Supplementary Methods and Results
Empowering the crowd: Feasible strategies to minimize the spread of COVID-19 in informal settlements

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1 Parameterization of the model

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

To estimate the latent period ($1/\delta_{\mathrm{E}}$), we calculated the difference between randomly generated incubation ($1/\delta_{\mathrm{E}} + 1/\delta_{\mathrm{P}}$) and presymptomatic ($1/\delta_{\mathrm{P}}$) 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 follows 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 .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 ?? 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 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 $\tau\beta_{\mathrm{P}}$, $\tau\beta_{\mathrm{A}}$, $\tau\beta_{\mathrm{I}}$ or $\tau\beta_{\mathrm{H}}$ depending upon whether the infected individual is in the presymptomatic (P_i), symptomatic (I_i), asymptomatic (A_i), or hospitalized compartment (H_i), respectively. We estimated these parameters in two steps. In the following section, we estimate the β_{X} parameters ($\mathrm{X} \in \{\mathrm{P,A,I,H}\}$) which represent the relative transmissibility of each stage with respect to the maximum transmissibility τ. After this calculation, we present our estimate for the maximum transmissibility parameter τ.

Relative transmissibilities β (Table ?? in Main Text)

We start by considering the transmissibility of the presymptomatic stage for those individuals who become symptomaticas a reference ($\beta_{\mathrm{P}\rightarrow\mathrm{I}} = 1$), 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 $\beta_{\mathrm{X}}/\gamma_{\mathrm{X}}$, with $1/\gamma_{\mathrm{X}}$ the duration of stage X. Following He et al, we estimate the proportion of infectivity in individuals who go on to develop symptoms that occurs at the presymptomatic stage ($\mathrm{X} \in \{\mathrm{P}\}$) as the area under the infectivity curve prior to symptom onset, AUC_{P}, and the proportion of infectivity that occurs at symptomatic stages ($\mathrm{X} \in \{\mathrm{I,H}\}$) as the area under the infectivity curve after symptom onset, $1 - AUC_{\mathrm{P}}$ [8]:

$$\frac{AUC_{\mathrm{P}}}{(1 - AUC_{\mathrm{P}})} \approx \frac{\frac{\beta_{\mathrm{P}\rightarrow\mathrm{I}}}{\delta_{\mathrm{P}}}}{\frac{\beta_{\mathrm{I}}}{\gamma_{\mathrm{I}}} + \frac{\beta_{\mathrm{H}}}{\gamma_{\mathrm{H}}}} \tag{1}$$

We then considered the quantity ρ_{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 ρ_{AI} [10]:

$$\beta_{\mathrm{A}} = \beta_{\mathrm{I}}\rho_{\mathrm{AI}} \tag{2}$$

$$\beta_{\mathrm{H}} = \beta_{\mathrm{I}}\rho_{\mathrm{HI}} \tag{3}$$

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

$$\beta_{\mathrm{I}} = \frac{\beta_{\mathrm{P}\rightarrow\mathrm{I}}\gamma_{\mathrm{I}}\gamma_{\mathrm{H}}(1 - AUC_{\mathrm{P}})}{AUC_{\mathrm{P}}\delta_{\mathrm{P}}(\gamma_{\mathrm{H}} + \rho_{\mathrm{HI}}\gamma_{\mathrm{I}})} \tag{4}$$

$$\beta_{\mathrm{A}} = \frac{\rho_{\mathrm{AI}}\beta_{\mathrm{P}\to\mathrm{I}}\gamma_{\mathrm{I}}\gamma_{\mathrm{H}}(1 - AUC_{\mathrm{P}})}{AUC_{\mathrm{P}}\delta_{\mathrm{P}}(\gamma_{\mathrm{H}} + \rho_{\mathrm{HI}}\gamma_{\mathrm{I}})} \tag{5}$$

$$\beta_{\mathrm{H}} = \frac{\rho_{\mathrm{HI}}\beta_{\mathrm{P}\to\mathrm{I}}\gamma_{\mathrm{I}}\gamma_{\mathrm{H}}(1 - AUC_{\mathrm{P}})}{AUC_{\mathrm{P}}\delta_{\mathrm{P}}(\gamma_{\mathrm{H}} + \rho_{\mathrm{HI}}\gamma_{\mathrm{I}})}. \tag{6}$$

The values of AUC_{P}, ρ_{AI} and ρ_{HI} are presented in Table 1, and the values of β_{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 ($\beta_{\mathrm{P}\to\mathrm{I}} = 1$), we need to go back and derive the transmissibility of all presymptomatic individuals (β_{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 (ρ_{AI}). With the proportion of presymptomatic cases that will become developed symptoms f (encoded in in Eqs. 24–32 in Main Text), we compute the mean transmissibility of all presymptomatic individuals as:

$$\beta_{\mathrm{P}} = f\beta_{\mathrm{P}\to\mathrm{I}} + (1 - f)\rho_{\mathrm{AI}}\beta_{\mathrm{P}\to\mathrm{I}}. \tag{7}$$

Parameter	Description	Value	Distribution	Reference
AUC_{P}	Presymptomatic area under infectivity curve	0.44 (95% CI: .30-.57)	Gaussian	[8]
ρ_{AI}	Ratio of asymptomatic to symptomatic infectiousness	0.58 (95% CI: .34-.99)	Lognormal	[10]
ρ_{HI}	Ratio of hospitalized to symptomatic infectiousness	0.48	-	[9]

Table 1: **Relative transmissibility parameters.**

Maximum transmissibility parameter τ

In the following, to simplify the notation we define $\kappa_i = (l_i\gamma_{\mathrm{I}} + h_i\eta + g_i\alpha)$. To estimate the probability of infection if there is a contact between a susceptible and an infected individual (parameter τ) we proceed as follows [11, 12, 13]. We start by considering the subsystem containing the infected population:

$$\dot{E}_i = \lambda_i S_i - \delta_{\mathrm{E}}E_i \tag{8}$$

$$\dot{P}_i = \delta_{\mathrm{E}}E_i - \delta_{\mathrm{P}}P_i \tag{9}$$

$$\dot{A}_i = (1 - f)\delta_{\mathrm{P}}P_i - \gamma_{\mathrm{A}}A_i \tag{10}$$

$$\dot{I}_i = f\delta_{\mathrm{P}}P_i - \kappa_i I_i \tag{11}$$

$$\dot{H}_i = h_i\eta I_i - \gamma_{\mathrm{H}}H_i. \tag{12}$$

For the sake of simplifying the notation, let us consider the following ordering of the variables in the vector $x = (E_1, ..., E_M, P_1, ..., P_M, A_1, ..., A_M, I_1, ..., I_M, H_1, ..., H_M)$, 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 τ, which is initially unknown, but does not change when interventions are introduced. Considering the contacts matrix for the null model (Eq. **??** in Main Text), the rate of exposurebecomes

$$\lambda_i = \frac{\tau}{N}\sum_{j=1}^{M} c_i\left(\beta_{\mathrm{P}}P_j + \beta_{\mathrm{A}}A_j + \beta_{\mathrm{I}}I_j + \beta_{\mathrm{H}}H_j\right). \tag{13}$$

In the following, we use bold symbols for vectors and matrices, and the symbols $\odot$ and $\oslash$ for the element-wise multiplication and division, respectively. Following this notation, the linearized system can be written in the form $\dot{\boldsymbol{x}} = (\boldsymbol{T} + \boldsymbol{\Sigma})\boldsymbol{x}$, where:

3

$$\boldsymbol{T} = \tau \begin{bmatrix} \mathbf{0} & \boldsymbol{\Theta}_P & \boldsymbol{\Theta}_A & \boldsymbol{\Theta}_I & \boldsymbol{\Theta}_H \\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} \end{bmatrix} \tag{14}$$

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

$$\boldsymbol{\Sigma} = \begin{bmatrix} -\delta_E \boldsymbol{I} & \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ \delta_E \boldsymbol{I} & -\delta_P \boldsymbol{I} & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & (1-f)\delta_P \boldsymbol{I} & -\gamma_A \boldsymbol{I} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & f\delta_P \boldsymbol{I} & \mathbf{0} & -\mathrm{diag}(\boldsymbol{\kappa})\boldsymbol{I} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \eta\,\mathrm{diag}(\boldsymbol{h})\boldsymbol{I} & -\gamma_H \boldsymbol{I} \end{bmatrix} \tag{15}$$

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

$$\boldsymbol{\Sigma}^{-1} = \begin{bmatrix} -\frac{1}{\delta_E}\boldsymbol{I} & \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ -\frac{1}{\delta_P}\boldsymbol{I} & -\frac{1}{\delta_P}\boldsymbol{I} & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ -\frac{(1-f)}{\gamma_A}\boldsymbol{I} & -\frac{(1-f)}{\gamma_A}\boldsymbol{I} & -\frac{1}{\gamma_A}\boldsymbol{I} & \mathbf{0} & \mathbf{0} \\ -f\,\mathrm{diag}(\boldsymbol{\kappa}^{-1})\boldsymbol{I} & -f\,\mathrm{diag}(\boldsymbol{\kappa}^{-1})\boldsymbol{I} & \mathbf{0} & -\mathrm{diag}(\boldsymbol{\kappa}^{-1})\boldsymbol{I} & \mathbf{0} \\ -\frac{f\eta}{\gamma_H}\mathrm{diag}(\boldsymbol{h}\oslash\boldsymbol{\kappa})\boldsymbol{I} & -\frac{f\eta}{\gamma_H}\mathrm{diag}(\boldsymbol{h}\oslash\boldsymbol{\kappa})\boldsymbol{I} & \mathbf{0} & -\frac{\eta}{\gamma_H}\mathrm{diag}(\boldsymbol{h}\oslash\boldsymbol{\kappa})\boldsymbol{I} & -\frac{1}{\gamma_H}\boldsymbol{I} \end{bmatrix} \tag{16}$$

The NGM with large domain can now be found by $\boldsymbol{K_L} = -\boldsymbol{T}\boldsymbol{\Sigma}^{-1}$. However, since we know that each individual who gets infected becomes exposed (E compartment), we focus on the NGM with small domain, $\mathbf{K}_S$, 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 Σ^{-1}. We then find:

$$\boldsymbol{K_S} = \tau\left[\frac{1}{\delta_P}\boldsymbol{\Theta}_P + \frac{(1-f)}{\gamma_A}\boldsymbol{\Theta}_A + f\,\mathrm{diag}\left(\boldsymbol{h}^{-1}\right)\boldsymbol{\Theta}_I + \frac{f\eta}{\gamma_H}\mathrm{diag}(\boldsymbol{h}\oslash\boldsymbol{\kappa})\boldsymbol{\Theta}_H\right]. \tag{17}$$

The reproduction number is related to the dominant eigenvalue of $\boldsymbol{K_S}$, i.e. $R_0 = |\lambda_1|$, and τ is estimated from the dominant eigenvalue of $\tilde{K}_S = K_S/\tau$. Considering the null model parameters $(\tilde{\lambda}_1^0)$, we have the expression:

$$\tau = \frac{R_0}{|\tilde{\lambda}_1^0|}. \tag{18}$$

1.4 Epidemiological severity proportions (Table ?? in Main Text)

In the Main Text, we presented the proportions in which clinical symptomatic individuals resolve into critical (q_i^D), severe (q_i^H) and recovered (q_i^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 (η for H, α for D and γ_I for R) we introduced three parameters, h_i, g_i and l_i, to distribute individuals according to the desired proportions following the equations:

$$q_i^H = h_i \eta \kappa_i^{-1} \tag{19}$$

$$q_i^D = g_i \alpha \kappa_i^{-1} \tag{20}$$

$$q_i^R = 1 - q_i^H - q_i^D = l_i \gamma_I \kappa_i^{-1}, \tag{21}$$

where $\kappa_i = h_i \eta + g_i \alpha + l_i \gamma_I$. 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:

$$h_i = \frac{\alpha q_i^H}{\eta q_i^D} g_i, \tag{22}$$

$$g_i = \gamma_I \left(\frac{\alpha}{q_i^D} + \frac{\gamma_I \alpha q_i^H}{\eta q_i^D} + \gamma_I - \frac{\alpha q_i^H}{q_i^D} - \alpha \right)^{-1}. \tag{23}$$

2 Parameterization of the interventions (Table ?? 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 $\xi_{ij} = 0.2$. Each individual in the green zone can interact with a limited number (c_{visit}) 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 I or H compartments from one zone to susceptible individuals from a different zone is set to zero (see parameters ζ_I and ζ_H in Eq. ?? in Main Text). In the following, we derive the values of parameters ϵ_{ij} and ω_{ij}, modifying the rate at which individuals become exposed (see Eq. ?? 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_i, 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 ϵ_{ij}:

$$\begin{aligned}
\epsilon_{ij} &= \vartheta c_{\text{visit}}/c_i \quad (i,j \text{ in different zones}) \\
\epsilon_{ij} &= 1 - \vartheta c_{\text{visit}}/c_i \quad (i,j \text{ in same zone}).
\end{aligned}$$

We define ϑ as[1]:

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

If we assume that visitors are always different, the quantity $f_{\text{o,visit}} = c_{\text{visit}} \frac{N_g}{N_o}$ 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

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

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

with the Heaviside function $\Theta(f_{\text{o,visit}} - 1) = 1$ if $f_{\text{o,visit}} \geq 1$. We chose values well below this saturation threshold (a maximum of 10 contacts per week, i.e. 1.42 contacts per day).

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 ω_{ij} (see Eq. **??** in Main Text). More specifically, the proportion N_i/N of individuals of class i in the null model becomes N_i/N_X with N_X the total number of individuals in zone $X = \{\mathrm{o}, \mathrm{g}\}$. This yields the following values for ω_{ij}:

$$\omega_{ij} = \left(\tfrac{N_i}{N_\mathrm{X}}\right)/\left(\tfrac{N}{N_i}\right) = \frac{N}{N_\mathrm{X}} \quad (i, j \text{ in same zone } X)$$

$$\omega_{ij} = \left(\tfrac{N_i}{N_\mathrm{Y}}\right)/\left(\tfrac{N}{N_i}\right) = \frac{N}{N_\mathrm{Y}} \quad (i \in X \text{ and } j \in Y).$$

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

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 we start by expanding our model (Eqs 24–32 in Main Text) to consider a new compartment for each demographic-class. To model the fact that symptomatic individuals require some time to recognize their symptoms and to self-isolate, we split the symptomatic compartment in two: symptomatic prior to identification, O_i, and symptomatic following identification, I_i. Following these considerations, the structure of the model is represented in Fig. 1, which follows the equations:

$$\dot{S}_i = -\lambda_i S_i \tag{24}$$

$$\dot{E}_i = \lambda_i S_i - \delta_{\mathrm{E}} E_i \tag{25}$$

$$\dot{P}_i = \delta_{\mathrm{E}} E_i - \delta_{\mathrm{P}} P_i \tag{26}$$

$$\dot{A}_i = (1-f)\delta_{\mathrm{P}} P_i - \gamma_{\mathrm{A}} A_i \tag{27}$$

$$\dot{O}_i = f\delta_{\mathrm{P}} P_i - (l_i\gamma_I + h_i\delta_{\mathrm{O}} + g_i\alpha)O_i \tag{28}$$

$$\dot{I}_i = \delta_{\mathrm{O}} O_i - (l_i\gamma_I + h_i\eta' + g_i\alpha)I_i \tag{29}$$

$$\dot{H}_i = h_i(\eta' I_i + \delta_{\mathrm{O}} O_i) - \gamma_{\mathrm{H}} H_i \tag{30}$$

$$\dot{R}_i = \gamma_{\mathrm{A}} A_i + l_i\gamma_{\mathrm{I}}(I_i + O_i) + (1-\sigma)\gamma_{\mathrm{H}} H_i \tag{31}$$

$$\dot{D}_i = g_i\alpha(I_i + O_i) + \sigma\gamma_{\mathrm{H}} H_i \tag{32}$$

where the duration of O_i follows a Gaussian distribution with means $1/\delta_{\mathrm{O}} \in \{12, 24, 48\}$ hours. The duration for which an individual spends in the clinical symptomatic compartment, $1/\eta'$, is then calculated as the difference between the symptomatic period if there is no isolation, $1/\eta$, and the duration spent in the symptom onset compartment ($1/\delta_{\mathrm{O}}$). The remaining parameters determining the rate of change of individuals at O_i, namely the rate of progression towards recovery or death (γ_i or α, respectively), as well as the relative transmissibility, β_{I}, remain the same as those of the symtomatic compartment, I_i.
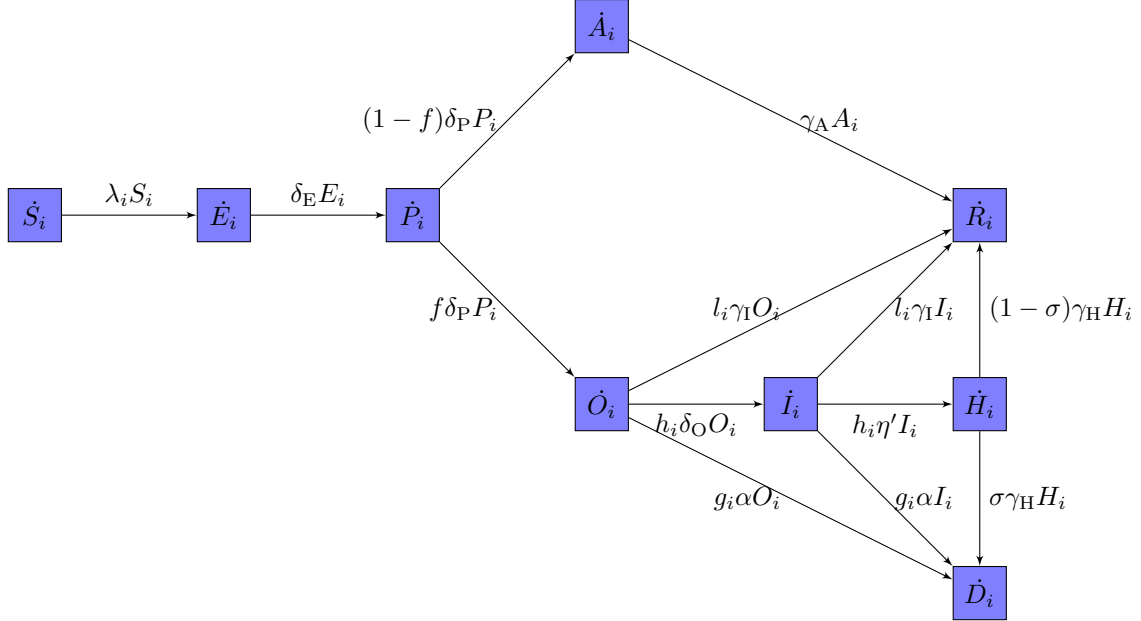

Figure 1: **Diagram of the model including the self-isolation intervention.** The model considers an additional symptom onset compartment (O).

The second modification required is a delineation between the isolated and non-isolated population within the clinical symptomatic compartment after identification of symptoms (I_i). This does not entail the creation of a new compartment, but rather the indentification of the number of individuals in each class in isolation ($\tilde{I}_i$). We first compute the total number of infected individuals across every class, $N_{\mathrm{I}} = \sum_i I_i$, 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. $\tilde{I}_i = \tilde{N}I_i/N_{\mathrm{I}}$. Once this is ascertained, we can encode the different contact rates of these two subpopulations (isolated and not isolated) with other classes in λ_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 λ_i with the parameters ϵ_{ij} and ω_{ij} (see Eq ?? in Main Text).

Let us start by considering the rate of exposure of younger adults with no comorbidities, hereafter indexed k. If this class has N_{care} carers with c_{care} contacts per day with isolated individuals and each class j has $\tilde{I}_j$ isolated individuals, the mean number of contacts that each carer has per day with individuals in isolation is $\tilde{c}_k = c_{\text{care}} \sum_j \tilde{I}_j / N_{\text{care}}$. In our simulations, we let each carer interact with isolated individual per day, i.e. $c_{\text{care}} = 1$. We envisage these interactions occuring 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% ($\xi = 0.2$). Given these assumptions, a general expression for the rate of exposure of younger adults with no comorbidities, λ_k, is given by

$$\lambda_k = \tau \sum_j \underbrace{\xi_{ij} \beta_{\text{I}} \tilde{c}_k \tilde{P}(k \to j)}_{\text{isolated}} + c_k \underbrace{\left(\frac{N_j - \tilde{I}_j}{N} \right) \left(\frac{\beta_{\text{P}} P_j + \beta_{\text{A}} A_j + \beta_{\text{I}} O_j + \beta_{\text{I}} \Theta(N_{\text{I}} - \tilde{N})(I_j - \tilde{I}_j) + \beta_{\text{H}} H_j}{N_j - \tilde{I}_j} \right)}_{\text{not isolated}}, \quad (33)$$

where $\tilde{P}(k \to j)$ is the probability that an individual of class k encounters an isolated individual of class j. For the not-isolated term, we maintain the well-mixed population assumption explained in the Main Text, with the incorporation of a Heaviside function, $\Theta(N_{\text{I}} - \tilde{N})$, which activates interaction with clinical symptomatic individuals when their number N_{I} exceeds the isolation capacity $\tilde{N}$. We set the probability of interacting with symptomatic non-isolated individuals in class j proportional to their fraction of the class' population, $(I_j - \tilde{I}_j)/(N_j - \tilde{I}_j)$. For the isolated term however, the well-mixed assumption is no longer valid, because by definition, those fulfilling their roles as carers will interact with isolated individuals, hence $\tilde{P}(k \to j) = 1$. This yields the following expression for the subpopulation of carers

$$\lambda_k^{\text{care}} = \tau \sum_j \underbrace{\xi_{ij} \beta_{\text{I}} \tilde{c}_k}_{\text{isolated}} + c_k \underbrace{\left(\frac{N_j - \tilde{I}_j}{N} \right) \left(\frac{\beta_{\text{P}} P_j + \beta_{\text{A}} A_j + \beta_{\text{I}} O_j + \beta_{\text{I}} \Theta(N_{\text{I}} - \tilde{N})(I_j - \tilde{I}_j) + \beta_{\text{H}} H_j}{N_j - \tilde{I}_j} \right)}_{\text{not isolated}}, \quad (34)$$

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:

$$\lambda_k^{\overline{\text{care}}} = c_k \underbrace{\left(\frac{N_j - \tilde{I}_j}{N} \right) \left(\frac{\beta_{\text{P}} P_j + \beta_{\text{A}} A_j + \beta_{\text{I}} O_j + \beta_{\text{I}} \Theta(N_{\text{I}} - \tilde{N})(I_j - \tilde{I}_j) + \beta_{\text{H}} H_j}{N_j - \tilde{I}_j} \right)}_{\text{not isolated}}. \quad (35)$$

The total rate of exposure of younger adults without comorbidities, then becomes:

$$\lambda_k = \frac{N_{\text{care}}}{N_k} \lambda_k^{\text{care}} + \left(\frac{N_k - N_{\text{care}}}{N_k} \right) \lambda_k^{\overline{\text{care}}}, \quad (36)$$

which can be made explicit and simplified to yield:

$$\lambda_k = \tau \sum_j \underbrace{\xi_{ij} \beta_{\text{I}} c_{\text{care}} \frac{\tilde{I}_j}{N_k}}_{\text{isolated}} + c_k \underbrace{\left(\frac{\beta_{\text{P}} P_j + \beta_{\text{A}} A_j + \beta_{\text{I}} O_j + \beta_{\text{I}} \Theta(N_{\text{I}} - \tilde{N})(I_j - \tilde{I}_j) + \beta_{\text{H}} H_j}{N} \right)}_{\text{not isolated}}. \quad (37)$$

We can express this equation following the parameterization presented in Eq. ?? in the Main Text where $\epsilon_{ij} = (c_{\text{care}}/c_i)(\tilde{I}_j/N_i)$ and $\omega_{ij} = N/N_j$ for $j \in$ isolated individuals in class j, while $\epsilon_{ij} = 1$ and $\omega_{ij} = 1$ for $j \in$ 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. 37:

$$\lambda_i = \tau \sum_j c_i \underbrace{\frac{\beta_{\text{P}} P_j + \beta_{\text{A}} A_j + \beta_{\text{I}} O_j + \beta_{\text{I}} \Theta(N_{\text{I}} - \tilde{N})(I_j - \tilde{I}_j) + \beta_{\text{H}} H_j}{N}}_{\text{not isolated}}. \quad (38)$$

We should note that Eq. 37 does not depend on the number of carers, N_{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_{care} maintained explicit as in Eq. 34.

3 Supplementary figures

Figure 2: **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 $\sigma = 1$.

figures/FigS3.pdf

Figure 3: **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.

figures/FigS4.pdf

Figure 4: **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.

figures/FigS5.pdf

Figure 5: **Time to self-isolation.** 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 time that individuals require to recognize their symptoms and self-isolate.

figures/FigS6.pdf

Figure 6: **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 7: **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.

Figure 8: **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 (Kruskal-Wallis test, p-val$< 10^{-15}$).

figures/FigS9.pdf

Figure 9: **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.

figures/FigS10.pdf

Figure 10: **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.

figures/FigS11.pdf

Figure 11: **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 12: **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.

figures/FigS13.pdf

Figure 13: **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).

Intervention	<20 cases	Total	% of total
safety	16	270	5.9
safety + evac	20	249	8
safety + lock 50%	5	171	2.9
safety + self 20%	19	188	10
safety + 50 tents	11	240	4.6
safety + self 50%	14	64	22
safety + 50 tents + lock 50%	14	154	9.1
safety + 50 tents + evac	33	239	14
safety + 50 tents + self 20%	31	144	22
safety + 50 tents + self 50%	25	38	66
safety + 50 tents + evac + lock 50% + self 20%	53	110	48
safety + 50 tents + evac + lock 50% + self 50%	18	20	90
safety + 50 tents + evac + lock 90% + self 50%	6	8	75

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 = 500 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. 13.

4 Appendix: List of Experiments

Simul.	Experim.	Structure	Npop	Evac.	N.Tents	Onset	Contacts	Tcheck	lock	self	H-fate
1	A. Shielding and limits	Null Mixed	2000	No	0	0	MF	No	No	No	D
2											R
3		Shield (20%)					2/7				D
4								Yes			R
5							10/7				D
6											R
7	B. Role individual tents	Null Mixed	2000	No	10	24	MF	No	No	No	D
8					20						
9					50						
10					100						
11					250						
12					500						
13					∞						
14		Shield (20%)			10		2/7	Yes			
15					20						
16					50						
17					100						
18					250						
19					500						
20					∞						
21	C. Time to isolation	Null Mixed	2000	No	50	12	MF	No	No	No	D
22						24					
23						48					
24		Shield (20%)				12	2/7	Yes			
25						24					
26						48					
27	D. Population shielded	Shield (age3)	2000	No	0	0	2/7	Yes	No	No	D
28		Shield (age2)									
29		Shield (20%)									
30		Shield (25%)									
31		Shield (30%)									
32	E. Population size	Null Mixed	500	No		0	MF	No	No	No	D
33			1000								
34		Shield (20%)	500				2/7	Yes			
35			1000								
36	F. Safety zone testing	Shield (20%)	2000	No	0	0	2/7	No	No	No	D
37								Yes			
38							10/7	No			
39								Yes			

Table 4: **List of simulations performed.** Npop = Population size. Evac. = Is people requiring hospitalization evacuated? N. tents = Number self-isolation tents per camp. Onset = Mean time that an individual takes to self-isolate from onset of symptoms. 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.

Simul.	Experim.	Structure	Npop	Evac.	N. tents	Onset	Contacts	Tcheck	lock	self	H-fate
40	G. Lockdown	Shield (20%)	2000	No	0	0	2/7	Yes	0.5	No	D
41									0.9		
42									0.99		
43	H. Self-distancing	Null Mixed	2000	No	0	0	MF	No	No	0.1	D
44										0.2	
45										0.3	
46										0.4	
47										0.5	
48		Shield (20%)					2/7	Yes		0.1	
49										0.2	
50										0.3	
52										0.4	
53										0.5	
54	J. Evacuation	Null Mixed	2000	Yes	0	0	MF	No	No	No	D
55		Shield (20%)					2/7	Yes			
56	K. Combined interventions	Null Mixed	2000	No	50	24	MF	No	No	0.2	D
57										0.5	
58		Shield (20%)					2/7	Yes	No	0.2	
59									0.5	No	
61									0.5	0.2	
62									0.9	0.2	
63									0.5	0.5	
64									0.9	0.5	
65		Null Mixed	2000	Yes	50	24	MF	No	No	0.2	D
66										0.5	
67		Shield (20%)					2/7	Yes	No	0.2	
68									0.5	No	
69									0.5	0.2	
70									0.9	0.2	
71									0.5	0.5	
72									0.9	0.5	
73			500						No	0.2	
74									0.5	No	
75									0.5	0.2	

Table 5: **List of simulations performed (II).** Npop = Population size. Evac. = Is people requiring hospitalization evacuated? N. tents = Number self-isolation tents per camp. Onset = Mean time that an individual takes to self-isolate from onset of symptoms. 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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