

Supplementary Methods and Results

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

Authors names TBA

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Add your affiliation here.

Model

We consider a stochastic model governed by the following set of differential equations:

$$\dot{S}_i = -\lambda_i S_i \tag{1}$$

$$\dot{E}_i = \lambda_i S_i - \delta_E E_i \tag{2}$$

$$\dot{P}_i = \delta_E E_i - \delta_P P_i \tag{3}$$

$$\dot{A}_i = (1 - f)\delta_P P_i - \gamma_A A_i \tag{4}$$

$$\dot{I}_i = f\delta_P P_i - ((1 - g_i - h_i)\gamma_I + h_i\eta + g_i\alpha)I_i \tag{5}$$

$$\dot{H}_i = h_i\eta I_i - \gamma_H H_i \tag{6}$$

$$(R/D)_i = \gamma_H H_i \tag{7}$$

$$\dot{R}_i = \gamma_A A_i + (1 - g_i - h_i)\gamma_I I_i \tag{8}$$

$$\dot{D}_i = g_i\alpha I_i \tag{9}$$

where

$$\lambda_i = \sum_{j=1}^n \beta_{ij} \frac{P_j + A_j + I_j + H_j}{N_j} \tag{10}$$

with $\beta_{ij} = \tau C_{ij}$, τ is the probability of infection if there is a contact between a susceptible and an infected person, and C_{ij} is the average number of contacts of an individual of class i with an individual of class j per day. The model is illustrated in Fig. 1, and the rest of the parameters are described in Table 1.

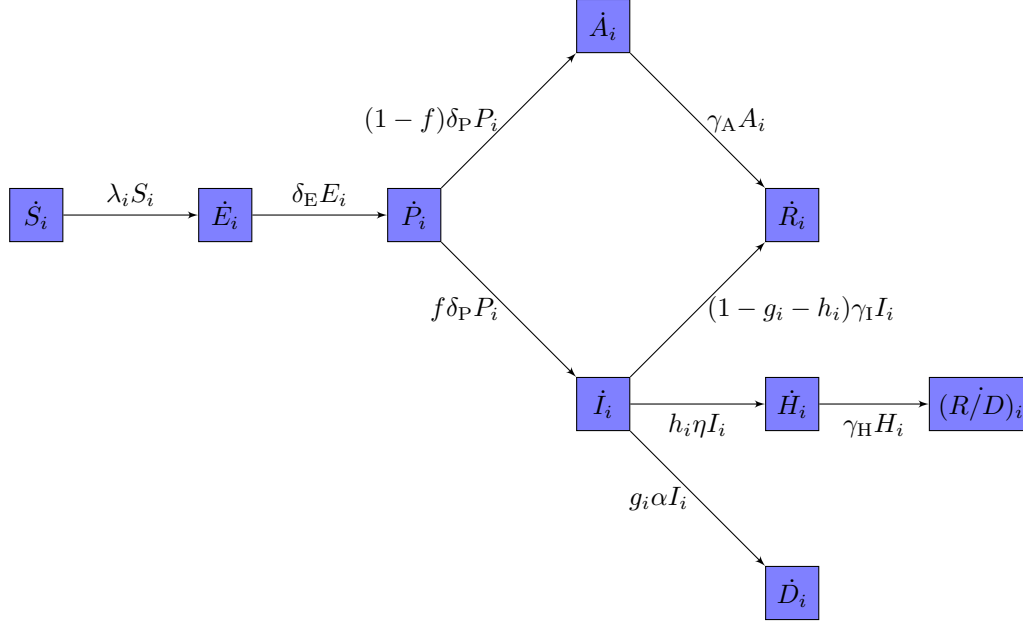


Figure 1: **Diagram of the model.** The model considers the following compartments: susceptible (S), exposed (E), infectious-presymptomatic (P), infectious-asymptomatic (A), infectious-symptomatic (I), infectious-requiring hospitalization (H), recovered (R) and dead (D). Our model considers 3 potential outcomes for symptomatic cases (I): mild cases will recover (R) after the typical infectious period, severe cases will have an extended infectious period during which they require hospitalization (H), and critical cases requiring ICU care will die (D) after the symptomatic period. Since the fate of individuals in the H compartment is uncertain if healthcare is not available, we run simulations considering two possibilities: either all recover, or all die, represented by the R/D compartment.

Table 1: **Model parameters.**

Parameter	Description	Value	Distribution	Reference
$1/\delta_E + 1/\delta_P$	Incubation period (days)	5.2 (95% CI: 4.1-7.0)	Lognormal	[1]
$1/\delta_P$	Presymptomatic infectious period (days)	2.3 (95% CI: 0.8-3.8)	Gaussian	[2]
$1/\delta_E$	Latent period (days)	$(1/\delta_E + 1/\delta_P) - 1/\delta_P$ (Minimum = .5 days)		[Calculated]
$1/\gamma_I$	Symptomatic infectious period (days)	7		[2, 3]
$1/\gamma_A$	Asymptomatic infectious period (days)	7		[2, 3]
$1/\eta$	Time from symptom onset to requiring hospitalization (days)	7 (IQR: 4-8)	Gamma	[4]
$1/\alpha$	Time from symptom onset to death (critical cases, days)	10 (IQR: 6-12)	Gamma	[4]
$1/\gamma_H$	Time from requiring hospitalization to recovery (days)	10 (IQR: 7-14)	Gamma	[4]
f	Probability an infectious individual is symptomatic	0.84 (95% CI: 0.8-0.88)	Binomial	[5]
h_i	Fraction of symptomatic cases severe	Age and comorbidity-dependent (Table 2)		[6, 7]
g_i	Fraction of symptomatic cases critical	Age and comorbidity-dependent (Table 2)		[6, 7]
	Reduction in probability of infection from contact in buffer zone	80%		[Assumed]

To estimate latent period ($1/\delta_E$), we calculated the difference between randomly generated incubation ($1/\delta_E + 1/\delta_P$) and presymptomatic ($1/\delta_P$) periods. The presymptomatic period ($1/\delta_P$) was estimated following the values reported in He et al, with a mean of 2.3 days (95% CI: 0.8-3.0). [2] This follows a Gompertz distribution and is left-skewed, suggesting that the presymptomatic period would not be much longer than 3 days. Since the presymptomatic period can indeed be longer than 3 days, we estimated that the true presymptomatic period follows a Gaussian distribution about the mean (95% CI: 0.8-3.8). With this distribution, there is a non-negligible probability of having a negative latent period. For these cases, we assumed a minimum latent period of .5 days. 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] We assume that in the buffer zone between shielded and non-shielded populations, non-compliance with distancing and mask wearing guidance will be such that the probability of transmission from contacts will be reduced by 80% compared to the baseline.

Population structure

Table 2: **Population structured parameters.**

Population class	Age 1 (0-12)	Age 2 (13-50), no comorbidities	Age 2 (13-50), comorbidities	Age 3 (>50), no comorbidities	Age 3 (>50), comorbidities	Reference
Fraction in class	.407	.471	.0626	.022	.0373	[8, 9, 10]
h_i	.064	.067	.199	.183	.445	[6, 7]
g_i	.0065	.02	.094	.063	.222	[6, 7]
\bar{c}_i ,	25	15	15	10	10	[From camp managers]

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+. [10] 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. [9, 8] 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.

We estimated 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. [6] We estimated the class-specific fractions of symptomatic cases in adults that would become severe and critical using the age and comorbidity-specific fractions of symptomatic cases with known outcomes that required hospitalization, without and with ICU admission, respectively in the US. [7] 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+.

Table 3: **Fraction of population in each zone by safety zone scenario.**

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

One of the interventions we simulate are different scenarios for allocating members of each population class to

the safe, 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 (necessarily children <13 in age group 1 and healthy adults in age group 2). In accordance with camp manager 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 <13 and 60% of the remainder of the green zone to adults 13-50 without comorbidities. We also consider a baseline scenario in which there is no green zone.

Parameterization of the contact matrix

We estimated the average number of contacts that individuals of class i have in a camp, \bar{c}_i , and we parameterized the contact matrix assuming that, in a well-mixed population, these contacts will be distributed among classes relative to the fraction of individuals within each class, i.e.

$$C_{ij}^0 = \bar{c}_i N_j / N, \quad (11)$$

with N the total population size and N_j the population size of class j . A well-mixed population will be considered the null model, and parameters derived under the null model assumptions are indexed with the superscript 0, e.g. the null contact matrix is C_{ij}^0 . Some of the interventions we considered, either reduce the average number of contacts a class i (e.g. self-isolation) or the probability that individuals of class i interact with those of class j (e.g. safety zone strategies). We model the first type of intervention introducing the parameter ϵ_{ij} , representing the fraction of the average number of contacts observed in the null model that prevail after the intervention: $\bar{c}_i = \epsilon_i \bar{c}_i^0$. Similarly, we model the second type of intervention with the matrix m_{ij} , representing the fraction of population j visible to population i after the intervention. The contact matrix resulting from management strategies can therefore be written with respect to the null model as:

$$C_{ij} = \epsilon_i m_{ij} \bar{c}_i^0 N_j / N = \epsilon_i m_{ij} C_{ij}^0 = M_{ij} C_{ij}^0. \quad (12)$$

We name the matrix M_{ij} the management matrix. Substituting Eq. 12 in the explicit expression of λ (Eq. 10) leads to a general expression for management strategies acting on the contact matrix:

$$\lambda_i = \frac{\tau}{N} \sum_{j=1}^n \epsilon_{ij} \bar{c}_i^0 m_{ij} (P_j + A_j + I_j + H_j) \quad (13)$$

Derivation of the transmissivity parameter τ

Estimation of the Next Generation Matrix

To estimate the probability of infection if there is a contact between a susceptible and an infected individual (parameter τ) we proceed as follows [Ref]. We start considering the subsystem containing the infectious population:

$$\dot{E}_i = \lambda_i S_i - \delta_E E_i \quad (14)$$

$$\dot{P}_i = \delta_E E_i - \delta_P P_i \quad (15)$$

$$\dot{A}_i = (1 - f) \delta_P P_i - \gamma_A A_i \quad (16)$$

$$\dot{I}_i = f \delta_P P_i - \kappa_i I_i \quad (17)$$

$$\dot{H}_i = h_i \eta I_i - \gamma_H H_i. \quad (18)$$

For the sake of simplifying the notation, let us consider the following ordering of the variables in the vector $x = (E_1, \dots, E_M, P_1, \dots, P_M, A_1, \dots, A_M, I_1, \dots, I_M, H_1, \dots, H_M)$, 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 unknown, and that does not change when interventions are introduced. For the null model, Eq. 13 becomes

$$\lambda_i = \frac{\tau}{N} \sum_{j=1}^n \bar{c}_i^0 (P_j + A_j + I_j + H_j).$$

Following this notation, the linearized system can be written in the form $\dot{\mathbf{x}} = (\mathbf{T} + \mathbf{\Sigma})\mathbf{x}$, where:

$$\mathbf{T} = \tau \begin{bmatrix} \mathbf{0} & \mathbf{\Theta} & \mathbf{\Theta} & \mathbf{\Theta} & \mathbf{\Theta} \\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} \end{bmatrix} \quad (19)$$

is the transmission matrix, with $\mathbf{\Theta} = \text{diag}(p_i \bar{c}_i^0) \mathbf{U}$, $p_i = N_i/N$, and \mathbf{U} being the all-ones matrix of size M . The transition matrix is

$$\mathbf{\Sigma} = \begin{bmatrix} -\delta_E \mathbf{I} & \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ \delta_E \mathbf{I} & -\delta_P \mathbf{I} & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & (1-f)\delta_P \mathbf{I} & -\gamma_A \mathbf{I} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & f\delta_P \mathbf{I} & \mathbf{0} & -\text{diag}(\kappa_i) \mathbf{I} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \eta \text{diag}(h_i) \mathbf{I} & -\gamma_H \mathbf{I} \end{bmatrix} \quad (20)$$

Where \mathbf{I} and $\mathbf{0}$ are the identity and null matrices of size M , and $\kappa_i = ((1 - g_i - h_i)\gamma_I + h_i\eta + g_i\alpha)$. We next compute the inverse of the transition matrix

$$\mathbf{\Sigma}^{-1} = \begin{bmatrix} -\frac{1}{\delta_E} \mathbf{I} & \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ -\frac{1}{\delta_P} \mathbf{I} & -\frac{1}{\delta_P} \mathbf{I} & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ -\frac{(1-f)}{\gamma_A} \mathbf{I} & -\frac{(1-f)}{\gamma_A} \mathbf{I} & -\frac{1}{\gamma_A} \mathbf{I} & \mathbf{0} & \mathbf{0} \\ -f \text{diag}(\frac{1}{\kappa_i}) \mathbf{I} & -f \text{diag}(\frac{1}{\kappa_i}) \mathbf{I} & \mathbf{0} & -\text{diag}(\frac{1}{\kappa_i}) \mathbf{I} & \mathbf{0} \\ -\frac{f\eta}{\gamma_H} \text{diag}(\frac{h_i}{\kappa_i}) \mathbf{I} & -\frac{f\eta}{\gamma_H} \text{diag}(\frac{h_i}{\kappa_i}) \mathbf{I} & \mathbf{0} & -\frac{\eta}{\gamma_H} \text{diag}(\frac{h_i}{\kappa_i}) \mathbf{I} & -\frac{1}{\gamma_H} \mathbf{I} \end{bmatrix} \quad (21)$$

The NGM with large domain can now be found by $\mathbf{K}_L = -\mathbf{T}\mathbf{\Sigma}^{-1}$. However, as we know that each individual that gets infected will become an exposed individual (E compartment), we focus on the NGM with small domain, \mathbf{K}_S that only consists of the E compartment [Heffernan]. We do this by removing from \mathbf{T} the rows that correspond to the other compartments and from $\mathbf{\Sigma}^{-1}$ the columns. We then find:

$$\mathbf{K}_S = \tau \left[\left(\frac{1}{\delta_P} + \frac{(1-f)}{\gamma_A} \right) \mathbf{\Theta} + \text{diag} \left(\frac{f}{\kappa_i} \left(1 + \frac{h_i\eta}{\gamma_H} \right) \right) \mathbf{\Theta} \right].$$

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

$$\tau = \frac{R_0}{|\tilde{\lambda}_1^0|}. \quad (22)$$

Parameterization of the interventions

Safety zone

We considered the existence of a safety zone to isolate certain fraction f_S of the population, mostly those more vulnerable. In practice, this is made dividing the camp in two areas, a “green” zone (denoted g) for the vulnerable population and an “orange” zone (o) for the remaining population. These two populations could eventually interact via a “buffering” zone, under controlled conditions. In particular, we considered that individuals entering in this zone

will meet in an open space, maintaining 2m of distance and using contention measures such as masks. We estimated that these measures would reduce the infectivity by an 80% [Ref], i.e. $\hat{\tau} = 0.2\tau$. In addition, each individual of the green zone, will be allowed to interact with a limited number c_{visit} of members (hereafter “visitors”) from the orange zone per day. Finally, in some interventions we considered that individuals visiting the buffering zone will have a health check (e.g. temperature measurement), aimed at excluding symptomatic patients from the buffering zone. In the model, the transmission probability between individuals from the orange zone at an I or H stage and individuals from the green zone is set to zero.

As we said, setting a safety zone implies a reduction in the number of contacts between classes of the green zone and the orange zone, but not in the mean number of contacts that each individual has per day, therefore \bar{c}_i does not vary. Therefore, it is needed to estimate how the contacts will be redistributed from those occurring in a well-mixed model with individuals from a different zone towards members living in the same zone. We model this redistribution of the contacts with the parameter ϵ_i :

$$\begin{aligned}\epsilon_i &= \rho c_{\text{visit}} / \bar{c}_i \quad (i, j \text{ in different areas}) \\ \epsilon_i &= 1 - \rho c_{\text{visit}} / \bar{c}_i \quad (i, j \text{ in same area}).\end{aligned}$$

If we assume that visitors are always different, the quantity $f_{o,\text{visit}} = c_{\text{visit}} \frac{N_g}{N_o}$ is the fraction of the orange population susceptible of visiting the buffering zone. We define ρ as¹:

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

Next, we model the probability of interaction between a member of the class i and the class j , depending on whether they belong to the same or to different areas. Since in the intervention classes are constrained to interact within their own area, compared to the null model any individual will experience an increased likelihood of finding members of the classes staying in the same area. More specifically, the proportion N_i/N of individuals for class i in the null model will become N_i/N_X with N_X the total number of individuals in the area $X = \{o, g\}$. This leads to the following values for m_{ij} :

$$\begin{aligned}m_{ij} &= \left(\frac{N_i}{N_X}\right) / \left(\frac{N}{N_i}\right) = \frac{N}{N_X} \quad (i, j \text{ in same area } X) \\ m_{ij} &= \left(\frac{N_i}{N_Y}\right) / \left(\frac{N}{N_i}\right) = \frac{N}{N_Y} \quad (i \in X \text{ and } j \in Y).\end{aligned}$$

We finally define the management matrix as $M_{ij} = \epsilon_i m_{ij}$.

Estimation of the infectivity of the isolated and evacuated populations

To estimate the infectivity of the population isolated we depart from the following assumptions. Firstly, the population class taking care of the isolated individuals belongs to the one of adults with no comorbidities. We considered a number N_{care} of carers having c_{care} contacts per day and carer with the isolated population. The individuals that can belong to the group of carers are those alive individuals having no symptoms. We denote the number of individuals fulfilling these requirements with N_{exp} (number of exposed). To continue with, we considered that when the number of symptomatic individuals exceeds the isolation capacity, \tilde{N} , the individuals in excess are fully infectious (note that we use a tilde to denote variables related to the isolated population). In addition, the occupancy of the isolation beds is distributed among classes proportionally to the number of symptomatic individuals that each class contributes, i.e. $\tilde{N}_j = \tilde{N} \left(I_j / \sum_j I_j \right)$. Finally, symptomatic individuals developing symptoms that would require hospitalization, are either evacuated or they become fully infectious. The rationale behind the latter

¹If c_{visit} is large enough ($c_{\text{visit}} \approx 28$ contacts per week) it should be considered that this function saturates, because every member of the orange zone would eventually visits the buffering zone:

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

with the Heaviside function $H(f_{o,\text{visit}} - 1) = 1$ if $f_{o,\text{visit}} \geq 1$. We chose values well below these values.

choice is that, if an individual requires a more dedicated care, the available means in the camps to protect the population from these patients would be insufficient. In particular, it is unlikely that this person can stay alone in a tent. We model the evacuation considering a parameter $\epsilon = 0$ if evacuation is put in place and $\epsilon = 1$ otherwise. Evacuated individuals are no longer infectious.

Given these assumptions, the number of contacts that the adult and healthy population class will have with the isolated population will be $c_{\text{care}}N_{\text{care}}/N_{\text{exp}}$ per individual and day. The expression clearly shows that, increasing the number of carers, the number of isolated individuals, and the number of contacts per day between carers and individuals, will increase the rate of infection. Hence, we expect that, for fixed N_{care} and c_{care} , the positive effects coming from isolating individuals will be less pronounced for increasingly large \tilde{N} values. We further assume that this interaction is regulated following the guidelines introduced for a safety zone, and the infectivity becomes thus reduced by a factor $\xi = 0.2$. Finally, we should note that the probability of finding an isolated individual belonging to class j , is equal to $(N_j/N)(\tilde{N}_j/N_j)$, but this probability is equal to one for the healthy adult population (due to their role of carers) and equals zero for the remainder classes (since they have no access to the isolation area).

For simplicity, we assume that there is one carer for each infected person in the class j , ($N_{\text{care},j} = \tilde{N}_j$), having only one contact per day (c_{care}). Note the convenience of this choice, since if the number of symptomatic individuals is larger than the number of individuals susceptible of being carers, the ratio $\tilde{N}_j/N_{\text{exp}} > 1$, meaning that more than one contact per day is needed to take care of that population class. With these considerations, the rate of infection for the healthy adult population class (indexed k) becomes:

$$\lambda_k = \tau \sum_j \xi \frac{\tilde{N}_j}{N_{\text{exp}}} + C_{kj} \frac{P_j + A_j + \Theta(N_I - \tilde{N})(I_j - \tilde{I}_j) + \epsilon H_j}{N_j},$$

where Θ is the Heaviside function and N_I the total number of symptomatic individuals at time t . For the remainder classes ($i \neq k$) the rate of infection becomes:

$$\lambda_i = \tau \sum_j C_{ij} \frac{P_j + A_j + \Theta(N_I - \tilde{N})(I_j - \tilde{I}_j) + \epsilon H_j}{N_j}.$$

A last consideration is that symptomatic individuals require some time to recognize their symptoms and to decide that self-isolation is needed. To model this fact, we considered whenever the isolation intervention is incorporated, that the symptomatic compartment is split in two compartments: onset of symptoms, I_i^{onset} and symptomatic, I_i . We considered three that the time at I_i^{onset} followed a Gaussian distribution with means 12, 24 or 48 hours on average. The time that the individual can isolate is then calculated as the difference between the time at the symptomatic compartment if there is no isolation, $1/\eta$, and the time at the onset compartment.

Supplementary figures

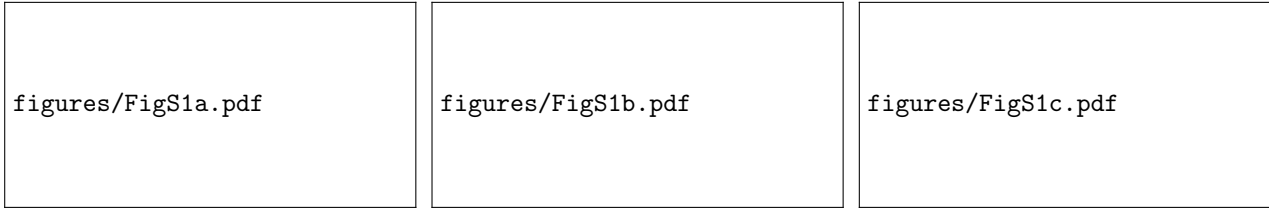


Figure 2: **Lower and upper bounds when interventions are absent.** Probability of outbreak (left), fraction of casualties (middle) and time in which the number of symptomatic cases peaks (right), as a function of the fate of the individuals in the H compartment, that may all recover or all die.

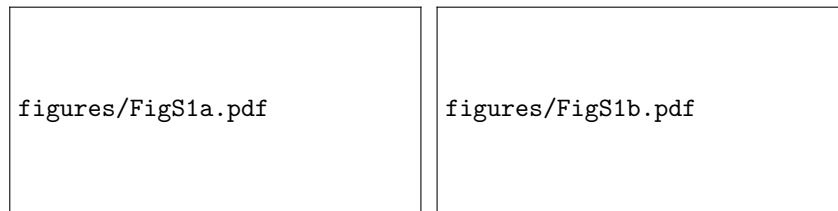


Figure 3: **Self-distancing.** CFR (left), and fraction of recovered population (right) as a function of the reduction in the fraction of contacts that the whole population experience self-distancing.

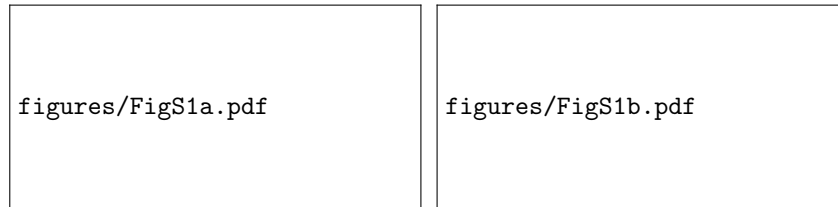


Figure 4: **Self-isolation.** CFR (left), and fraction of recovered population (right) as a function of the number of tents available in the camp.

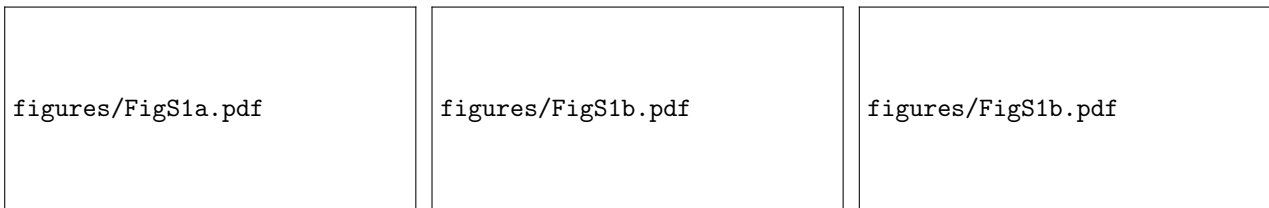


Figure 5: **Time to self-isolation.** Probability of outbreak (left), fraction of casualties (middle) and time in which the number of symptomatic cases peaks (right), as a function of the time that individuals require to recognize their symptoms and self-isolate.

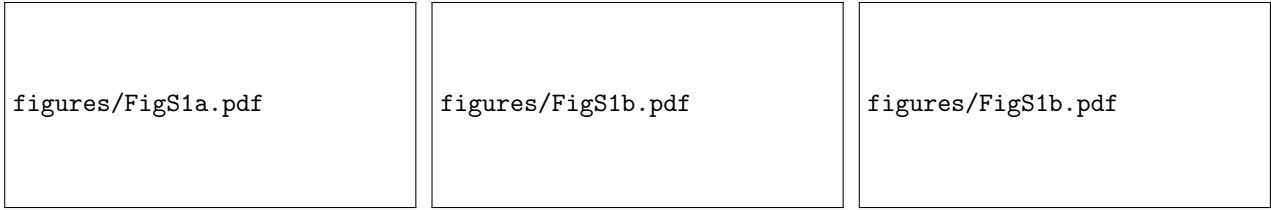


Figure 6: **Evacuation.** Probability of outbreak (left), fraction of casualties (middle) and time in which the number of symptomatic cases peaks (right), depending on whether individuals requiring hospitalization are evacuated to isolation centers.

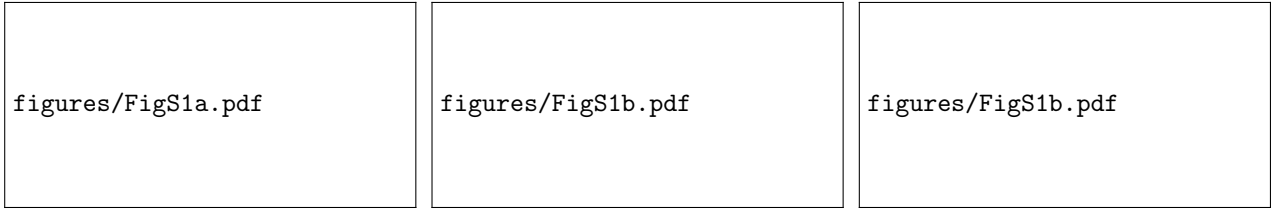


Figure 7: **Health-checks in the buffering zone.** Probability of outbreak (left), fraction of casualties (middle) and time in which the number of symptomatic cases peaks (right), depending on whether health-checks are implemented to avoid that symptomatic individuals access to the buffering zone separating the orange and green zones.

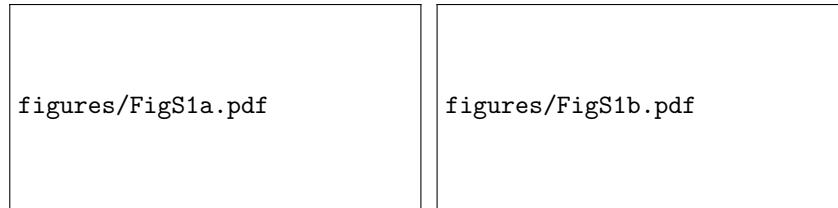


Figure 8: **Number of contacts in the buffering zone.** CFR (left), and fraction of recovered population (right) as a function of the number of contacts per week and individual permitted in the buffering zone, in which the populations living in the green and orange zones are allowed to interact.



Figure 9: **Population moving to the safety zone.** Probability of outbreak (top-left), fraction of casualties (top-middle), time in which the number of symptomatic cases peaks (top-right), CFR (bottom-left) and fraction of recovered individuals (bottom-right), as a function of the population moving to the safety zone.



Figure 10: **Efficiency of safety zone for different population sizes.** Probability of outbreak (top-left), fraction of casualties (top-middle), time in which the number of symptomatic cases peaks (top-right), CFR (bottom-left) and fraction of recovered individuals (bottom-right), as a function of the total population size of the camp.



Figure 11: **Lockdown of the safety zone.** Probability of outbreak (top-left), fraction of casualties (top-middle), time in which the number of symptomatic cases peaks (top-right), CFR (bottom-left) and fraction of recovered individuals (bottom-right), as a function of the reduction in the number of contacts permitted in the buffering zone.

References

Maximum number of family members permitted

[APG, the following should be reviewed, it is unfinished] We considered until now that people from the orange area visiting the buffering zone will be always different within the same week. An interesting question for management is how to keep the fraction of the orange population using the buffering zone as small as possible to minimize the spread of the infection. This relies on the assumption that, since symptomatic members are excluded from the buffering zone, increasing this fraction increases the chances for asymptomatic or presymptomatic individuals to visit the buffering zone. As we will see immediately, this observation could be used to improve the management strategies.

To illustrate this point we note that the fraction of the orange population getting in contact with the green population can be estimated through the quantity $0.2\epsilon_{ij}m_{ij}$ ($i \in o$), which we aim to keep lower than one. Expanding this expression we obtain

$$c_{\text{visit}} < 5\sqrt{\bar{c}_i \frac{N_o}{N}}$$

with a value of ≈ 17 individuals per day for the adult class and assuming 20% of the population shielded. Note that the estimation heavily relies on the efficiency of the protection measures.

The clan’s dilemma

In the previous section, we considered that the family members visiting were always different. However, if the population in the camps is structured in large families (hereafter clans) it opens the question of whether it would be more beneficial to keep all members of the same clan in the same area or in different areas. If all members of the same clan live in the same zone, there would be little need to interact with members of the other zone, hence reducing c_{visit} . However, since the capacity of the green zone is limited, shielding whole clans may lead to leaving other clans completely outside of the green zone, even if the strategy may be better to minimize the overall impact in the population. On the other hand, if clans are split, it increases the chances that the same relatives visit the buffering zone several times. Under the assumption that minimizing the number of different people would reduce the probability of infection, this could also be a positive strategy.

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