Empowering the crowd: feasible strategies for epidemic management in high-density informal settlements. The case of COVID-19 in Northwest Syria

Alberto Pascual-García, Jordan D Klein, Jennifer Villers, Eduard Campillo-Funollet, Chamsy Sarkis

ABSTRACT

More than 1 billion people live in informal settlements worldwide, where precarious living conditions pose unique challenges to managing a COVID-19 outbreak. Taking Northwest Syria as a case study, we simulated an outbreak in high-density informal Internally Displaced Persons (IDP) camps using a stochastic Susceptible-Exposed-Infected-Recovered model. Expanding on previous studies, taking social conditions and population health/structure into account, we modelled several interventions feasible in these settings: moderate self-distancing, self-isolation of symptomatic cases and protection of the most vulnerable in ‘safety zones’. We considered complementary measures to these interventions that can be implemented autonomously by these communities, such as buffer zones, health checks and carers for isolated individuals, quantifying their impact on the micro-dynamics of disease transmission. All interventions significantly reduce outbreak probability and some of them reduce mortality when an outbreak does occur. Self-distancing reduces mortality by up to 35% if contacts are reduced by 50%. A reduction in mortality by up to 18% can be achieved by providing one self-isolation tent per eight people. Protecting the most vulnerable in a safety zone reduces the outbreak probability in the vulnerable population and has synergistic effects with the other interventions. Our model predicts that a combination of all simulated interventions may reduce mortality by more than 90% and delay an outbreak’s peak by almost 2 months. Our results highlight the potential for non-medical interventions to mitigate the effects of the pandemic. Similar measures may be applicable to controlling COVID-19 in other informal settlements, particularly IDP camps in conflict regions, around the world.

INTRODUCTION

The spread of airborne infectious diseases with pandemic potential in regions immersed in protracted armed conflicts, with large displaced populations, is an important challenge. When the displaced population exceeds official resettlement and refugee camp capacity, internally displaced persons (IDPs) must live in informal settlements (hereafter named ‘camps’). These regions must contend with the public health challenges resulting from violence, the deterioration of health systems, especially of critical care, and the breakdown of essential public infrastructure such as water and sanitation...
systems. Urgent action is needed to contain the spread of disease in these settings, a task which necessarily involves the engagement of the communities living in them.

This study focuses on the spread of COVID-19 in the Northwest region of Syria (NWS): a relatively small geographical area with 4.2 million people, of which 1.15 million (27.4%) are IDPs living in camps, and where the number of cases increased 20-fold between 8 September and 20 October 2020. The health status of households in camps in NWS is poor; 24% have a member with a chronic disease, of whom 41% have no access to medicines. As in other conflict regions, the political instability in NWS hinders coordinated public health actions, and the ongoing movements of IDPs create ample opportunity for infectious disease transmission, while making contact tracing interventions infeasible.

To investigate feasible COVID-19 prevention interventions in the camps, we considered a Susceptible-Exposed-Infectious-Recovered model similar to the one presented by Gatto et al. in which the camps’ populations are divided into classes reflecting their estimated age structures and comorbidity prevalence. We use this model to propose various interventions aimed at reducing the number of contacts within and between population classes in general, and with symptomatic individuals in particular. We paid special attention to how the living conditions in informal camps inform the assumptions underlying our proposed interventions, a question often neglected. We modelled interventions previously proposed for African cities, such as self-isolation of symptomatic individuals and the creation of a ‘safety zone’ in which more vulnerable members of the population are protected from exposure to the virus.

Building on the approach used to model the impact of these interventions in African cities, our model includes a parameterisation of the number of contacts each individual has per day. We further elaborate on this approach by making a more explicit representation of contacts and other parameters in the model. We consider the microdynamics of contacts, the effect of having carers to attend to isolated individuals, and the existence of a buffer zone in which exposed and protected population classes can interact under certain rules. We examine a potential worst-case scenario in which there is no access to any healthcare facility. Since empowering local communities in conflict regions to understand how to control diseases like COVID-19 is possibly the most (and perhaps only) effective way to minimise its spread, our models are of utmost importance for informing the implementation of realistic interventions in these regions.

**METHODS**

**The model**

We consider a model simulating a viral outbreak in a single camp over a 12-month period inspired by those proposed by Gatto et al. and Bertuzzo et al. (see figure 1). The model is adapted to the context of NWS IDP camps and is divided into compartments containing individuals at different possible stages along the disease’s progression, governed by the following set of differential equations:

\[ \dot{S}_i = -\lambda S_i \]  
\[ \dot{E}_i = \lambda S_i - \delta E_i \]  
\[ \dot{P}_i = \delta E_i - \delta P_i \]  
\[ \dot{A}_i = (1 - \alpha f) \delta P_i - \gamma_A A_i \]  
\[ \dot{H}_i = \beta P_i - (\gamma_H + \gamma_I + g) A_i I \]  
\[ \dot{I}_i = f \beta P_i - (\gamma_H + \gamma_I + g) A_i I \]  
\[ \dot{R}_i = \gamma_A A_i + (1 - \alpha f) \gamma_H H_i \]  
\[ \dot{D}_i = g \alpha I_i + \sigma \gamma_H H_i \]

The susceptible population \( S_i \) becomes exposed at rate \( \lambda \), while exposed individuals \( E_i \) progress through the latent period at rate \( \delta \) to a preclinical infectious stage \( P_i \), which then progresses to (at rate \( \delta_p \)) either a clinical (symptomatic, \( I_i \), with probability \( f \)) or subclinical (asymptomatic, \( A_i \), with probability \( 1 - f \)) infectious stage. Asymptomatic cases recover \( R_i \) at rate \( \gamma_A \). Symptomatic cases have three potential outcomes: mild cases will recover at rate \( \gamma_I \), severe cases will progress to an...
Finally we run simulations considering two possibilities: either compartment is uncertain if healthcare is not available, and behaviour to account for heterogeneity with respect to clinical risk realistically evaluate our proposed interventions. More- computational tractability and the requisite specificity to and strike an appropriate balance between generality, and us to encode behavioural assumptions in the model (see section Interventions and online supplemental sections considering cultural and context- within population classes allows us to design interven- over, the explicit representation of contacts between and considered a stochastic implementation, with an integer of ordinary differential equations (Equations 1–8) we description of the population in which a state is encoded a classical system of ordinary differential equations (Equations 1–8) we considered a stochastic implementation, with an integer description of the population in which a state is encoded in a vector $X = (S, E, P, I, A, H, R, D)$, with the total popu- size conserved throughout the simulation $N = S + E + P + I + A + H + R + D$, where an arrow indicates transitions in which the source compartment transfers one individual to the target compartment. The mean transition rates corresponding to each transition are displayed. The system then evolves following a continuous-time Markov process which is simulated following the Gillespie algorithm implemented in the R package adaptivetau.  

**Demographic and behaviour classes**

The model splits the population into classes (indexed $i$) to account for heterogeneity with respect to clinical risk and behaviour. Working with population classes allows us to encode behavioural assumptions in the model and strike an appropriate balance between generality, computational tractability and the requisite specificity to realistically evaluate our proposed interventions. Moreover, the explicit representation of contacts between and within population classes allows us to design interventions considering cultural and context-specific assumptions (see section Interventions and online supplemental material for details). Under a null model where no interventions are implemented, the distinctions between classes are only dependent on age and comorbidity status (hereafter ‘demographic classes’). $h_i$, $g_i$, and $l_i$ are demo- graphic class-specific parameters, adjusted to ensure that the proportions of symptomatic cases progressing through each of the three potential clinical outcomes (mild, severe, and critical) are consistent with the literature (see section Epidemiological severity assumptions).

Under some interventions, the demographic classes may be subdivided further into subclasses according to behaviour (‘behaviour classes’). Consequently, different interventions may require models with different numbers of classes. We refer to both demographic and behaviour classes generically as ‘classes’ (see section Interventions for the modelling of behaviour classes).

**Population structure and demographic classes**

We parameterised the model with data from IDPs in NWS. The population sizes of informal camps are skewed, with a mean of 1212. We simulated camps with populations of 500, 1000 and 2000 individuals. Since interventions tend to be less effective in larger camps, the results presented refer to simulations with 2000 individuals, unless otherwise specified. For our demographic classes, we considered three age groups: children (age 1, 0–12 years old), younger adults (age 2, 13–50 years.) and older adults (age 3, 50 or more). For ages 2 and 3, we considered two subclasses comprising healthy individuals and individuals with comorbidities (see table 2).

**Transmissibility assumptions**

Although individuals in IDP camps share tents with other co-occupants, whom they may be more likely to infect than occupants of different tents, we ignore spatial

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**Table 1 Fixed parameters**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
<th>Value</th>
<th>Distribution</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>$1/\delta_E + 1/\delta_P$</td>
<td>Incubation period (days)</td>
<td>5.2 (95% CI 4.1 to 7.0)</td>
<td>Lognormal</td>
<td>45</td>
</tr>
<tr>
<td>$1/\delta_P$</td>
<td>Presymptomatic infectious period (days)</td>
<td>2.3 (95% CI 0.8 to 3.8)</td>
<td>Gaussian</td>
<td>18, 46</td>
</tr>
<tr>
<td>$1/\delta_E$</td>
<td>Latent period (days)</td>
<td>$1/\delta_E + 1/\delta_P - 1/\delta_P$ (Maximum=0.5 days)</td>
<td>Derived</td>
<td></td>
</tr>
<tr>
<td>$1/\gamma$</td>
<td>Symptomatic infectious period (days)</td>
<td>7</td>
<td>---</td>
<td>18, 47</td>
</tr>
<tr>
<td>$1/\gamma_A$</td>
<td>Asymptomatic infectious period (days)</td>
<td>7</td>
<td>---</td>
<td>18, 47</td>
</tr>
<tr>
<td>$1/\eta$</td>
<td>Time from symptom onset to requiring hospitalisation (days)</td>
<td>7 (IQR: 4–8)</td>
<td>Gamma</td>
<td>48</td>
</tr>
<tr>
<td>$1/\alpha$</td>
<td>Time from symptoms onset to death (critical cases, days)</td>
<td>10 (IQR: 6–12)</td>
<td>Gamma</td>
<td>48</td>
</tr>
<tr>
<td>$1/\gamma_H$</td>
<td>Time from requiring hospitalisation to recovery/death (days)</td>
<td>10 (IQR: 7–14)</td>
<td>Gamma</td>
<td>48</td>
</tr>
<tr>
<td>$f$</td>
<td>Probability an infectious individual is symptomatic</td>
<td>0.84 (95% CI 0.8 to 0.88)</td>
<td>Gamma</td>
<td>49</td>
</tr>
<tr>
<td>$\sigma$</td>
<td>Indicator of whether hospitalised recover or die</td>
<td>$\sigma \in {0, 1}$</td>
<td>---</td>
<td>Assumed</td>
</tr>
</tbody>
</table>

See online supplemental material for details.
structure in our model and assume a well-mixed population. This is justified because individuals from different tents share common spaces (e.g. latrines) and have frequent interactions with each other, especially among children. Consequently, our following derivation of the transmissivity parameter itself, \( \tau \), is not spatially explicit.

The rate at which susceptible individuals become exposed is

\[
\lambda_i = \sum_{j=1}^{n} C_{ij} \beta_{ij} \frac{I_j \tau A_j + I_j \tau H_j}{N_j}
\]

where \( C_{ij} \) is the average number of contacts that individuals of class \( i \) have with individuals of class \( j \) per day and \( N_j \) is the total population size of class \( j \). We parameterised \( C_{ij} \) by multiplying the mean number of total contacts that individuals from a population class \( j \) have per day, \( c_i \), by the probability of random interaction with individuals of class \( j \). Considering a well-mixed population, this probability is proportional to class \( j \)'s fraction of the total population, that is, \( C_{ij} = c_i N_j / N \). If interventions are absent, we consider demographic classes only and, hence, different values of \( c_i \) reflect heterogeneity in the number of contacts by demographic class. We assume specific values of \( c_i \) for each class based on conversations with camp managers in NWS (see table 2).

The probability of infection if there is a contact between a susceptible and an infected person is \( \tau \beta_r \), \( \tau \beta_s \), \( \tau \beta_i \), or \( \tau \beta_h \) depending on whether the infected individual is in the presymptomatic (\( P \)), symptomatic (\( I \)), asymptomatic (\( A \)) or hospitalised compartment (\( H \)), respectively. The \( \tau \) parameter represents the maximum transmissivity, which is observed at the presymptomatic stage for individuals who go on to become symptomatic.\(^{18}\) Thus, we selected the transmissivity of these individuals as a reference \((\beta_{r-1} = 1)\) with the remaining parameters set relative to \( \beta_{r-1} \) \((\beta_r < \beta_{r-1}, r \in \{P, A, H, I\})\), where the mean transmissibility of all presymptomatic individuals \((\beta_r)\) is estimated as a weighted average of the transmissibility of individuals that will become symptomatic and asymptomatic (see table 3, online supplemental materials for derivation).

The \( \tau \) parameter was estimated by randomly generating a value for the basic reproduction number, \( R_0 \), following a Gaussian distribution with a mean of \( 4 \) (99% CI 3 to 5) and dividing this value by the dominant eigenvalue of the next-generation matrix (see section Computational implementation for details and online supplemental material for the analytical results). The distribution of \( R_0 \) was a compromise between values reported in the literature from regions with high-density informal settlements: \( R_0 = 2.77 \) in Abuja and 3.44 in Lagos, Nigeria,\(^{19}\) 3.3 in Buenos Aires\(^{20}\) and 5 in Rohingya refugee camps in Bangladesh.\(^{21}\)

### Table 2 Demographic class-specific parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
<th>Age 1 (0–12)</th>
<th>Age 2 (13–50) no comorbidities</th>
<th>Age 2 (13–50) comorbidities</th>
<th>Age 3 (&gt;50) no comorbidities</th>
<th>Age 3 (&gt;50) comorbidities</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fraction in class</td>
<td>–</td>
<td>0.407</td>
<td>0.471</td>
<td>0.0626</td>
<td>0.022</td>
<td>0.0373</td>
<td>17 50</td>
</tr>
<tr>
<td>( c_i ) Mean contacts per day</td>
<td>25</td>
<td>15</td>
<td>15</td>
<td>10</td>
<td>10</td>
<td>From camp managers</td>
<td></td>
</tr>
</tbody>
</table>

Estimated proportions of individuals in the population and mean number of contacts per individual per day for each demographic class. See online supplemental materials for derivations.

### Table 3 Transmissibility parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
<th>Value</th>
<th>Distribution</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \tau ) Maximum transmissibility</td>
<td>0.14 (95% CI 0.05 to 0.40)</td>
<td>Lognormal</td>
<td>Derived</td>
<td></td>
</tr>
<tr>
<td>( \beta_{r-1} ) Presymptomatic transmissibility of individuals becoming symptomatic relative to ( \tau )</td>
<td>Reference stage (=1)</td>
<td>–</td>
<td>18 46</td>
<td></td>
</tr>
<tr>
<td>( \beta_r ) Mean presymptomatic transmissibility relative to ( \tau )</td>
<td>0.93 (95% CI 0.88 to 0.99)</td>
<td>Empirical</td>
<td>Derived</td>
<td></td>
</tr>
<tr>
<td>( \beta_s ) Asymptomatic transmissibility relative to ( \tau )</td>
<td>0.14 (95% CI 0.05 to 0.40)</td>
<td>Lognormal</td>
<td>Derived</td>
<td></td>
</tr>
<tr>
<td>( \beta_i ) Clinical symptomatic transmissibility relative to ( \tau )</td>
<td>0.24 (95% CI 0.11 to 0.60)</td>
<td>Lognormal</td>
<td>Derived</td>
<td></td>
</tr>
<tr>
<td>( \beta_h ) Hospitalised transmissibility relative to ( \tau )</td>
<td>0.11 (95% CI 0.05 to 0.29)</td>
<td>Lognormal</td>
<td>Derived</td>
<td></td>
</tr>
</tbody>
</table>

See online supplemental materials for details.
Since the rates at which these cases become severe, critical or recover are different, we introduced three parameters \( (h, g, \text{ and } l) \) to distribute individuals according to the desired proportions (values between parenthesis). The proportion of individuals recovering is computed as \( 1 – q^H – q^D \) (see online supplemental materials for details).

### Epidemiological severity assumptions

In NWS, there are four active and two planned COVID-19 referral hospitals, 74 ICU beds and 355 ward beds for 4.2 million people. Estimations based on an exponential growth model from Hariri et al predicted a collapse of health facilities 8 weeks into an outbreak. Although we do not have access to official data on healthcare occupancy, the reported number of cases suggests that this scenario could have been reached. Hence, we considered a worst-case scenario in which individuals will not have access to healthcare and assumed that all critical cases (those requiring ICU care) would die. However, there is greater uncertainty about the fate of severe cases, those requiring hospitalisation but not ICU care. We therefore considered a compartment for severe cases to account for a longer infectious period if they stay in the camp (see compartment \( H \), figure 1). This compartment also helps us model some interventions more realistically, for example, by noting that the symptoms of severe cases are incompatible with self-isolation. To estimate upper and lower bounds for the outcome variables of our model, we simulated two possible scenarios for the fate of this compartment: one in which all cases recover, and another in which all cases die. In the simulations presented in the Main Text, we consider the worst-case scenario in which all cases die.

The fractions of symptomatic cases that are severe \( (q^H) \), critical \( (q^D) \) and recover \( (q^R) \) are demographic class-specific (see table 4). We estimated the fractions of symptomatic cases in each demographic class that would become severe \( (q^H) \) and critical \( (q^D) \) using data from developed countries with superior population health. Following previous work, we mapped the age-specific case severity distributions of the NW Syrian adult population to those of age groups 10 years older in developed countries.

Since the rates at which clinical symptomatic individuals \( (I) \) resolve into these three epidemiological outcomes are different \( (\eta \text{ for } H, \alpha \text{ for } D \text{ and } \gamma \text{ for } R) \) we introduced three parameters, \( b, g \text{ and } l \), to distribute individuals according to the desired proportions. The analytic derivation is provided in online supplemental material and the specific values in table 4.

### Interventions

The interventions we consider are modelled by modifying the rate at which individuals become exposed (the term \( \lambda \), Eq. 9), and/or adding new population classes that govern behavioural changes (behaviour classes). Since \( \lambda \) can be factorised in four terms, the interventions may influence one or several of these terms. The factors present in \( \lambda \) and the terms modulating them in the interventions are (see Eq. 10): (1) the maximum transmissibility, \( \tau \), which is reduced in some interventions by a factor \( \xi \), when interactions are restricted to buffer zones (see below for details); (2) the average number of contacts that individuals in class \( i \) have per day, \( c_i \). This quantity can either be uniformly reduced across all classes, or the contact rate of class \( i \) with class \( j \) can be modified. We model this modification of contact rates using the matrix \( \omega \); (3) the probability of encounter between members of class \( i \) and \( j \) in a well-mixed population, \( N_j = N \). This probability can vary by modifying the visibility of a member of class \( j \) to a member of class \( i \), which we express with the matrix \( \omega \); and (4) the probability of becoming infected by individuals at specific stages of the disease (eg, for hospitalised individuals this is encoded in the term \( \beta H_i/N_i \) which can be modified by specific factors. Only the terms for individuals at the clinical symptomatic \( (I) \) and hospitalised \( (H) \) stages are modified in our interventions, through the parameters \( \zeta \) and \( \zeta_i \), respectively. Following these considerations, the generic form of \( \lambda_i \) under the interventions becomes:

\[
\lambda_i = \sum_j N_j (\beta H_i + \beta A_j + \zeta_i \beta I_i + \zeta_i \beta H_i) / N_i
\]

All interventions, self-distancing (see figure 2-1), self-isolation (see figure 2-2), safety zone (see figure 2-3) and evacuation (see figure 2-4) can be parameterised following this expression. The specific values of the parameters are presented in table 5 and their derivations in online supplemental materials.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
<th>Age 1 (0–12)</th>
<th>Age 2 (13–50) no comorbidities</th>
<th>Age 2 (13–50) comorbidities</th>
<th>Age 3 (&gt;50) no comorbidities</th>
<th>Age 3 (&gt;50) comorbidities</th>
</tr>
</thead>
<tbody>
<tr>
<td>( q^H(h_i) )</td>
<td>Fraction of symptomatic cases severe</td>
<td>0.064 (0.064)</td>
<td>0.067 (0.066)</td>
<td>0.199 (0.191)</td>
<td>0.183 (0.178)</td>
<td>0.445 (0.406)</td>
</tr>
<tr>
<td>( q^D(g_i) )</td>
<td>Fraction of symptomatic cases critical</td>
<td>0.0065 (0.009)</td>
<td>0.020 (0.028)</td>
<td>0.094 (0.129)</td>
<td>0.063 (0.088)</td>
<td>0.222 (0.289)</td>
</tr>
</tbody>
</table>
Self-distancing

The first non-medical intervention that we modelled is a reduction in the mean number of contacts per individual per day for the whole camp population (see figure 2-1). The average number of contacts of each class \( c_i \) (see table 2) is reduced by a class-independent factor, hence the matrix \( c_i \) is uniform for all classes, that is, \( c_i = c, \quad c \in \{0.9, 0.8, 0.7, 0.6, 0.5\} \). No further adjustments are required for this intervention. Since the mean number of inhabitants per tent in a camp is 5.5 and sanitation facilities are shared,\(^1\) we inferred that the number of contacts per day cannot be reduced by more than 50% (\( c = 0.5 \)). For a younger adult, this would mean 7.5 contacts per day.

Self-isolation

Self-isolation is a challenge in informal settlements, where households consist of a single (often small) space, water is collected at designated locations, sanitation facilities are communal and food supplies are scarce. We considered the possibility of those showing symptoms (i.e. in compartment I) self-isolating in individual tents in dedicated parts of the camps. We excluded individuals with severe symptoms (H compartment) from the intervention since they require additional care not compatible with self-isolation. Instead, we considered the possibility of evacuating these individuals from the camp as an additional intervention (see below). We simulated self-isolation with various numbers of isolation tents per camp, ranging from 10 to 2000 for a camp of 2000 people (see figure 2-2a). In addition, we modelled the role of carers dedicated to providing for isolated individuals (see figure 2-2a). Carers are drawn from the younger adults’ class with no comorbidities who are not at a stage of the disease during which they display symptoms (i.e. not in compartments I or H), while isolated individuals may belong to any class. Since this intervention only modifies the infectiousness of individuals in the I compartment and the exposure of carers, the consideration of additional behavioural classes is not required. Instead, the intervention can be encoded in \( \lambda_i \) by simply splitting the contribution of symptomatic individuals to the rate of exposure into two terms, one for the isolated individuals and another for the remaining population. Hence, its implementation requires deriving the parameters \( c_i \) and \( \omega_{ij} \) for interaction between healthy younger adults (from which carers are drawn), the remaining classes, and isolated and non-isolated individuals separately. In table 5, we present these terms and their derivation in online supplemental materials.

In addition, interactions between carers and isolated individuals were restricted to buffer zones, which we envisioned as open spaces, with guidelines in place to limit occupancy to four individuals wearing masks with at least 2 m of distance between them, where we assume transmissivity is reduced by 80% (\( \xi = 0.2 \)). In considering one carer per isolated individual with one contact per day, we do not neglect their probability of infecting the rest of the camp.

Safety zone

In this intervention, the camp is divided in two areas: a safety zone, in which more vulnerable people live (hereby referred to as a ‘green’ zone following previous studies),\(^1\) and an exposed (‘orange’) zone with the remaining population. In our simulations, the first exposed individual always belongs to the orange zone. The living conditions within both zones remain the same, so the overall contact rate does not change unless self-distancing is also implemented. A consequence of maintaining the overall contact rate is that reducing contacts with individuals living in a different zone implies an increase in
### Table 5  Parameterisation of the interventions

<table>
<thead>
<tr>
<th>Intervention</th>
<th>$\epsilon_{ij}$</th>
<th>$\omega_{ij}$</th>
<th>$\zeta_{I}$</th>
<th>$\zeta_{H}$</th>
<th>$\xi_{ij}$</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Self-distancing</td>
<td>$\in {0.9,0.8,0.7,0.6,0.5}$</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>Constant $\forall i,j$</td>
</tr>
<tr>
<td>Self-isolation</td>
<td>$(c_{\text{care}}/c_i) (\tilde{I}_i/N_i)$</td>
<td>$N_i/N_t$</td>
<td>1</td>
<td>1</td>
<td>0.2</td>
<td>$i \in$ young healthy adults</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>1</td>
<td>$\Theta(N_i - \tilde{N})$</td>
<td>1</td>
<td>$j \in$ isolated individuals in class $j$</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0</td>
<td>0</td>
<td></td>
<td>0</td>
<td>$j \in$ individuals not isolated in class $j$</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>1</td>
<td></td>
<td>1</td>
<td>$i \not\in$ young healthy adults</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>$j \in$ isolated individuals in class $j$</td>
</tr>
<tr>
<td>Safety zone</td>
<td>$\vartheta c_{\text{visit}}/c_i$</td>
<td>$N_i/N_Y$</td>
<td>(0,1) see (*)</td>
<td>(0,1) see (*)</td>
<td>0.2</td>
<td>$i \in$ zone $X, j \in$ zone $Y$</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>($X, Y \in {\text{green, orange}}$)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>$i,j \in$ zone $X$</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>($X \in {\text{green, orange}}$)</td>
</tr>
<tr>
<td>Evacuation</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

Values of $\epsilon_{ij}, \omega_{ij}, \zeta_{I}, \zeta_{H}$ and $\xi_{ij}$ considered in the interventions (see online supplemental materials for their derivations). $c_{\text{visit}} = \text{number of contacts per day of each isolated individual with carers. } N_i = \text{Total number of isolated cases. } \tilde{N} = \text{Number of tents available for self-isolation. } \tilde{I}_i = \text{Number of isolated cases of class } j. \Theta(x) = \text{Heaviside function. } c_{\text{visit}} = \text{maximum number of contacts that individuals in the green zone can have with individuals from the orange zone per day (}c_{\text{visit}} = \left\{ \frac{2}{15}, \frac{2}{7} \right\}). \vartheta = \text{fraction of the population in the orange (green) zone capable of coming in contact with individuals from the green (orange) zone. } \vartheta = 1 \text{ if class } i \text{ is in the green zone and is proportional to } c_{\text{visit}} N_i/N_t \text{ if in the orange zone. } N_X \text{ and } N_Y \text{ generically refer to the total population in either the green (}N_i\text{) or the orange (}N_t\text{) zones.} *\text{The parameters are set to 0 if health checks to access to the buffer zone are implemented in the intervention and set to 1 otherwise.}
contacts with individuals in the same zone (see online supplemental material). Although we do not expect this assumption to be true in general, it allows us to investigate undesired side effects of this intervention, such as older adults having increased contacts among themselves if isolated together. Since proposals for partitioning the population may be received differently across camps, we considered several scenarios for allocating a camp population to the two zones (see figure 2-3). Implementing this intervention thus requires the split of some demographic classes into two behaviour classes, depending on the scenario. For example, if some healthy younger adults are allocated into the green zone, we split the demographic class ‘healthy younger adults’ into ‘orange’ and ‘green’ behaviour classes, to model the different contact rates that these two subclasses of healthy younger adults will have among themselves and with other classes. In online supplemental table 2, we present the classes considered in each scenario.

Interactions between the two zones are limited to a buffer zone, reducing transmissivity (i.e. \( \xi \) = 0.2, see previous section). Individuals in the green zone cannot leave and thus need to be provided with supplies by individuals in the orange zone, which will take place in the buffer zone. In our simulations, we considered limiting individuals in the green zone to 10 or 2 contacts with individuals from the orange zone per week (see figure 2-3a). Other variations of this intervention we explored include preventing symptomatic individuals from entering the buffer zone (incorporating health checks, see figure 2-3b) and a ‘lockdown’ of the green zone, where the number of weekly contacts in the buffer zone is reduced by 50% or 90% (see figure 2-3c). Although overall contact rates are conserved in this intervention, we modify the contact rates between individuals in the same or different zones (see table 5 for specific values and online supplemental materials for derivation).

Evacuation
The last intervention we simulated is the evacuation of severe cases (individuals in the hospitalisation compartment). Since they require more intensive care that cannot be delivered while adhering to the guidelines of a buffer zone, severe cases were assumed to be fully infectious and not able to self-isolate. Once severe cases are evacuated, their infectivity is reduced to zero (\( \zeta \) = 0, see figure 2-4). The fate of severe cases is not altered by this intervention since we assumed that hospitals are saturated and that evacuees are transferred to isolation centres instead.

Computational implementation and statistical analysis
The specific values of the parameters shown in Eqs. 1-8 and of \( R_0 \) are independently drawn at each integration step from the probability distributions shown in tables 1, 3 and 4. We note that by generating transition rates from the empirically determined residence times, we are not reproducing these residence times for the simulated individuals. In our simulations, individuals will experience exponentially-distributed residence times with a mean equal to the mean of the corresponding distribution (to 1/\( \kappa \) in the case of the symptomatic compartment, see online supplemental material 1 for the definition of \( \kappa \)). The generation of random values around the mean following the empirical distribution at each time step is aimed at adding noise to the mean, to partially account for the empirical uncertainty.

The next-generation matrix is also computed at each integration step from the parameters drawn and \( r \) estimated. In the code provided, it is possible to fix the seed to exactly reproduce the results presented. Our simulations start with a completely susceptible population where one person in the younger adult population is exposed to the virus (who is also in the orange zone if the safety zone intervention is in place). We verified that a steady state was always reached before the end of each simulation. We did not consider migration, births, nor deaths due to other causes, since they are small enough in magnitude to not significantly impact the course of an outbreak, provided additional conflict does not erupt.

For each implementation of the interventions, we ran 2500 simulations and compared results between them. The main variables considered are the fraction of simulations in which at least one death is observed (a proxy for the probability of an outbreak), the fraction of the population that dies and the time until the symptomatic population peaks, as well as the infection fatality rate (IFR), and the fractions of the population that have recovered and remain susceptible at steady state. For consistency, we only considered simulations in which there was an outbreak when comparing the outcome of a variable between interventions. We used the Shapiro-Wilk test\(^{27}\) to verify that our results do not exhibit normally distributed residuals, and Conover-Iman test for multiple comparisons.\(^{28}\) We used the R package PMCMRplus.\(^{29}\) Confidence intervals for the probability of outbreak were computed with Wilson’s method\(^{30}\) implemented in the R package binom.\(^{31}\) The model and all statistical analyses were implemented in R.\(^{32}\)

RESULTS
In the absence of interventions, the mean IFR is \(~ 2.5\%\) in simulations where all severe cases requiring hospitalisation recover (see online supplemental figure 1), and \(~ 11\%\) in simulations where all severe cases die. We consider the latter scenario to evaluate the effect of non-medical preventive interventions. In this scenario, the probability of observing an outbreak is close to 0.84, in which \(~ 10\%\) of the camp dies, the number of symptomatic cases peaks after 40 days, \(~ 84\%\) of the population recovers, and \(~ 5\%\) remain susceptible.

Self-distancing
Our results show that self-distancing has a notable effect on reducing the probability of an outbreak which decreases roughly linearly as the number of contacts
is reduced (see figure 3A). A near-quadratic trend is observed for the fraction of the population dying, with a greater decrease for larger reduction in daily contacts, achieving a 34% reduction in mortality when the number of contacts is reduced by 50% (see figure 3B). Self-distancing also significantly extends the time until the peak of the outbreak, from ~40% days when no interventions are in place to 72 days when contacts are reduced by 50% (see figure 3C). The proportion of the population remaining susceptible after 12 months also increases to nearly 33% when a 50% reduction is considered, and the proportion of the population recovered after 12 months (which informs on the potential population level protection against future outbreaks) is reduced from 84% to 60% (see online supplemental figure 2).

Self-isolation
With only 10 tents for a camp of 2000 people (i.e. 1 tent for every 200 people), self-isolation yields a marked decrease in the probability of observing an outbreak (~26%) (see figure 3D) and a low decrease in mortality (~7%) (see figure 3E), suggesting that with a low number of tents the intervention is mostly effective at isolating index cases and preventing the epidemic from starting. In order to observe a greater mortality reduction (~18%) and an increase in time (~6%) until the number of symptomatic cases peaks, further increasing the number of tents up to at least one tent for every eight people is required. Increasing the number of tents does not further reduce the probability of observing an outbreak. There is also an increase in the number of susceptible individuals at the end of the simulation when the number of tents is increased. We finally observed an artificial increase in the IFR explained by an increasingly large number of simulations in which very few individuals are infected since, if at least one of them dies, we obtain a high IFR value (see online supplemental figure 3).

Evacuation
We observe no significant effects when severe cases requiring hospitalisation are evacuated (see online supplemental figure 4). Since we considered that these individuals will not receive healthcare (they are evacuated to isolation centres), their fate remains the same as if they were to stay in the camp. Hence, we expect evacuation to only have an effect on their infectivity. Although these individuals are infectious for a longer period of time than those with milder symptoms (~10 days longer), the
number of individuals under these conditions is only a small fraction of the total infectious population at any given time, explaining why we do not observe significant effects for this intervention.

Safety zone
In this section, we consider the scenario in which all older adults, younger adults with comorbidities and their family members up to 20% of the camp population live in the green zone, unless otherwise specified. Creating a green zone improves the effect of the previous interventions overall, but with sometimes opposite outcomes in the exposed and protected populations. For example, the probability of an outbreak decreases for the protected population, by around 11%, if only two contacts are allowed per week in the buffer zone (see figure 3G). Notably, most of this reduction is only achieved when health checks excluding symptomatic individuals from the buffer zone are in place (see online supplemental figure 5 for the effect of removing health-checks). On the other hand, the probability of an outbreak may slightly increase for the exposed population, a consequence of the relative increase in intra-zone contacts. By shifting the burden of an outbreak towards the less vulnerable population in the orange zone, another important outcome of this intervention is the notable increase in time (62%) until the number of symptomatic cases peaks for the vulnerable population, and a 36% increase in time for the whole population (see figure 3I). Nevertheless, this intervention only has a modest reduction on overall mortality (see figure 3H and online supplemental figures 6 and 7), possibly due to the high infectiousness of presymptomatic individuals.

Considering different scenarios for allocating people to the green zone, the lowest probability of an outbreak is achieved when only older adults or at most older adults and younger adults with comorbidities move there, with probabilities below 0.4 and 0.65, respectively (see online supplemental figure 8). Positive effects of the safety zone intervention are even more marked in camps with smaller populations, especially for probability of an outbreak in the green zone (which decreases by 55%) and overall mortality (which decreases by 20%) when the population is reduced from 2000 to 500. However, we also observe the adverse effect of a decrease in time until symptomatic cases peak (see online supplemental figure 9). The incorporation of a lockdown has the greatest effect on reducing the probability of an outbreak in the green zone, to under 0.25 when contacts in the buffer zone are reduced by 90%. While lockdowns show no positive effect on green zone fatalities in the few instances where an outbreak does reach there, they decrease overall IFR and fatalities by further concentrating outbreaks in the less vulnerable population (see online supplemental figure 10).

Combined interventions
The effects of the interventions observed when we examine them individually build on each other when multiple interventions are implemented in tandem (see figure 4 and online supplemental figure 11). The protective effects of the safety zone intervention are most fully realised when paired with other interventions. They become so effective that outbreaks in the green zone become exceptionally rare, but so well controlled when they do happen, that the majority of outbreaks are small enough for us to observe an anomalous increase in IFR in some of the most effective interventions, driven by the discretisation of the values it can take (e.g. if there is only one case and this person dies, see online supplemental table 13). When all interventions are implemented together: strict self-distancing (50% reduction in contacts), self-isolation of symptomatic cases (1 tent for every 40 people), a safety zone with 2 contacts per week in the buffer zone, health checks, a strict lockdown (90%) and evacuation of severe cases, mortality is reduced by ~94% and the probability of outbreak in the green zone is very small (~0.005). In other combinations with a higher probability of outbreak (e.g. considering in the previous combination a 20% reduction in contacts instead of 50%) the time to peak of symptomatic cases in the green zone is delayed by 48 days.

DISCUSSION
In this study, we propose a number of interventions of immediate applicability to informal settlements. We focused on IDP settlements in NW Syria, taking into account the interventions’ feasibility, cultural acceptance and their need for low cost. When confronted with different possible scenarios, we generally considered the worst-cases, highlighting the interventions that are most effective in the direst conditions, but possibly resulting in an overestimate of mortality. This potential overestimation does not change the qualitative picture of the results, which is built on the relative comparison of outcomes between different combinations of interventions, or the lack thereof.

Our results align with previous simulation studies of potential COVID-19 interventions in similarly densely populated, low-resource settings where informal settlements are present, such as urban areas of sub-Saharan Africa. In these settings, social distancing is demonstrated to be an effective intervention, and even small changes are estimated to have large effects on outbreaks in some cases determining whether or not already inadequate healthcare systems become overwhelmed. van Zandevoort et al show that similar measures to the ones we consider—self-isolation, physical distancing and ‘shielding’ the vulnerable—may reduce mortality by 60%–75% in African cities. Self-distancing proves to be an effective measure in our models as well; reducing contacts by 50% has the greatest effect across most outcomes of interest in any of the interventions we examined. However, the difficulty of achieving a reduction of this magnitude cannot be overlooked, especially considering the large proportion...
of the population composed of children, a group with an already high contact rate that may prove difficult to control.\textsuperscript{17} To illustrate the microdynamics of this intervention, in online supplemental figure 12, we plot the maximum proportion of the population exposed at any given point in time in each simulation, against the time it takes for symptomatic cases to peak. We observe that until reaching approximately 60\%, successive reductions in contacts reduce the maximum proportion of the population exposed while increasing the time until symptomatic cases peak. Additional reductions in contacts beyond 60\% however abruptly decrease both the time until cases peak and overall mortality, suggesting that outbreaks die out before the virus spreads widely throughout the camp’s population. This suggests the existence of a critical threshold for the number of individuals exposed over which large outbreaks become established in the population, significantly increasing mortality.

We also propose self-isolation using individual tents which can be located in a dedicated zone or next to the tents of relatives, where contact with non-isolated individuals is mediated by a buffer zone. This intervention is effective in preventing an outbreak in the camp with even a small number of isolation tents, as low as 5–10 tents per 1000 camp residents. But it requires at least 125 tents per 1000 camp residents to substantially reduce mortality. After conversations with camp managers, we found that this intervention is more likely to be accepted in NW Syria than evacuation to community-based isolation centres. Community-based isolation not only poses cultural challenges; the capacity required to implement it has hardly been met,\textsuperscript{23} and it is still one of the main challenges in

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**Figure 4** Combinations of interventions. Probability of an outbreak (top), fraction of the population dying (middle) and time until peak symptomatic cases (bottom) for different combination of interventions. For combinations of interventions including a safety zone, we distinguish between the population living in the green zone, in the orange zone and the whole population. Evac = evacuation of severely symptomatic; lock = lockdown of the buffer zone; safety = safety zone; self = self-distancing; tents = number of available self-isolation tents.
the region. We note that in our simulated intervention individuals become isolated as soon as they have symptoms. Recognising symptoms, however, may require some time and we should expect this intervention to be less effective unless systematic checks for symptomatic individuals are put in place.

Setting up a safety zone has two positive effects that most stand out: a reduction in the probability of an outbreak in the vulnerable population, and an increase in the time until the number of symptomatic cases peaks. Much of the success or failure of the safety zone intervention hinges on the functioning of the buffer zone. The number of interzone contacts per week, the implementation of health checks, and potential lockdowns all have notable effects. Also important is the portion of the population that is protected; protecting only the vulnerable may have the most beneficial effects, but it is precisely these vulnerable individuals, older adults, and people with comorbidities, who may most need family members to care for them. While safety zone scenarios with more family members accompanying their vulnerable relatives may confer greater epidemiological risk, they may also engender greater well-being and social cohesion.

Despite these benefits, we do not observe a clear decrease in IFR with this intervention, although it is possible that our model may overestimate mortality from an outbreak in the green zone in the few instances when there is one. Since it is unlikely that the camps have the economic means to increase the number of tents when implementing this intervention, we assumed that individuals do not reduce their contacts when moved to the green zone since household sizes will not decrease, which implies an increase in the number of contacts between vulnerable individuals. Despite this increase in contacts, we do not observe an increase in mortality in the vulnerable population when the safety zone is implemented. These results address concerns raised around this type of intervention from previous experiences with large numbers of fatalities registered in nursing-homes in developed countries. While nursing-homes in developed countries may be seen as analogous to the safety zone intervention, the alternative to nursing homes in developed countries for the elderly population typically involves remaining at home with few contacts with younger individuals (in a scenario of lockdown), while in the camps the alternative is living in tents shared with younger individuals with high contacts rates (especially children). This may explain why we observe a positive effect from this intervention despite a relative increase in contacts among the most vulnerable subpopulation.

An instrumental consideration for our models is the fraction of the population recovered from COVID-19 after a steady state is reached. Although the duration for which SARS-CoV-2 infection confers immunity is uncertain, the proportion of the population recovered after an outbreak should play a role in its protection against future ones. For all interventions except self-distancing >30%, we observed that the fraction of the population recovered meets or exceeds 50%.

Other important considerations for interpreting our results are the modelling assumptions we made. One important parameter in our model is the relative transmissibility of the different infectious stages, whose specific values still have large margins of variability. This is particularly relevant for non-medical interventions that rely on the identification of symptomatic individuals because the higher the relative infectivity of presymptomatic and symptomatic individuals, the lower the effectiveness of such interventions. For instance, Bullock et al assumed a higher infectiousness of the presymptomatic stage and hence self-isolation of symptomatic individuals had little effect. Self-isolation also becomes ineffective under the assumptions made by Hernandez-Suarez et al when they considered isolating only severe symptomatic cases (whose fraction is small), hence mildly symptomatic individuals were effectively considered asymptomatic. On the other hand, Gilman et al showed that self-isolation was effective when considering individuals at different stages to be equally infectious. We assumed that presymptomatic individuals have the highest relative infectivity, consistent with the most recent estimations and the interventions we proposed are still effective. Considering a different scenario in which all compartments are assumed to be equally infectious, our interventions become even more effective (see previous version of our manuscript).

It is also important to acknowledge the benefits and limitations of different possible computational implementations. For instance, there are interventions that do not have a natural implementation within our framework, such as those requiring interventions targeting very specific interactions between individuals (as opposed to large groups of individuals), or the reproduction of empirically observed residence times. An example might be the isolation of an individual and his/her family, as proposed by Gilman et al which, since we do not explicitly model interactions at the family level, would require the creation of as many classes as families. When this level of detail is required, individual based models (IBMs) may be more appropriate.

However, IBMs require a rich amount of data for their parameterisation which, although increasingly available, is scarce for informal IDPs camps. Our framework is powerful enough to simulate a large number of scenarios with little computational cost, which would be an optimal strategy as a first approximation in the design of interventions to narrow down the most relevant scenarios (for a reference, in <24 hours with just 12 cores we model 75 scenarios requiring quarter million simulations). The scenarios selected could then be further investigated with more detailed interventions using IBMs, if data is available.

A key limitation of our approach is that it simulates an outbreak started by one infectious individual in a single camp with a closed population. We acknowledge that this approach does not fully capture the complexities of the NWS
region, where IDPs live interspersed throughout the region in several hundred camps. The dynamics of an outbreak in the region are undoubtedly influenced by inter-community contacts, and the dynamics of an outbreak in a single camp by these region-wide dynamics, as it has been demonstrated in other countries.\cite{1,2} We expect our results to be robust to changes in population, as long as these changes are relatively small compared to the total population size in the camp, implying sporadic inputs of infected individuals. This is the expected behaviour in informal IDP camps, which are often small and located in rural areas, where substantial population movements such as those observed in large camps, are infrequent. This fact, in addition to the relatively young population in IDP camps, may help limit the damage done by an outbreak, as observed in some rural areas in sub-Saharan Africa.\cite{27}

Other unaccounted for social and cultural dynamics will undeniably complicate the feasibility of our proposed interventions. One example we have not addressed here is the unlikeliness of children under 13 self-isolating. Although the number of challenges to implementing our proposed interventions are potentially endless, the community-based nature of our approach may make it more robust to such challenges than approaches relying on healthcare system, which often depend on complex political decisions and may take years to build the required capacity for an effective response. If the dynamics of the virus are well understood by local communities and at least some of the interventions we propose are implemented, the impacts of COVID-19 can be mitigated even in an environment as challenging as NW Syria.

CONCLUSION

Given a rapidly changing environment and slow responses of local and international authorities in conflict regions where political control is disputed, with international authorities often leaving these communities aside in their priorities,\cite{4} empowering local communities themselves is perhaps the best, if not the only way, to help them avoid the worst consequences of the pandemic. Such an approach may achieve greater compliance with non-medical interventions, especially where there is a mistrust of external authority. This not only applies to IDP camps in NW Syria, but more generally to refugee camps in conflict-torn regions, and potentially other informal settlements and vulnerable communities around the world: the low cost, effective interventions we present are feasible, needed and urgent.

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Supplementary Methods and Results
Empowering the crowd: feasible strategies for epidemic management in high-density informal settlements.
The case of COVID-19 in Northwest Syria

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1 Parameterization of the model

1.1 Derivation of fixed parameters (Table 1 in Main Text)

To estimate the latent period ($1/\delta_0$), we calculated the difference between randomly generated incubation ($1/\delta_\text{p} + 1/\delta_\text{v}$) and presymptomatic ($1/\tau$) periods. We estimated the presymptomatic period using results reported by He et al. [1] and found they best fit a Gompertz distribution with a mean of 2.3 days (95% CI: 0.8-3.0). Since a correction of these by Ashcroft et al. [2] suggests they significantly underestimate the presymptomatic period’s upper bound, we estimated that the true presymptomatic period should rather be closer to a Gaussian distribution around the mean (95% CI: 0.8-3.8). However, this presymptomatic period distribution implies a non-negligible probability of a negative latent period. To correct this discrepancy, we assumed a minimum latent period of 0.5 days [3]. Time from symptom onset to death in critical cases ($1/\alpha$) is estimated using time from symptom onset to ICU admission in Wang et al. [4].

1.2 Population structure of demographic-classes derivation (Table 2 in Main Text)

In April, 2020, 40.7% of the population in informal IDP camps in Northern Syria was aged 0-12, 53.4% aged 13-50, and 5.9% aged 51+ [5]. To estimate the proportion of each age group with comorbidities, we calculated the weighted average age-specific comorbidity prevalence of the 4 most common comorbidities in the Syrian refugee populations and 5.9% aged 51+ [5]. To estimate the proportion of each age group with comorbidities, we calculated the weighted average age-specific comorbidity prevalence of the 4 most common comorbidities in the Syrian refugee populations in Jordan and Lebanon: hypertension, cardiovascular disease, diabetes, and chronic respiratory disease [6, 7]. We standardized these weighted averages to the age structure of IDPs in Northern Syria and estimated that 11.7% of people aged 13-50 have comorbidities, while 62.9% of people aged 51+ have comorbidities.

1.3 Derivation of transmissibility parameters

The probability of infection if there is a contact between a susceptible and an infected person depends on the stage of the disease, denoted $\tau P \beta_P$, $\tau A \beta_A$, $\tau H \beta_H$ or $\tau V \beta_V$ depending upon whether the infected individual is in the presymptomatic ($P$), symptomatic ($I$), asymptomatic ($A$), or hospitalized compartment ($H$), respectively. We estimated these parameters in two steps. In the following section, we estimate the $\beta_X$ parameters ($X \in \{P, A, I, H\}$) which represent the relative transmissibility of each stage with respect to the maximum transmissibility $\tau$. After this calculation, we present our estimate for the maximum transmissibility parameter $\tau$.

Relative transmissibilities $\beta$ (Table 1 in Main Text)

We start by considering the transmissibility of the presymptomatic stage for those individuals who become symptomatic as a reference ($\beta_{P \rightarrow I} = 1$), since the probability of infection from contact with an individual at this epidemiological stage is highest [8]. Next, we set the contribution of each epidemiological stage to infectivity as proportional to $\beta_X/\gamma_X$, with $1/\gamma_X$ the duration of stage X. Following He et al, we estimate the proportion of infectivity in individuals who go on to develop symptoms that occur at the presymptomatic stage ($X \in \{P\}$) as the area under the infectivity curve prior to symptom onset, $AUC_P$, and the proportion of infectivity that occurs at asymptomatic stages ($X \in \{A, I, H\}$) as the area under the infectivity curve after symptom onset, $1 - AUC_P$ [8]:

$$
AUC_P \approx \frac{\beta_{P \rightarrow I}}{\gamma_P} + \frac{\beta_{A \rightarrow I}}{\gamma_A} + \frac{\beta_{H \rightarrow I}}{\gamma_H}.
$$

We then considered the quantity $\rho_{HI}$, the ratio of the viral culture positive test rate in hospitalized patients 7-16 days since start of symptoms to the positive test rate in patients 0-6 days since start of symptoms from van Kampen et al. [9]. Similarly, the relative risk of asymptomatic transmission to symptomatic transmission according to Byambasuren et al. is expressed as $\rho_{AI}$ [10]:

1. $\beta_A = \rho_{AI} \beta_I$
2. $\beta_H = \rho_{HI} \beta_I$

Considering these relationships we rewrite Eq. 1 to obtain the desired parameters:

$$
AUC_P = \frac{\beta_A \gamma_A}{\gamma_P \gamma_A} + \frac{\beta_H \gamma_H}{\gamma_P \gamma_H}.
$$
\[ \beta_I = \frac{\beta_{P \rightarrow I} \gamma_I \gamma_H (1 - \text{AUC}_P)}{\text{AUC}_P \delta_P (\gamma_H + \rho_H \gamma_I)} \]  
\[ \beta_A = \frac{\rho_A \beta_{P \rightarrow A} \gamma_I \gamma_H (1 - \text{AUC}_P)}{\text{AUC}_P \delta_P (\gamma_H + \rho_H \gamma_I)} \]  
\[ \beta_H = \frac{\rho_H \beta_{P \rightarrow H} \gamma_H (1 - \text{AUC}_P)}{\text{AUC}_P \delta_P (\gamma_H + \rho_H \gamma_I)} . \]  

The values of \( \text{AUC}_P, \rho_A \) and \( \rho_H \) are presented in Table 1, and the values of \( \beta_X \) in Main Text. Noting that the starting point for these derivations is the transmissibility of individuals in the presymptomatic stage who go on to develop symptoms (\( \beta_{P \rightarrow I} = 1 \)), we need to go back and derive the transmissibility of all presymptomatic individuals (\( \beta_P \)), taking those that will be asymptomatic into account. We assumed that the relative transmissibility of presymptomatic individuals who will become asymptomatic to those that will become symptomatic, is equal to the relative transmissibility of asymptomatic to symptomatic individuals (\( \rho_A \)). With the proportion of presymptomatic cases that will become developed symptoms \( f \) (encoded in in Eqs. 1–8 in Main Text), we compute the mean transmissibility of all presymptomatic individuals as:

\[ \beta_P = f \beta_{P \rightarrow I} + (1 - f) \rho_A \beta_{P \rightarrow A}. \]  

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
<th>Value</th>
<th>Distribution</th>
<th>Reference</th>
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<td>( \text{AUC}_P )</td>
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<td>0.44 (95% CI: .30-.57)</td>
<td>Gaussian</td>
<td>[8]</td>
</tr>
<tr>
<td>( \rho_A )</td>
<td>Ratio of asymptomatic to symptomatic infectiousness</td>
<td>0.58 (95% CI: .34-.99)</td>
<td>Lognormal</td>
<td>[10]</td>
</tr>
<tr>
<td>( \rho_H )</td>
<td>Ratio of hospitalized to symptomatic infectiousness</td>
<td>0.48</td>
<td>-</td>
<td>[9]</td>
</tr>
</tbody>
</table>

**Table 1: Relative transmissibility parameters.**

**Maximum transmissibility parameter \( \tau \)**

In the following, to simplify the notation we define \( \kappa_i = (\lambda_1 \gamma_I + h_i \eta + g_i \alpha) \). To estimate the probability of infection if there is a contact between a susceptible and an infected individual (parameter \( \tau \)) we proceed as follows [11, 12, 13].

We start by considering the subsystem containing the infected population:

\[ \dot{E}_i = \lambda_2 S_i - \delta_E E_i \]  
\[ \dot{P}_i = \delta_E E_i - \delta_P P_i \]  
\[ \dot{A}_i = (1 - f) \delta_P P_i - \gamma_A A_i \]  
\[ \dot{I}_i = f \delta_P P_i - \kappa_i I_i \]  
\[ \dot{H}_i = h_i \eta I_i - \gamma_H H_i . \]  

For the sake of simplifying the notation, let us consider the following ordering of the variables in the vector \( \mathbf{x} = (E_1, ..., E_M, P_1, ..., P_M, A_1, ..., A_M, I_1, ..., I_M, H_1, ..., H_M) \), with \( M \) the number of population classes. We are interested in the parameterization of the null model, which will serve as a baseline to estimate the parameter \( \tau \), which is initially unknown, but does not change when interventions are introduced. Considering the contacts matrix for the null model (Eq. 9 in Main Text), the rate of exposure becomes

\[ \lambda_i = \frac{\tau}{N} \sum_{j=1}^{M} c_{ij} (\beta_P P_j + \beta_A A_j + \beta_I I_j + \beta_H H_j). \]
In the following, we use bold symbols for vectors and matrices, and the symbols ⨂ and ⊘ for the element-wise multiplication and division, respectively. Following this notation, the linearized system can be written in the form \( \dot{x} = (T + \Sigma)x \), where:

\[
T = \begin{bmatrix}
0 & \Theta_P & \Theta_A & \Theta_l & \Theta_R \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0
\end{bmatrix}
\]

(14)

is the transmission matrix, with \( \Theta_X = \beta_X \text{diag}(p \circ c)U \), \( p = N/N \), \( U \) is the all-ones matrix of size \( M \), and \( \beta_X \) the infectiousness of compartment \( X \) relative to the presymptomatic compartment (see Main Text for details). The transition matrix is

\[
\Sigma = \begin{bmatrix}
-\delta_E I & 0 & 0 & 0 & 0 \\
\delta_E I & -\delta_P I & 0 & 0 & 0 \\
0 & (1 - f)\delta_P I & -\gamma_A I & 0 & 0 \\
f\delta_P I & 0 & -\text{diag}(\kappa)I & 0 & 0 \\
0 & 0 & 0 & \eta\text{diag}(h)I & -\gamma_I I
\end{bmatrix}
\]

(15)

Where \( I \) and \( 0 \) are the identity and null matrices of size \( M \), and \( \kappa = l\gamma_I + h\eta + g\alpha \). We next compute the inverse of the transition matrix

\[
\Sigma^{-1} = \begin{bmatrix}
\frac{1}{\delta_E} I & 0 & 0 & 0 & 0 \\
-\frac{1}{\delta_E} I & \frac{1}{\gamma_A} I & 0 \gamma_A & 0 & 0 \\
-\frac{1}{\gamma_A} I & -\frac{1}{(1 - f)} I & -\frac{1}{\gamma_A} I & 0 & 0 \\
-f\text{diag}(\kappa^{-1})I & -f\text{diag}(\kappa^{-1})I & 0 & -\text{diag}(\kappa^{-1})I & 0 \\
-\frac{h\alpha}{\gamma_I} \text{diag}(h \circ \kappa)I & \frac{h\alpha}{\gamma_I} \text{diag}(h \circ \kappa)I & 0 & -\frac{h\alpha}{\gamma_I} \text{diag}(h \circ \kappa)I & -\frac{h\alpha}{\gamma_I} I
\end{bmatrix}
\]

(16)

The NGM with large domain can now be found by \( K_L = -T\Sigma^{-1} \). However, since we know that each individual who gets infected becomes exposed (\( E \) compartment), we focus on the NGM with small domain, \( K_S \), which only consists of the \( E \) compartment [14]. We do this by removing the rows that correspond to the other compartments from \( T \) and the columns from \( \Sigma^{-1} \). We then find:

\[
K_S = \tau \begin{pmatrix}
\frac{1}{\delta_E} \Theta_P + \frac{(1 - f)}{\gamma_A} \Theta_A + f\text{diag}(h^{-1}) \Theta_I + \frac{\eta}{\gamma_I} \text{diag}(h \circ \kappa) \Theta_H
\end{pmatrix}.
\]

(17)

The reproduction number is related to the dominant eigenvalue of \( K_S \), i.e. \( R_0 = |\lambda_1| \), and \( \tau \) is estimated from the real dominant eigenvalue of \( \tilde{R}_S = K_S/\tau \). Considering the null model parameters (\( \lambda_1^0 \)), we have the expression:

\[
\tau = \frac{R_0}{|\lambda_1^0|}.
\]

(18)

### 1.4 Epidemiological severity proportions (Table 4 in Main Text)

In the Main Text, we presented the proportions in which clinical symptomatic individuals resolve into critical (\( q^D \)), severe (\( q^H \)) and recovered (\( q^R \)) cases. We assigned the fractions of symptomatic cases in children aged <13 that would become severe and critical from the fractions of symptomatic cases in children aged <11 that were severe and critical in China [15]. We assigned the class-specific fractions of symptomatic cases in adults that would become severe and critical based on age and comorbidity-specific fractions of symptomatic cases with known outcomes that required hospitalization, without and with ICU admission, respectively in the United States [16]. To account for poorer health among Syrian adults compared to their similarly aged peers in developed countries, estimates for US adults aged 19-64 were used for Syrian adults aged 13-50, while estimates for US adults aged 65+ were used for Syrian adults aged 51+.
Since the rates at which these individuals progress are different \((\eta_i \text{ for } H, \alpha_i \text{ for } D \text{ and } \gamma_i \text{ for } R)\) we introduced three parameters, \(h_i\), \(g_i\) and \(l_i\), to distribute individuals according to the desired proportions following the equations:

\[
q_i^H = h_i \eta_i \kappa_i^{-1}
\]

\[
q_i^D = g_i \alpha_i \kappa_i^{-1}
\]

\[
q_i^R = 1 - q_i^H - q_i^D = l_i \gamma_i \kappa_i^{-1},
\]

where \(\kappa_i = h_i \eta_i + g_i \alpha_i + l_i \gamma_i\). The system has three unknowns and three equations but one equation linearly depends on the other two, hence we introduce the constraint \(l_i = 1 - h_i - g_i\) to solve the system as:

\[
h_i = \frac{\alpha q_i^H}{\gamma q_i^D} g_i,
\]

\[
g_i = \gamma_i \left( \frac{\alpha}{q_i^D} + \frac{\gamma q_i^H}{\eta q_i^D} + \frac{\gamma_i}{q_i^D} - \alpha \right)^{-1}.
\]

## 2 Parameterization of the interventions (Table 5 in Main Text)

### 2.1 Safety zone

We considered the existence of a safety zone to protect a certain fraction, \(f_S\), of the population, mostly those more vulnerable. In practice, this involves dividing the camp in two areas, a “green” zone (denoted \(g\)) for the protected population and an “orange” zone (\(o\)) for the exposed population, and dividing each demographic-class into two behaviour-classes for each respective zone. These two populations interact via a buffer zone, under controlled conditions where we assumed transmissivity is reduced by 80%, encoded in the parameter \(\xi_i\). Each individual in the green zone can interact with a limited number \((c_v)\) of family members (hereafter “visitors”) from the orange zone per day. In some interventions we considered that individuals visiting the buffer zone will have a health check (e.g. temperature measurement), aimed at excluding symptomatic individuals. When the health check is applied, the probability of transmission by individuals in the \(s\) or \(H\) compartments from one zone to susceptible individuals from a different zone is set to zero (see parameters \(\zeta_s\) and \(\zeta_H\) in Eq. 24 in Main Text). In the following, we derive the values of parameters \(\epsilon_{ij}\) and \(\zeta_{ij}\), modifying the rate at which individuals become exposed (see Eq. 24 in Main Text).

Although setting up a safety zone implies a reduction in the number of contacts between classes of the green zone and the orange zone, the mean number of contacts that each individual has per day, \(c_v\), is conserved. Therefore we need to estimate how contacts will be redistributed from individuals from a different zone to individuals living in the same zone. We model this redistribution of contacts with the parameter \(\epsilon_{ij}\):

\[
\epsilon_{ij} = \frac{\vartheta c_v}{c_i} \quad (i, j \text{ in different zones})
\]

\[
\epsilon_{ij} = 1 - \frac{\vartheta c_v}{c_i} \quad (i, j \text{ in same zone}).
\]

We define \(\vartheta\) as\(^1\):

\[
\vartheta = \begin{cases} 
1 & \text{if } i \in g \\
\frac{f_{o,\text{visit}}}{f_{o,\text{visit}}} & \text{if } i \in o
\end{cases}
\]

\(^1\)If \(c_{\text{visit}}\) is large enough (\(c_{\text{visit}} \approx 15 \text{ contacts per day}\), \(\vartheta\) should saturate, because every member of the orange zone would eventually visit the buffer zone, following the expression:

\[
\vartheta = \begin{cases} 
1 & \text{if } i \in g \\
\frac{f_{o,\text{visit}}}{f_{o,\text{visit}}} (1 - \Theta(f_{o,\text{visit}} - 1) f_{o,\text{visit}} - 1) & \text{if } i \in o
\end{cases}
\]

with the Heaviside function \(\Theta(f_{o,\text{visit}} - 1) = 1\) if \(f_{o,\text{visit}} \geq 1\). We chose values well below this saturation threshold (a maximum of 10 contacts per week, i.e. 1.42 contacts per day).
If we assume that visitors are always different, the quantity \( f_{\text{visit}} = c_{\text{visit}} \frac{N_i}{N_X} \) is the fraction of the orange population that visits the buffer zone.

Next, we estimate how the probability of interaction between a member of class \( i \) and class \( j \) is modified with respect to the null model, depending on the zones from which class \( i \) and class \( j \) are drawn. Suppose classes \( i \) and \( j \) are separated from the rest of the camp’s population and confined within a restricted zone. The probability that an individual of class \( i \) randomly encounters an individual of class \( j \) would now be higher than in a well-mixed population where interaction with individuals belonging to other classes is not restricted. This modification of relative probability of interaction is encoded in the parameter \( \omega_{ij} \) (see Eq. 24 in Main Text). More specifically, the proportion \( \frac{N_i}{N} \) of individuals of class \( i \) in the null model becomes \( \frac{N_i}{N_X} \) with \( N_X \) the total number of individuals in zone \( X \) = \{o, g\}. This yields the following values for \( \omega_{ij} \):

\[
\omega_{ij} = \frac{\left( \frac{N_i}{N_X} \right)}{\left( \frac{N}{N_X} \right)} \quad (i, j \text{ in same zone } X)
\]

\[
\omega_{ij} = \frac{\left( \frac{N_i}{N_Y} \right)}{\left( \frac{N}{N_Y} \right)} \quad (i \in X \text{ and } j \in Y).
\]

<table>
<thead>
<tr>
<th>Scenario</th>
<th>Age 1, orange</th>
<th>Age 1, green</th>
<th>Age 2 no comorbidities, orange</th>
<th>Age 2 no comorbidities, green</th>
<th>Age 2 comorbidities, orange</th>
<th>Age 2 comorbidities, green</th>
<th>Age 3 no comorbidities, green</th>
<th>Age 3 comorbidities, green</th>
</tr>
</thead>
<tbody>
<tr>
<td>Only age 3 in green zone</td>
<td>.407</td>
<td>0</td>
<td>.471</td>
<td>0</td>
<td>.0626</td>
<td>0</td>
<td>.022</td>
<td>.0373</td>
</tr>
<tr>
<td>Age 3 + age 2 with comorbidities</td>
<td>.407</td>
<td>0</td>
<td>.471</td>
<td>0</td>
<td>0</td>
<td>.0626</td>
<td>.022</td>
<td>.0373</td>
</tr>
<tr>
<td>in green zone</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20% green zone capacity</td>
<td>.376</td>
<td>.0312</td>
<td>.424</td>
<td>.0469</td>
<td>0</td>
<td>.0626</td>
<td>.022</td>
<td>.0373</td>
</tr>
<tr>
<td>25% green zone capacity</td>
<td>.356</td>
<td>.0512</td>
<td>.394</td>
<td>.0769</td>
<td>0</td>
<td>.0626</td>
<td>.022</td>
<td>.0373</td>
</tr>
<tr>
<td>30% green zone capacity</td>
<td>.336</td>
<td>.0712</td>
<td>.364</td>
<td>.107</td>
<td>0</td>
<td>.0626</td>
<td>.022</td>
<td>.0373</td>
</tr>
</tbody>
</table>

Table 2: Fraction of population in each zone by safety zone scenario and behaviour-class. Behaviour-classes that are not considered in a given scenario have a proportion equal to zero.

Following this parameterization, we explore different scenarios, summarized in Table 2, for allocating members of each population class to the safety, or “green” zone, and the exposed, or “orange” zone. In one scenario, we only place individuals in age group 3 (>50) in the green zone, while in another we place all vulnerable individuals, age group 3 and age group 2 (13-50) with comorbidities, in the green zone. In 3 additional scenarios, after all vulnerable individuals are allocated to the green zone, we set the green zone’s capacity to a certain percentage of the camp’s population (20%, 25%, 30%), and allocate its remainder to non-vulnerable family members, who by necessity are either children <13 in age group 1 or healthy younger adults in age group 2. In accordance with camp managers’ expectations that many vulnerable individuals will have non-vulnerable spouses, while fewer vulnerable individuals will have young children, in these scenarios we allocate 40% of the remainder of the green zone to children and 60% of the remainder of the green zone to younger adults without comorbidities. We also consider a baseline scenario in which there is no green zone.
2.2 Self-isolation and evacuation

To implement self-isolation it is required a delineation between the isolated and non-isolated population within the clinical symptomatic compartment after identification of symptoms ($I_j$). This does not entail the creation of a new compartment, but rather the estimation of the number of individuals in each class in isolation ($\tilde{I}_j$). We first compute the total number of infected individuals across every class, $N_1 = \sum_i I_i$, at each integration step in the simulation. Next, we consider the isolation capacity of the camp ($\tilde{N}$), and we assume that, when the infectious population exceeds this capacity, the number of isolated individuals for each class is proportional to the number of clinical symptomatic individuals in the class, i.e. $\tilde{I}_j = NI_j/\tilde{N}$. Once this is ascertained, we can encode the different contact rates of these two subpopulations (isolated and not isolated) with other classes in $\lambda_i$. Similar reasoning can be followed regarding the modelization of carers. We do not create a new class since carers are exclusively composed of younger adults with no comorbidities and thus have identical epidemiological parameters to this demographic-class; we only need to modify carers’ rate of exposure. Therefore, we can proceed by encoding this intervention in $\lambda_i$ with the parameters $\epsilon_{ij}$ and $\omega_{ij}$.

Let us start by considering the rate of exposure of younger adults with no comorbidities, hereafter indexed $k$.

The general expression of $\lambda_k$ presented in the Main Text is:

$$\lambda_k = \sum_{j=1}^{n} \tau \xi_{kj} c_k \xi_{kj} \omega_{kj} \frac{N_j}{N} \left( \frac{\beta_P P_j + \beta_A A_j + \zeta_1 \beta_I I_j + \zeta_2 \beta_H H_j}{N_j} \right).$$

(24)

If we have $N_{\text{care}}$ carers, the expression of $\lambda_k$ can be split in two contributions:

$$\lambda_k = \frac{N_{\text{care}} \lambda_{k_{\text{care}}}}{N_k} + \left( \frac{N_k - N_{\text{care}}}{N_k} \right) \lambda_{k_{\text{notcare}}},$$

(25)

where the first term in the r.h.s. of the equation is the contribution from carers, and the second term from the remaining population of the class. We can proceed similarly now splitting $\lambda_{k_{\text{care}}}$ in two, one term to model the interaction with the $I_j$ isolated individuals and another term to account for the interaction with the $I_j - \tilde{I}_j$ not-isolated individuals (if the total number of symptomatic individuals $N_i$ exceeds the capacity of the camp $\tilde{N}$):

$$\lambda_{k_{\text{care}}} = \tau \sum_{j=1}^{n} \tilde{\xi}_{kj} \tilde{c}_k \xi_{kj} \omega_{kj} \frac{N_j}{N} \left( \frac{\tilde{P}(k \rightarrow j)}{N_j} \right) + \xi_{kj} c_k \xi_{kj} \omega_{kj} \frac{N_j}{N} \left( \frac{\beta_P P_j + \beta_A A_j + \zeta_1 \beta_I (I_j - \tilde{I}_j) + \zeta_2 \beta_H H_j}{N_j} \right).$$

(26)

where we indicate with tildes those parameters in the isolated population and we note that, if these parameters are the same than for the not-isolated population, we retrieve back Eq. 24. The split presented in Eq. 26 is valid under the well-mixed assumption. However, this assumption does not hold for the isolated population, which is confined under very particular conditions. More specifically, we should re-estimate the term highlighted as $\tilde{P}(k \rightarrow j)$ which is the probability of interaction of the population of carers with isolated individuals (because, by definition, those fulfilling their roles as carers will interact with isolated individuals), and hence $\tilde{P}(k \rightarrow j) = 1$.

Therefore, for the isolated term, we should just estimate the coefficients $\tilde{\xi}_{kj}$ and $\tilde{c_k}$. To estimate $\tilde{c}_k$ we note that, if the class has $N_{\text{care}}$ carers, each isolated individual requires $c_{\text{care}}$ contacts per day with them, and each class $j$ has $I_j$ isolated individuals, the mean number of contacts that each carer has per day with individuals in isolation is $\bar{c}_k = c_{\text{care}} I_j / N_{\text{care}}$. In our simulations, each isolated individual receives one visit per day, i.e. $c_{\text{care}} = 1$. Finally, since we envisage these interactions occurring in what we call buffer zones, namely open spaces in which carers and isolated individuals maintain a distance and wear masks, reducing the transmissibility by 80%, we consider that $\tilde{\xi}_{ij} = 0.2$.

For the not-isolated term, we maintain the well-mixed population assumption explained in the Main Text, with the incorporation of a Heaviside function, $\zeta_j = \Theta(N_j - \tilde{N})$, which activates interaction with clinical symptomatic individuals when their number $N_j$ exceeds the isolation capacity $\tilde{N}$. We should also readjust the probability of interacting with symptomatic non-isolated individuals in class $j$ to be proportional to their fraction of the class’ population, $(I_j - \tilde{I}_j)/(N_j - \tilde{I}_j)$. This yields the following expression for the subpopulation of carers:
\[ \lambda_{\text{care}}^k = \tau \sum_j \xi_{ij} \beta_i \hat{c}_k + c_k \left( \frac{N_j - \hat{I}_j}{N} \right) \left( \frac{\beta P_j + \beta A_j + \beta \Theta(N_1 - \hat{N})(I_j - \hat{I}_j) + \beta H_j}{N_j - \hat{I}_j} \right). \] (27)

Note that the mean number of contacts per day that carers have with the rest of the population, \( c_k \), is not reduced since we do not make additional assumptions about carers’ behavioural changes outside of their role as carers.

Younger adults without comorbidities who do not serve as carers will not interact with isolated individuals (i.e. \( \tilde{P}(k \to j) = 0 \)), and hence their rate of exposure becomes:

\[ \lambda_{\text{care}}^k = c_k \left( \frac{N_j - \hat{I}_j}{N} \right) \left( \frac{\beta P_j + \beta A_j + \beta \Theta(N_1 - \hat{N})(I_j - \hat{I}_j) + \beta H_j}{N_j - \hat{I}_j} \right). \] (28)

Inserting Eqs. 27 and 28 into Eq. 25 yields:

\[ \lambda_i = \tau \sum_j \xi_{ij} \beta_i c_{\text{care}} \frac{\tilde{I}_j}{N_h} + c_k \left( \frac{\beta P_j + \beta A_j + \beta \Theta(N_1 - \hat{N})(I_j - \hat{I}_j) + \beta H_j}{N} \right). \] (29)

We can express this equation following the parameterization presented in Eq. 24 making \( \tilde{\xi}_{ij} = 0.2 \), \( \tilde{\epsilon}_{ij} = (c_{\text{care}}/c_i)(\tilde{I}_j/N_j) \), \( \tilde{\omega}_{ij} = N/N_j \) and \( \tilde{\zeta}_i = 1 \) for \( j \in \text{isolated individuals in class } j \), while \( \xi = 1, \epsilon_{ij} = 1, \omega_{ij} = 1 \) and \( \zeta_i = \Theta(N_1 - \hat{N}) \) for \( j \in \text{individuals in class } j \) who are not isolated. The rate of exposure of the remaining classes (not younger adults without comorbidities, \( i \neq k \)) corresponds to the second term in the r.h.s of Eq. 29:

\[ \lambda_i = \tau \sum_j c_i \left( \frac{\beta P_j + \beta A_j + \beta \Theta(N_1 - \hat{N})(I_j - \hat{I}_j) + \beta H_j}{N} \right). \] (30)

We should note that Eq. 29 does not depend on the number of carers, \( N_{\text{care}} \), since we assume that the rate of exposure of carers is evenly distributed among all individuals in the class of healthy younger adults. If this assumption were not made, a specific class of carers could be created and the variable \( N_{\text{care}} \) maintained explicit.
3 Supplementary figures

Figure 1: Outcomes when all severe (hospitalized) cases recover ($\sigma = 0$) vs when all severe (hospitalized) cases die ($\sigma = 1$). Probability of an outbreak (top left), fraction of the population dying (top middle), time until peak symptomatic cases (top right), IFR (bottom left), and fraction of the population that recovers (bottom middle). Since we define outbreaks as simulations in which at least one person dies and probability of a case dying is higher when $\sigma = 1$, the probability of observing an outbreak is also necessarily higher when $\sigma = 1$.

Figure 2: Self-distancing. IFR (left), and fraction of the population that recovers (right) as a function of the proportion of contacts reduced per individual per day.
Figure 3: **Self-isolation.** IFR (left), and fraction of the population that recovers (right) as a function of the number of isolation tents available in the camp.

Figure 4: **Evacuation.** Probability of an outbreak (top left), fraction of the population dying (top middle), time until peak symptomatic cases (top right), IFR (bottom left), and fraction of the population that recovers (bottom middle), as a function of whether individuals requiring hospitalization are evacuated to isolation centers.
Figure 5: **Health-checks in the buffer zone.** Probability of an outbreak (top left), fraction of the population dying (top middle), time until peak symptomatic cases (top right), IFR (bottom left), and fraction of the population that recovers (bottom middle), as a function of whether health-checks are implemented in the buffer zone between the safety and exposed zones. Scenarios with 10 or 2 contacts in the buffer zone per person in the safety zone per week are plotted. All figures consider the scenario in which 20% of the camp’s population is allocated to the safety zone. Note that the mean of an outcome for the whole population is not the weighted mean of the exposed and safety zones, since outcomes are computed considering simulations in which at least one death was observed in the population class inhabiting the zone, i.e. the number of simulations considered to compute each mean may be different. This explains for example why there is a reduction in the mean time until symptomatic cases peak when moving from 2 contacts per week without health checks to considering health checks for the whole population, despite there being an increase in the safety zone.
Figure 6: **Effects of the safety zone on outcomes by population class.** Probability of an outbreak (top), and proportion that dies in each population class (bottom) when no interventions are implemented (Mixed), compared to protection of older adults in the safety zone with 2 contacts in the buffer zone per week (Safety zone). The fraction of deaths in the safety zone for the older population is significantly lower.
Figure 7: **Number of contacts in the buffer zone.** IFR (left), and fraction of the population that recovers (right) as a function of the number of contacts that each individual in the safety zone has in the buffer zone per week. All figures consider the scenario in which 20% of the camp’s population is allocated to the safety zone.

Figure 8: **Population moving to the safety zone.** Probability of an outbreak (top left), fraction of the population dying (top middle), time until peak symptomatic cases (top right), IFR (bottom left), and fraction of the population that recovers (bottom middle) as a function of the safety zone allocation scenario (see Table 2). All figures consider the scenario with 2 contacts in the buffer per person in the safety zone per week.
Figure 9: Efficacy of the safety zone for different population sizes. Probability of an outbreak (top left), fraction of the population dying (top middle), time until peak symptomatic cases (top right), IFR (bottom left), and fraction of the population that recovers (bottom middle) as a function of the total population size. The figures consider scenarios with no interventions (null), and with a safety zone comprising 20% of the camp’s population with 2 contacts in the buffer zone per person in the safety zone per week (safety 2).
Figure 10: **Lockdown of the safety zone.** Probability of an outbreak (top left), fraction of the population dying (top middle), time until peak symptomatic cases (top right), IFR (bottom left), and fraction of the population that recovers (bottom middle) as a function of the reduction in the number of contacts permitted in the buffer zone from a baseline of 2 per person in the safety zone per week. All figures consider the scenario in which 20% of the camp’s population is allocated to the safety zone.
Figure 11: Combined interventions. IFR (top), and fraction of the population that recovers (bottom) for different combinations of interventions. Evac = evacuation of severely symptomatic, self = self-distancing, tents = number of available self-isolation tents, safety = safety zone, lock = lockdown of the buffer zone. For combinations of interventions including a safety zone, we distinguish between the population living in the green zone, in the orange zone and the whole population. The increase in the IFR for the green zone is explained by the discretization of the possible values that the IFR can take when the number of cases is very low (see Supplementary Table 3).
<table>
<thead>
<tr>
<th>Intervention</th>
<th>&lt;20 cases</th>
<th>Total</th>
<th>% of total</th>
</tr>
</thead>
<tbody>
<tr>
<td>safety</td>
<td>36</td>
<td>1908</td>
<td>1.9</td>
</tr>
<tr>
<td>safety + evac</td>
<td>48</td>
<td>1894</td>
<td>2.5</td>
</tr>
<tr>
<td>safety + lock 50%</td>
<td>40</td>
<td>1454</td>
<td>2.8</td>
</tr>
<tr>
<td>safety + self 20%</td>
<td>56</td>
<td>1582</td>
<td>3.5</td>
</tr>
<tr>
<td>safety + 50 tents</td>
<td>37</td>
<td>1330</td>
<td>2.8</td>
</tr>
<tr>
<td>safety + self 50%</td>
<td>187</td>
<td>719</td>
<td>26</td>
</tr>
<tr>
<td>safety + 50 tents + lock 50%</td>
<td>58</td>
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Table 3: **Efficacy of the safety zone in combination with other interventions.** <20 cases = number of outbreaks in the green zone with fewer than 20 cases recorded. Total = total number of simulations where an outbreak in the green zone occurs (at least one death). % of total = percent of outbreaks where fewer than 20 cases are recorded. N = 2500 simulations for each combination of interventions. For the most effective combinations, the majority of simulations where an outbreak occurs in the green zone see fewer than 20 cases. In these simulations, the discretization of the possible values that the IFR can take explains its apparently anomalous increase in Fig. 11.

Figure 12: **Critical number of exposed individuals.** (Left) Reducing the number of contacts reduces the maximum number of individuals simultaneously exposed while increasing the time until symptomatic cases peak. When the reduction goes beyond 60%, there are abrupt drops in the time until cases peak and the fraction of the population dying, suggesting there is a critical number of individuals who must be exposed, under which outbreaks die out before spreading widely throughout the population. Above this threshold, the virus becomes established in the population over a longer period of time, increasing mortality. (Right) Fraction of the simulations which do not achieve the critical number of exposed individuals (that we set to 2% of the population). The abrupt transition between 50 and 70% reductions in contacts is apparent.
## 4 Appendix: List of Experiments

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Table 4: List of simulations performed. Npop = Population size. Evac. = Is people requiring hospitalization evacuated? N. tents = Number self-isolation tents per camp. Contacts = Number of contacts per day between populations shielded. Tcheck = Are temperature checks performed? Lock = Is lockdown applied after first symptomatic case is identified? self = Fraction of contacts remaining after self-distancing is implemented. H-fate = Final compartment for hospitalized people. MF = Mean field. Shield = Population shielded. age3 = elderly population. age2 = adults with comorbidities and spouses. (20-30%) = kids from adults shielded up to x% of total population.
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Table 5: **List of simulations performed (II).** Npop = Population size. Evac. = Is people requiring hospitalization evacuated? N. tents = Number self-isolation tents per camp. Contacts = Number of contacts per day between populations shielded. Tcheck = Are temperature checks performed? Lock = Is lockdown applied after first symptomatic case is identified? self = Fraction of contacts remaining after self-distancing is implemented. H-fate = Final compartment for hospitalized people. MF = Mean field. Shield = Population shielded. age3 = elderly population. age2 = adults with comorbidities and spouses. (20-30%) = kids from adults shielded up to x% of total population.
References


