_{I_i} I_i \quad (7)$$

$$\dot{D}_i = \alpha_i V_i \quad (8)$$

where all the variables S_i (susceptibles), E_i (exposed), A_i (infectious and asymptomatic), I_i (infections and mild symptomatic), V_i (severe symptoms), R_i (recovered) and D_i (dead) are class-specific. The model is illustrated for a single class in Fig. 1[APG. Note that the lambda parameter is not properly represented, see definition below].

Some of the parameters are also class-specific and are labelled with the subindex i . The λ_i parameter models the transmission rate and will be discussed below. The following parameters are independent of the class: the

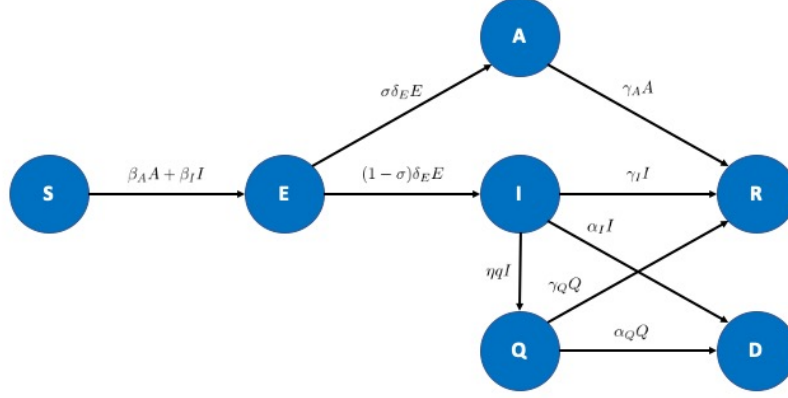


Figure 1: **Schematic representation of the model.** Note that the model does not represent the distinction in classes. In particular, the rate $\beta_A A + \beta_I I$ is only valid if there is one class only. See definition of λ_i in the text.

rate at which exposed get into the latent stage δ_E , and either symptomatic or asymptomatic δ_{\P} and the recovery rate of asymptomatic γ_A .

On the other hand, the following parameters depend on the population class: the fraction of the population that will become infectious and symptomatic after being exposed, f_i , the fraction of infectious that will become critical, g_i , the rate of recovery of symptomatic γ_{Ii} , the rate they will become critical η_i , and the fatality rate of critical cases, α_i . Note that 100% of critical cases will die.

****APG: NOT UPDATED FROM HERE**

Therefore, in the model we are making the following assumptions. Firstly, there is no distinction between being exposed or latent, we considered that every individual exposed becomes latent. This is justified by the fact that the population is well mixed and the latent times are typically low [APG. Is this true?]. Secondly, there is no hospitalized compartment because there is no access to any facility. As a consequence, we are considering that every symptomatic patient may develop a critical condition and hence we do not explicitly consider a gradient of symptoms.

The quarantine variable Q deserves special consideration. In its current form there is a fraction of the population that can be removed at a certain rate. This may make sense in a context in which there is a regulation within the population to become quarantined if certain conditions happen (e.g. being in contact with a symptomatic), in which case the quarantine rate is density-dependent (and hence the term ηQ). On the other hand, if quarantine happens because an external (e.g. WHO agent) remove symptomatics from the camp, the capacity of the agency to remove patients will saturate at some point and it would be more appropriate to model it as a constant or a sigmoidal-like function. In addition, note that in the current form of the model the parameter q is not implemented.

To finish, the transmission rate λ_i depends on the average contacts between the different classes. To determine its value, we define the maximum transmission rates of asymptomatic, β_A , and symptomatic, β_I , and the reduction of these transmission values between the class i and class j with the matrix $C_{ij} \in [0, 1]$. This reduction may be motivated by social constraints (e.g. groups of men not getting in contact with certain groups of women) or management actions (e.g. a class is shielded from the remaining population). Its value represents the fraction of contacts that the classes i and j have with respect to the mean contacts between two individuals in the whole population. Taking these considerations we define the transmission rate of the class i as:

$$\lambda_i = \frac{1}{N} \sum_j C_{ij} (\beta_A A_j + \beta_I I_j + \beta_{\P} P_j) \quad (9)$$

where j runs across all classes including the reference class i .

Estimation of parameters

The basic reproduction number

To estimate the basic reproduction number R_0 we note that it is an adimensional quantity composed of three factors: the transmissibility (i.e. the infection between infected and susceptible given there is a contact), the average rate of contacts and the duration of infectiousness. The first two factors are encoded for each population class in the infection rate λ_i . The duration of infection for each class, τ_i , can be estimated as the inverse of the (weighted) average rates of recovery of symptomatic and asymptomatic compartments for each population class:

$$\tau_i = \left(\frac{\sigma_i \gamma_{Ai} + (1 - \sigma_i)(\gamma_{Ii} + \alpha_{Ii} + \eta)}{\sigma_i + (1 - \sigma_i)} \right)^{-1}.$$

Multiplying for each population class $\lambda_i \tau_i$ and summing up we get that $R_0 = \sum_i \lambda_i \tau_i$, or more explicitly:

$$R_0 = \frac{1}{N} \sum_i \frac{\sum_j C_{ij}(\beta_A A_j + \beta_I I_j + \beta_{\P} P_j)}{\sigma_i \gamma_{Ai} + (1 - \sigma_i)(\gamma_{Ii} + \alpha_{Ii} + \eta)}.$$

Since the β parameters are interpreted as the probability of infection *given* that there is a contact, we considered that these values do not differ from what is found in the literature, and we focus on the values of the matrix C_{ij} as the main difference with respect to more developed human settlements. The rational is that the expected increase in the infection rates is driven by an increase in the average rate of contacts due to the crowded living conditions rather than in an increase of the probability of infection. This approximation will allow us to concentrate in the reduction in the average mean of contacts as the main strategy to control the spread of the infection.

More specifically, we factorize the contacts matrix as $C_{ij} = \bar{C} m_{ij}$ with \bar{C} being the average contacts rate of the population in the absence of infection and m_{ij} the management matrix, representing the reduction in the average contacts rate between class i and j due to an intervention in which both subpopulations reduce their contact. In the absence of any intervention, $m_{ij} = 1 \quad \forall i, j$ so if an estimation of R_0 is given and assuming that $\beta_A = \beta_I = \beta_{\P}$ we can estimate the average contacts rate with the expression:

$$\bar{C} = \frac{NR_0}{\beta} \left(\sum_i \frac{\sum_j A_j + I_j + P_j}{\sigma_i \gamma_{Ai} + (1 - \sigma_i)(\gamma_{Ii} + \alpha_{Ii} + \eta)} \right)^{-1}$$

Examples

Shielding vulnerable population

In this example exist only two classes, one vulnerable subpopulations and one healthy, differing in their epidemiological parameters. Then we considered two scenarios, one in which both populations are well-mixed, and a second scenario in which the average number of contacts between the healthy and vulnerable subpopulations are reduced to half the whole population values. The dynamics of both populations for both scenarios are shown in Fig. 2. We observe that the total number of deaths are reduced from 55 to 51.

Socially-structured population with shielding and carers

As a second proof of concept we consider now a more complex example in which the population is divided in three age-levels (young, adults and elderly). Starting population values were proportional to Syrian structure, and total population size is 2000. In addition, within each age-level there are two additional classes, healthy and vulnerable. The fraction of vulnerable population within each age class increases with age. Finally, each class is divided by gender. This leads to 12 population classes and 82 epidemiological variables.

We consider again one scenario in which the population is well-mixed, and where there is one infection starting in the class o adult healthy men. The second scenario considers two contact limitations. Firstly, healthy women have contacts only with their respective partners from the class of healthy men. Considering an average number of contacts in the whole population of 20, we reduce the transmission parameters between these subpopulations to 1/20. In addition, we shield the elderly vulnerable classes (male and female) from all the other subclasses except the one of adult healthy women, which will be considered the carers, again with a parameter 1/20.

The death toll for the different subclasses in both scenarios is shown in Fig. 3. Without shielding there is a 61% more deaths, and it becomes apparent that the reduction comes from the shielded population at age 3.

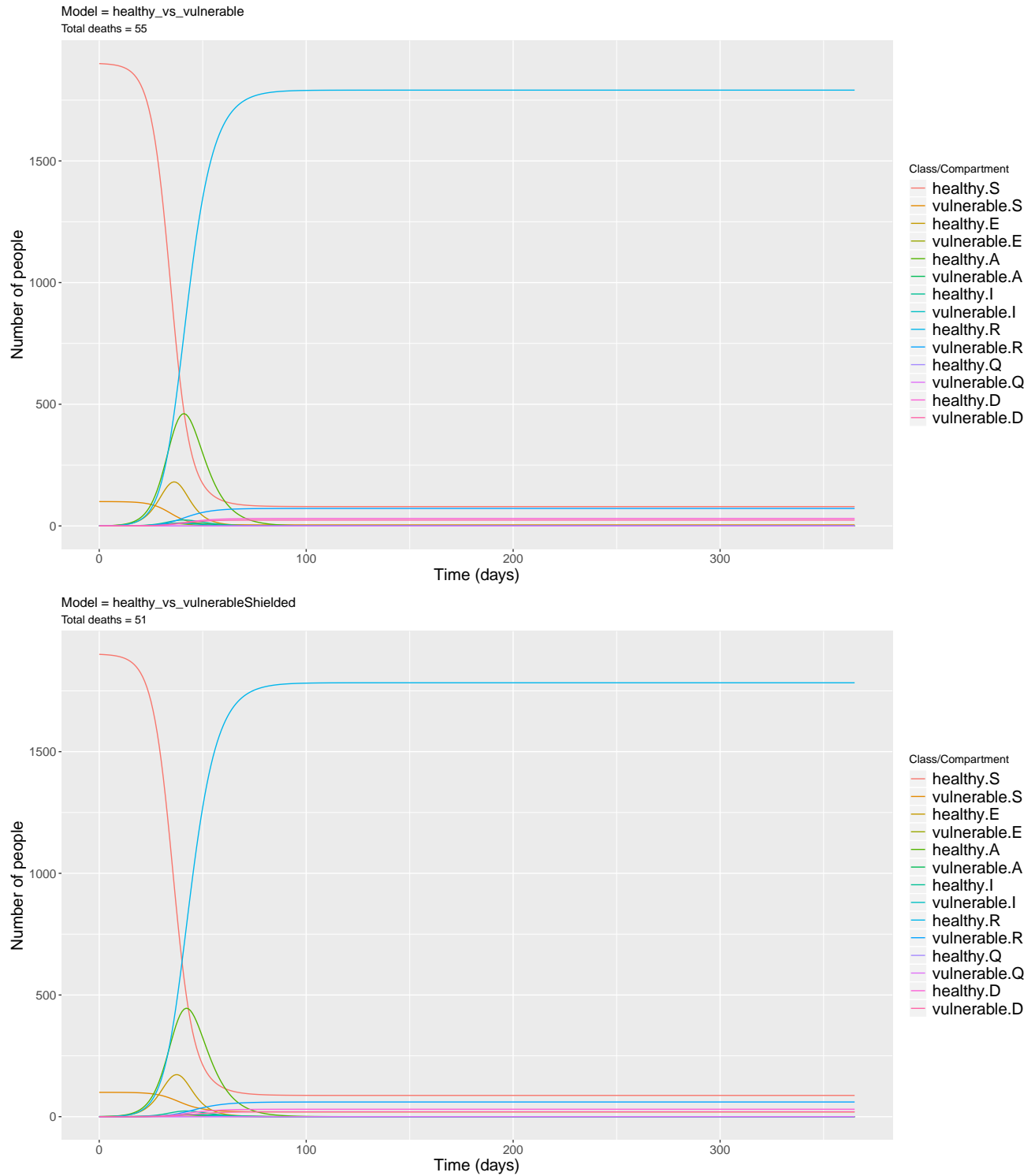


Figure 2: **Population dynamics of healthy and vulnerable subpopulations.** (Top) Well mixed scenario. (Bottom) Average contact between healthy and vulnerable populations are reduced to half their value. Total number of deaths are reduced from 55 to 51.

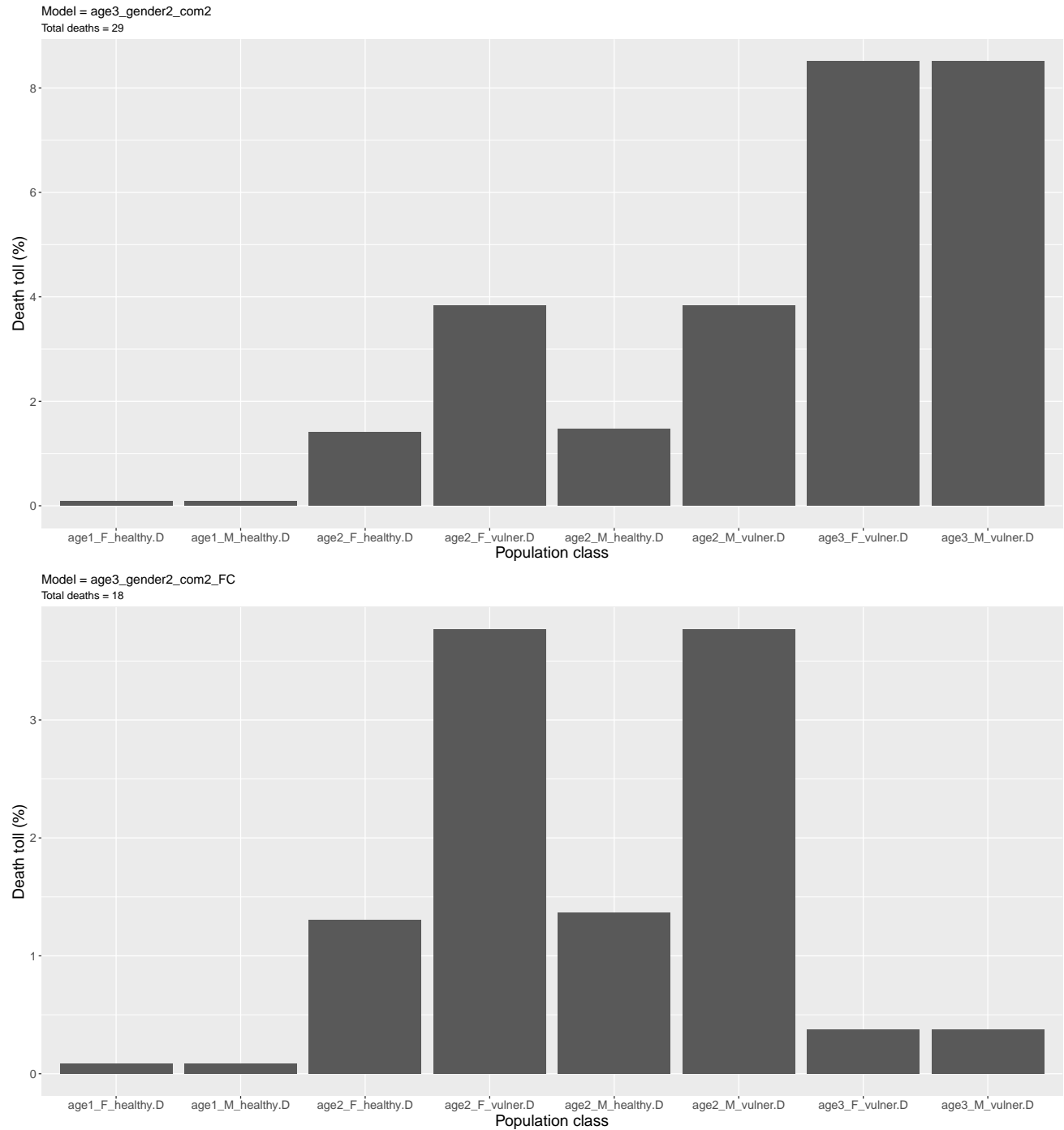


Figure 3: **Death tolls for a population with a strong social structure.** (Top) Well mixed scenario. (Bottom) A socially-structured shielding strategy is implemented (see Main Text for details). Note the change in the scale between both figures.