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IFAC PapersOnLine 56-2 (2023) 9733–9738

Testing and preemptive quarantine for the control of epidemics

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Abstract: Testing prior symptoms onset and preemptive quarantine of closest contacts are important control actions to mitigate epidemic spreading, especially when asymptomatic and pre-symptomatic shedding is relevant. In order to understand the effects of testing and preemptive quarantine, in this work we introduce a compartmental model that includes novel detection and isolation rates to better capture the state distribution of the close contacts of infected individuals. Moreover, since preemptive quarantine might be seen as an overlay restrictive measure and might be not properly followed, we explicitly include in the model the compliance with the entry and the exit from quarantine. We study the asymptotic stability of the disease-free equilibrium. We show that increasing the number of tests allows to eradicate the epidemic, while increasing the number of quarantined individuals per detected might negatively affect the stability, though it may reduce the peak of active cases.

Keywords: Contact tracing, Compartmental model, Resource allocation.

1. INTRODUCTION

In the early stages of Covid–19 epidemic, generalized lockdown has been the only control measure to contain the diffusion of the disease. In later stages, epidemic control has relied on testing prior symptoms onset and preemptive quarantine for contacts of infected. Increasing the number of tests per day and the number of quarantined contacts improves the effectiveness of the strategy but results in a higher social-economical cost due to the price of test kits and the amount of individuals on leave from work. Moreover, increasing the number of quarantined contacts per detected individual by including also more loose interactions might result in weaker compliance in the population and can compromise the control efforts. For these reasons, it is important to properly assess the effects of testing and preemptive quarantine in the epidemic evolution, especially focusing on their interplay and accounting for individual compliance.

Recently, many efforts have been devoted to devise novel models to predict and control disease spreading. Several agent-based models and simulators have been devised, e.g. Kerr et al. (2021). These models are usually accurate for forecasting but are often too complex for control design. For this reason, compartmental models are usually preferred in the literature. Most of the existing works on epidemic control have focused on the population-level actions that reduce the average number of contacts or the transmission probability. For instance, Köhler et al. (2021) propose an MPC to optimize the contact rate and Bin et al. (2021) propose a fast intermittent lockdown policy with a constant period. However, testing and preemptive quarantine are less studied in the control community. Giordano et al. (2020) devise a compartmental model including testing and conditional quarantine of known positive but the number of detected per day is assumed to be a fixed portion of the number of infected. A similar idea has been used by Calafiore et al. (2020), while a more advance model including test allocation specificity has been proposed by Niazi et al. (2021) but it is limited to testing. Similarly, models including preemptive quarantine of the contacts of detected have been proposed by Tang et al. (2020) and by Ngonghala et al. (2020). Also in this case, the number of preemptively quarantined individuals per day is assumed to be a fixed portion of susceptible. Moreover, compliance with preemptive quarantine has not been assessed.

The main contribution of the paper is to propose a novel compartmental model that encompasses different control actions available to the decision maker. We consider the actions aiming to reduce the disease transmissibility, either reducing the average number of contacts or reducing the transmission probability per contact, e.g. lockdown, curfew, mobility limitation, social distancing, use of personal protective equipment. More importantly, we propose a new model for detection and quarantining: rather than simple constant rates, we propose a more elaborated rate that depends on a weighted combination of the current state of all the compartments. The underlying idea is that the distribution of the states of the close contacts of an infected is only partially affected by the overall distribution of the states in the population. In other words, while the

⋆ This work has been supported by the Fonds de la Recherche Scientifique-FNRS under the grant 40003443 (Smart Testing).

10.1016/j.ifacol.2023.10.287

probability of being infected for an individual being picked randomly is proportional to the number of infected, the probability that a close contact of a positive is infected is more affected by the strength of the interaction than by the total number of infected in the population. In this sense, we go beyond the macroscopical approach used in the literature and we try to capture the microscopical characteristic of close contacts. Additionally, we include in the model the compliance with preemptive quarantine by properly decreasing the probability of entering and remaining in isolation when more contacts are quarantined.

Based on the proposed model, we rigorously study the asymptotic stability of the system highlighting the dependence on the number of tests and the number of quarantined contacts. Interestingly, we show that the asymptotic stability of the disease-free equilibrium is affected by the number of tests but it is not directly affected by the number of quarantined contacts. On the other hand, the number of traced contacts can be increased up to a certain threshold in order to reduce the peak of infected individuals. Above the threshold, preemptively quarantine has a negative effect on the epidemic evolution due to the lower compliance. We highlight that there probably is a trade-off between the financial efforts to increase the number of tests and the number of quarantined contacts.

The paper is organized as follows. In Sec. 2 we introduce the proposed model and we carefully describe its features. In Sec 3 we compute the basic reproduction number and we use it to draw relevant considerations about the effects of control actions on the stability of the disease-free equilibrium. In Sec. 4 we numerically show how the number of tests and the number of quarantined individuals per detected affect the evolution. The paper ends with Sec. 5.

2. MODEL

In this section, we introduce a new compartmental model for epidemics. We specifically include two kinds of control actions: 1) population-level interventions aiming to reduce the disease transmission rate by either reducing the average number of contacts (e.g. lockdown, curfew) or reducing the transmission probability per contact (e.g. social distancing, face masks), 2) targeted isolation of known infectious individuals and close contacts through testing and preemptive quarantine.

The states of the model are the number S of susceptible individuals not subject to quarantine, the number I of infected individuals not subject to quarantine, the number Q_s of susceptible individuals subjected to quarantine, the number Q_i of infected individuals subjected to quarantine, and the number R of recovered immune individuals. The states evolve as

$$\dot{S} = -\beta\rho SI/N - q_s(S,I,R) + \mu_s Q_s + \delta R \quad (1)$$
$$\dot{I} = \beta\rho SI/N - q_i(S,I,R) + \mu_i Q_i - \gamma I \quad (2)$$
$$\dot{Q}_s = q_s(S,I,R) - \mu_s Q_s \quad (3)$$
$$\dot{Q}_i = q_i(S,I,R) - \mu_i Q_i - \gamma Q_i \quad (4)$$
$$\dot{R} = \gamma I + \gamma Q_i - \delta R \quad (5)$$

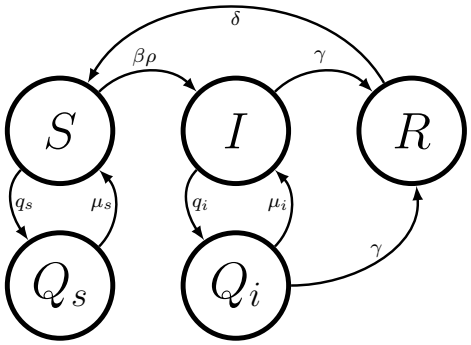

Fig. 1. Graphical representation of the proposed model and state transitions

where $N = S + I + Q_s + Q_i + R$ is the total amount of individuals, $\beta \in (0,1)$ is the disease transmission probability per individual in the case without control actions, $\rho \in [0,1]$ is the reduction factor of the disease transmission probability achieved through population-level control actions, $q_s(S,I,R)$ is the effective amount of susceptible individuals quarantined in the time unit, $q_i(S,I,R)$ is the effective amount of infected individuals quarantined in the time unit, $\mu_s \in (0,1)$ is the exit probability of a quarantined susceptible individual, $\mu_i \in (0,1)$ is the exit probability of a quarantined infected individual while being still infected, $\gamma \in (0,1)$ is the recovery probability of an infected individual, $\delta \in (0,1)$ is the probability of losing the immunity. All the probabilities must be intended in the time unit, which can be taken equal to a day for sake of simplicity. A pictorial representation of the model and of the transitions is given in Fig. 1. In the following we provide more details on the features of the model.

Population-level control actions. We propose to model the reduction factor ρ_u of the disease transmission probability for a population-level control action u as

$$\rho_u = 1 - \lambda_u \varepsilon_u \quad (6)$$

where $\varepsilon_u \in [0,1]$ is the efficacy of the control action and $\lambda_u \in [0,1]$ is the compliance with the control action in the population. For instance, a complete lockdown with perfect compliance would have $\varepsilon_u = 1$ and $\lambda_u = 1$, and it would result in a reduction factor $\rho_u = 0$. If we consider the case where a set $\mathcal{U}$ of population-level control actions is applied, under the assumption that the effects of different control actions on the disease transmission are independent, the total reduction factor can be expressed as $\rho = \prod_{u\in\mathcal{U}} \rho_u$. A more elaborated model can be obtained following Martins et al. (2022).

Testing. Under the simplifying assumption that quarantined individuals are not tested, the probability that a tested individual is infected can be modeled as

$$P_i = \frac{\beta_i I}{\beta_s S + \beta_i I + \beta_r R} \quad (7)$$

where β_s, β_i, $\beta_r \in \mathbb{R}_{>0}$ capture how effective is the testing strategy in finding positive individuals. For instance, if tests are allocated randomly within the non-quarantined population, then $\beta_s = \beta_i = \beta_r$. If a more effective testing

policy such as Pezzutto et al. (2021) is used, then $\beta_i > \beta_s$.

Remark 2.1. Let P_s be the probability that a tested individual is susceptible. Then, P_i/P_s represents how much larger the probability of testing an infected is than the probability of testing a susceptible. It is easy to see that, if tests are allocated randomly, then $P_i/P_s = I/S$, while, if tests are allocated according to an arbitrary testing policy, then $P_i/P_s = (\beta_i I)/(\beta_s S)$. It follows that the ratio β_i/β_s represents the scaling factor between the ratio P_i/P_s achieved by the arbitrary testing policy and the ratio P_i/P_s achieved by random testing.

Under the assumption that the number of tests used is fixed and individuals with clear symptoms are diagnosed without testing, the amount D of individuals detected in the time unit can be computed as

$$D = \sigma I + \frac{\beta_i I}{\beta_s S + \beta_i I + \beta_r R} T \tag{8}$$

where $\sigma \in [0, 1)$ is the probability that an infected individual shows symptoms in the time unit and T is the number of tests used in the time unit.

Remark 2.2. Direct diagnosis for symptomatic individuals is used with the aim of saving tests whose outcomes are known in advance with very high confidence. This is particularly relevant at the early stages of epidemics when the testing capacity is limited. If symptomatic individuals are diagnosed through testing, the model still applies but the number T of tests allocated to detect asymptomatic and pre-symptomatic individuals has to be reduced accordingly.

Quarantine. We assume that new detected individuals are quarantined. Moreover, we assume that a fixed number of individuals are preemptively quarantined per detected individual. Accordingly, the amount of infected individuals quarantined in the time unit can be modeled as

$$q_i^o(S, I, R) = \left(1 + \frac{\alpha_i I}{\alpha_s S + \alpha_i I + \alpha_r R} L\right) D \tag{9}$$

where L is the number of quarantined individuals per detected while α_s, α_i, $\alpha_r \in \mathbb{R}_{>0}$ capture how effective is the quarantining strategy in finding positive individuals. For instance, if individuals to quarantine are picked randomly from the population, then $\alpha_s = \alpha_i = \alpha_r$. However, if the closest contacts of an infected are chosen to be quarantined, then a higher number of infected is expected to be isolated, so $\alpha_i > \alpha_s$. The amount of susceptible individuals quarantined in the time unit can be modeled as

$$q_s^o(S, I, R) = \left(\frac{\alpha_s S}{\alpha_s S + \alpha_i I + \alpha_r R} L\right) D \tag{10}$$

Note that the number of preemptive quarantined individuals is proportional to the number of detected.

Remark 2.3. Notably, when close contacts are quarantined, the weights α_s, α_i, α_r are used to model the distribution of the states of the close contacts of a positive instead of the distribution of the states in the whole population. The rationale is that, since the population can be seen as a network of local interactions, the knowledge on the state of an individual is more affected by the knowledge that a close contact is positive, rather than the total number of infected. The use of a weighted combination of the states is an attempt to encapsulate this evidence about the microscopical local-level transmission mechanism in the macroscopical population-level model.

Remark 2.4. A reasoning similar to Remark 2.1 applies also for the ratio α_i/α_s. Notably, if close contacts of detected individuals are tested, then a reasoning similar to Remark 2.3 applies also for the ratio β_i/β_s.

Finally, taking into account the compliance with the quarantine request, the effective amount of susceptible individuals quarantined in the time unit and the effective amount of infected individuals quarantined in the time unit are

$$q_s(S, I, R) = \lambda_{q,s} q_s^o(S, I, R) \tag{11}$$
$$q_i(S, I, R) = \lambda_{q,i} q_i^o(S, I, R) \tag{12}$$

where $\lambda_{q,s} \in [0, 1]$, $\lambda_{q,i} \in [0, 1]$ are the compliance with the quarantine entry for a susceptible and for an infected, respectively. They can be defined as the probability that an individual will follow the restriction and actually self-isolate. In general, an imperfect compliance decreases the number of individuals quarantined in the time unit.

We model the quarantine exit rate of susceptible individuals and of infected individuals as

$$\mu_s = \mu + (1 - \lambda_{e,s}) \tag{13}$$
$$\mu_i = \mu + (1 - \lambda_{e,i}) \tag{14}$$

where $\mu \in (0, 1)$ is the rate at which quarantined individuals end the quarantine, while $(1 - \lambda_{e,s}) \in [0, 1)$ and $(1 - \lambda_{e,i}) \in [0, 1)$ are the rate at which quarantined susceptible and infected individuals leave the quarantine before the prescribed end. In this sense, $\lambda_{e,s}$ and $\lambda_{e,i}$ represent the compliance with the quarantine exit for a susceptible and for an infected individual. Indeed, with perfect compliance we have $1 - \lambda_{e,s} = 0$, $1 - \lambda_{e,i} = 0$ and quarantine exit rate coincides with the prescribed one.

In general, it has been observed that individuals are less adherent to interventions that are perceived as overlay restrictive (see e.g. Acuña-Zegarra et al. (2020)). Since increasing the number of quarantined individuals per detected is equivalent to quarantining more loose contacts, we assume that compliance $\lambda_{q,s}, \lambda_{q,i}, \lambda_{e,s}$, and $\lambda_{e,i}$ are non-increasing function of L.

3. BASIC REPRODUCTION NUMBER

It is easy to show that the system has an unique disease-free equilibrium $\bar{X} = (\bar{S}, \bar{I}, \bar{Q}_s, \bar{Q}_i, \bar{R}) = (N, 0, 0, 0, 0)$. The asymptotic behavior of the disease-free equilibrium is a key property of the system because it provides important insight on how to allocate control actions. In order to study it, we introduce

$$\mathcal{R}_0 = \frac{\beta \rho_u}{\gamma + \left(\frac{\gamma}{\gamma + \mu_i}\right) \frac{dq_i}{dI}(N, 0, 0)} \tag{15}$$

$$= \frac{\beta(1 - \lambda_u \varepsilon_u)}{\gamma + \lambda_{q,i}\left(\frac{\gamma}{\gamma + \mu + (1 - \lambda_{e,i})}\right)\left(\sigma + \frac{\beta_i}{\beta_s}\frac{T}{N}\right)} \tag{16}$$

Then we can provide the following proposition. Proof is given in Appendix.

Proposition 1. The disease-free equilibrium $\bar{X}$ is asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.

From the proposition above, we can see that asymptotic stability is ruled by $\mathcal{R}_0$, which represents the basic reproduction number of the proposed model.

As expected, $\mathcal{R}_0$ is non-decreasing with respect to the transmission rate β and the exit rate from quarantine μ, while it is non-increasing with respect to the recovery rate γ and the symptom appearance rate σ.

We can see that $\mathcal{R}_0$ is proportional to the reduction factor ρ_u of the transmission rate achieved by the population-level control action u. As expected, $\mathcal{R}_0$ decreases for a higher efficiency ε_u but increases for a lower compliance λ_u. This is in accordance with the common intuition that stricter interventions (usually more efficient) are convenient as long the population is compliant, and a population-level control action is optimal if it maximizes the product $\lambda_u \varepsilon_u$.

Notably, asymptotic stability is affected by the number of tests and the testing policy. In particular, we can see that $\mathcal{R}_0$ is non-increasing with respect to T and there exists a threshold T_c such that the disease-free equilibrium is asymptotically stable if $T > T_c$. Moreover, it is interesting to stress that the testing policy affects $\mathcal{R}_0$ through the term β_i/β_s, indicating how advanced test allocation strategies are important for epidemic control.

Surprisingly, $\mathcal{R}_0$ is not directly affected by the number of quarantined per detected as L does not appear in (16). It follows that asymptotic stability of the disease-free equilibrium cannot be achieved by an arbitrary high L. Even more, since $\lambda_{q,i}$ and $\lambda_{e,i}$ are non-increasing functions of L and $\mathcal{R}_0$ is a non-increasing function of $\lambda_{q,i}$ and $\lambda_{e,i}$, then $\mathcal{R}_0$ results to be non-increasing with respect to L. This means that it is counterproductive to increase the number of quarantined individuals per detected in terms of asymptotic stability. However, improvements might be obtained during the transient.

It is worth mentioning that $\mathcal{R}_0$ does not depend on $\lambda_{q,s}$ and $\lambda_{e,s}$. It follows that asymptotic stability is not affected by the compliance with quarantine for susceptible individuals but only by the compliance with quarantine for infected individuals. This suggests that it is important to enhance the adherence to quarantine for known positive e.g. through public awareness campaigns or targeted inspections to verify if detected individuals follow the isolation requirements.

4. NUMERICAL RESULTS

In this section, we use the proposed model to numerically evaluate the evolution of the number of infected for different values of the number of tests and of the number of quarantined per detected.

We consider the Covid–19 case. We set $\gamma = 1/14$ (as indicated by Ngonghala et al. (2020)) while we set $\beta = 0.1786$ in such a way the basic reproduction number without any intervention is equal to 2.5 (as indicated by Giordano et al. (2020)). Since the recommended period of quarantine for an infected individual is 14 days, we set $\mu = 1/14$ (as done in Ngonghala et al. (2020)). We assume that the probability that an infected individual shows symptoms at a given day is $\sigma = 0.1$ (as indicated by Biswas et al. (2020)). We assume that, on average, an individual

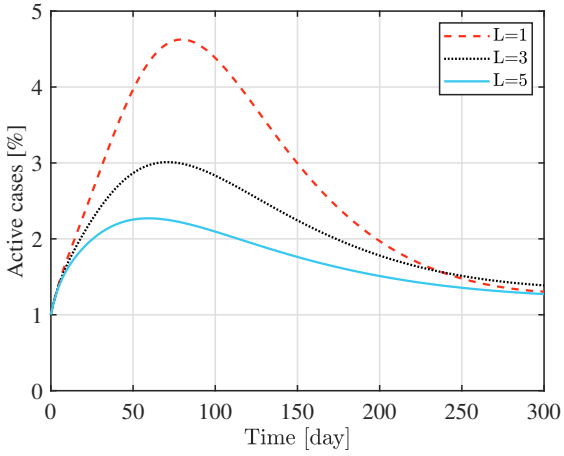

Fig. 2. Evolution of the percentage of active cases by varying the number of quarantined per detected

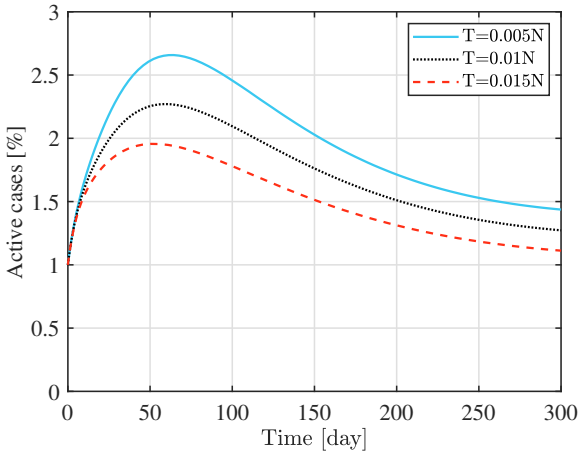

Fig. 3. Evolution of the percentage of active cases by varying the number of tests

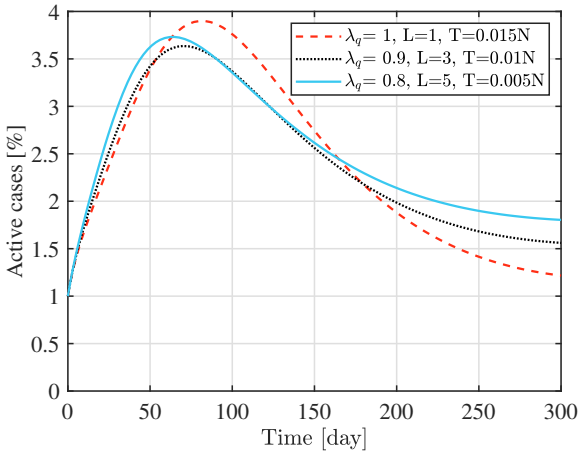

Fig. 4. Evolution of the percentage of active cases by varying the number of tests and the number of quarantined per detected

loses immunity after recovery in 180 days, so we set $\delta = 1/180$ (as indicated by Mandal et al. (2021)). In order to highlight the effects of testing and preemptive quarantine, we assume that no population-level control actions are applied, so we set $\epsilon_u = 0$. Moreover, we assume perfect compliance with the exit from quarantine $\lambda_{e,s} = \lambda_{e,i} = 1$ and, where not explicitly mentioned, perfect compliance with the entry to quarantine $\lambda_{q,s} = \lambda_{e,s} = 1$. Using the agent-based model by Bono Rossello et al. (2021), it has been possible to simulate the epidemic evolution with the considered parameters and to check the distribution of the states of the close contacts of detected individuals. In this way, we roughly estimate the parameters α_s, α_i, α_r to be equal to $0.2, 0.91, 0.27$, respectively. Moreover, the parameters β_s, β_i, β_r are equal to $0.41, 0.82, 0.41$. The total number of individuals N is set equal to 1 million.

In Fig. 2, we consider a fixed number of tests $T = 0.01N$ and we vary the number of people quarantined per detected as $L \in \{1, 3, 5\}$. As we can see, increasing L from 1 to 5 reduces the peak of infected individuals by 50%, even though all of the 3 curves tend to converge to the same value. These results show that preemptive quarantine can be effective to alleviate the outbreak of the epidemic during the transient phase.

In Fig. 3, we consider a fixed number of quarantined people per detected $L = 5$ and we vary the number of tests as $T \in \{0.005N, 0.01N, 0.015N\}$. We can see that the more we increase the number of tests, the lower is the peak of active cases and the lower is the number of infected at steady-state. In particular, the peak of active cases decreases by 25% when we increase the number of tests from the lowest value ($T = 0.005N$) to the highest one ($T = 0.015N$). It is worth mentioning that, with the considered disease parameters, a number of tests equal to $T = 0.06N$ is needed to achieve $\mathcal{R}_0 < 1$ and to converge to the disease-free equilibrium. Notably, $T = 0.01N$ is equal to the maximum number of daily tests made in Belgium during the Covid-19 pandemic (see Belgian Institute for Health (Sciensano) (2022)).

In Fig. 4, we study the interplay between testing and preemptive quarantine by varying both T and L. In this case, we take into account the lower compliance associated with a higher number of quarantined per detected. More specifically, we assume that $\lambda_{q,s} = \lambda_{q,i} = 1$ for $L = 1$, $\lambda_{q,s} = \lambda_{q,i} = 0.9$ for $L = 3$ and $\lambda_{q,s} = \lambda_{q,i} = 0.8$ for $L = 5$. As we can see, in the long term (time t=300 day), the number of tests and compliance play a determinant role in reducing the number of infected, while a higher number of quarantined per detected does not result in a lower steady-state number of infected. This is in accordance with Fig. 2 and Fig. 3. More interestingly, we can see that the smallest peak is achieved with a good balance between the number of tests and the number of quarantined per detected ($T = 0.01N$, $L = 3$). This suggests that there might be a trade-off between the economical resources allocated for testing (in terms of test kits) and those allocated for quarantine (in terms of individuals on leave from work). Moreover, the best resource allocation might change depending on if the objective is the worst case (i.e. peak of active cases) or the steady state of the pandemic. Remarkably, this insight can not be obtained with simpler models that do not consider testing and quarantine.

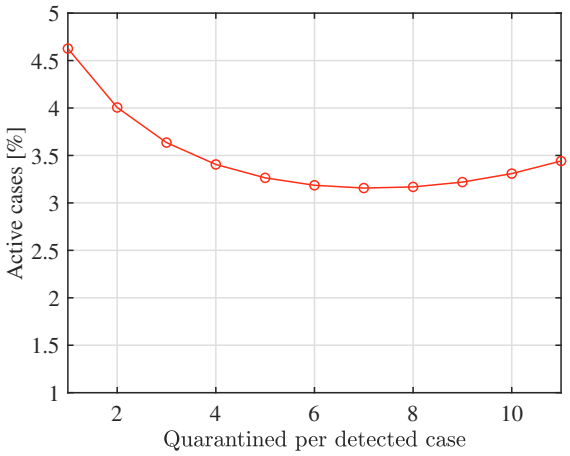

Fig. 5. Peak of the percentage of active cases by varying the number of quarantined per detected

In Fig. 5, we plot the maximum number of infected for different values of the number of quarantined per detected. We consider a fixed number of tests $T = 0.01N$, we increase the number of quarantined per detected gradually from $L = 1$ to $L = 10$, and, consequently, we decrease the compliance $\lambda_{q,i}$. Since there is no data in the literature relating the compliance with the preemptive quarantine, for illustrative purposes we propose to consider $\lambda_{q,i} = 1 - 0.05L$. On one hand, we can see that $L = 6$ achieves the smallest peak of active cases with an improvement of 30% with respect to $L = 1$. On the other hand, values greater than $L = 6$ lead to an increment in the peak of active cases due to the lower compliance and so they are not convenient for control purposes.

We can conclude that moderate values of the number of quarantined per detected can be effective to decrease the peak of active cases and, possibly, can compensate for a limited testing capacity. However, increasing too much the number of individuals preemptively quarantined would have a negligible, or even negative, effect on the epidemic evolution due to the lower compliance. In general, massive testing to detect asymptomatic and pre-symptomatic can achieve good results both in terms of peak and steady state value of active cases.

5. CONCLUSIONS

In this work, we have proposed a new compartmental model that takes into account testing, preemptive quarantine, and compliance with the control actions. We have mathematically studied the asymptotic stability of the disease-free equilibrium and we have numerically evaluated the effects of varying the number of tests and the number of quarantined individuals per detected.

As a future work, the interplay between the testing policy and the quarantine policy can be studied to understand if it is more convenient to test or to quarantine specific individuals, like the closest contacts of a positive. Moreover, the model can be used to study the general optimal resource allocation strategy combining population-level interventions with testing and preemptive quarantine.

6. APPENDIX

Proof. [Proposition 1] The proof relies on next generation matrix (see Diekmann et al. (2010)). Following the notation used by Van den Driessche and Watmough (2008), let $x = (I, Q_i)'$ be the state of disease compartments and $y = (S, Q_s, R)'$ be the state of non-disease compartments. Introduce the matrices

$$\mathcal{F}(x,y) = \begin{bmatrix} \mathcal{F}_1(x,y) \\ \mathcal{F}_2(x,y) \end{bmatrix} = \begin{bmatrix} \beta\rho SI \\ 0 \end{bmatrix}$$

$$\mathcal{V}(x,y) = \begin{bmatrix} \mathcal{V}_1(x,y) \\ \mathcal{V}_2(x,y) \end{bmatrix} = \begin{bmatrix} q_i(S,I,R) - \mu_i Q_i + \gamma I \\ -q_i(S,I,R) + \mu_i Q_i + \gamma Q_i \end{bmatrix}$$

$$g(x,y) = \begin{bmatrix} q_i(S,I,R) + \mu_s Q_s + \delta R - \beta\rho SI \\ q_s(S,I,R) - \mu_s Q_s \\ \gamma I + \gamma Q_i - \delta R \end{bmatrix}$$

where $\mathcal{F}$ contains the infection rate and $\mathcal{V}$ contains the disease progression, so that the model can be written in compact form as

$$\dot{x} = \mathcal{F}(x,y) - \mathcal{V}(x,y)$$
$$\dot{y} = g(x,y)$$

We now verify the assumptions made by Van den Driessche and Watmough (2008). Since $q_i(S,0,R) = 0$, then $\mathcal{F}_\ell(0,y) = 0$ and $\mathcal{V}_\ell(0,y) = 0$ for $\ell = 1,2$, thus (A1) holds. Since $\beta\rho \geq 0$, then $\mathcal{F}_\ell(x,y) \geq 0$ for $\ell = 1,2$ for non-negative x and y, thus (A2) holds. Since $q_i(S,0,R) = 0$, then $\mathcal{V}_1(0,Q_i,y) = -\mu_i Q_s \geq 0$ and, since $q_i(S,0,R) \geq 0$ for $S,I,R \geq 0$, the $\mathcal{V}_2(I,0,y) = -q_i(S,I,R) \geq 0$, thus (A3) holds. We have $\mathcal{V}_1(x,y) + \mathcal{V}_2(x,y) = \gamma I + \gamma Q_i \geq 0$ for non-negative x, thus (A4) holds. Since $q_i(S,0,R) = 0$, we have that $g(0,y) = (\delta R + \mu_s Q_s, \mu_s Q_s, -\delta R)'$. Since $\delta > 0$ and $\mu_s > 0$, it follows that $g(0,y) = 0$ if and only if $R = 0$ and $Q_s = 0$, and consequently $S = N$. We can conclude that the disease-free equilibrium $(0,0,\bar{y})$ is unique, thus (A5) holds. Denote

$$F = \frac{d\mathcal{F}}{dx}(0,0,\bar{y}) = \begin{bmatrix} \beta\rho & 0 \\ 0 & 0 \end{bmatrix}$$

$$V = \frac{d\mathcal{V}}{dx}(0,0,\bar{y}) = \begin{bmatrix} \frac{dQ_i}{dI}(0,0,\bar{y}) + \gamma & -\mu_i \\ \frac{dQ_i}{dI}(0,0,\bar{y}) & \mu_i + \gamma \end{bmatrix}$$

Then by direct computation we can show that the spectral radius of the next generation matrix $K = FV^{-1}$ is equal to $\mathcal{R}_0$ provided in 16. The statement of proposition then immediately follows from Theorem 1 by Van den Driessche and Watmough (2008).

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