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Research article

Controlling epidemic diseases based only on social distancing level: General case

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A B S T R A C T

The COVID-19 outbreak is an epidemic disease caused by the novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). When a new virus emerges, generally, little is known about it, and no vaccines or other pharmaceutical interventions are available. In the case of a person-to-person transmission virus with no vaccines or other pharmaceutical interventions, the only way to control the virus outbreak is by keeping a sustained physical distancing between the individuals. However, to adjust the level of the physical distancing accurately can be so complicated. Any level above the necessary can compromise the economic activity, and any level below can collapse the health care system. This work proposes a controller to keep the number of hospitalized individuals below a limit, and a new group-structured model to describe the COVID-19 outbreak. The proposed controller is robust to the uncertainties in the parameters of the model and keeps the number of infected individuals controlled only by adjusting the social distancing level. Numerical simulations, to show the behavior of the proposed controller and model, are done.

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1. Introduction

A novel coronavirus (SARS-CoV-2) emerged in the city of Wuhan in early December 2019 [1]. The SARS-CoV-2 causes the disease named coronavirus disease 2019 (COVID-19). The COVID-19 pandemic has the basic reproduction number relatively high and presents worrying hospitalization and death rates [2].

In literature, there are several works considering optimal control applied to epidemic diseases [3–6]. Recently, some works [7–11] investigated optimal control for the COVID-19 outbreak. Calvin Tsay et al. [7] reports a novel and complete dynamic optimization-based approach to the entire epidemiological modeling and outbreak control workflow for the US COVID-19 outbreak. Johannes Köhler et al. [8] investigates adaptive strategies to robustly and optimally control the COVID-19 pandemic via social distancing measures. Alex Perkins and Guido España [9] apply optimal control theory to determine optimal strategies for the implementation of non-pharmaceutical interventions to control COVID-19. In this work, the authors emphasize the importance of “careful estimation” of these parameters as the pandemic progresses. Markus Kantner et al. [10] investigates the optimal control to epidemics when an effective vaccine is impossible or never found, and the epidemic must be controlled with purely non-pharmaceutical measures. Alessandro Borri et al. [11], to optimally design the lock-down and reopening policies, proposes to minimize an aggregate cost function accounting for the number of individuals that decease due to the spread of COVID-19.

Still, based on optimal control strategy, we can highlight the works using predictive control [12–15]. Tamás Péni et al. [13] proposes a model predictive approach for constrained control of a nonlinear compartmental model that captures the key dynamical properties of COVID-19. Marcelo Morato et al. [14] formulates a Model Predictive Control (MPC) policy to mitigate the COVID-19 contagion in Brazil, designed as an optimal On–Off social isolation strategy. The authors, based on these uncertain models, propose an MPC-based control framework to determine in real-time whether to apply or not the social distancing policy. Marcelo Morato et al. [15] formulates a Nonlinear Model Predictive Control (NMPC) scheme to plan appropriate social distancing measures (and relaxations) to mitigate the effects of the COVID-19 pandemic. In this work, the authors propose a novel identification procedure composed of analytical regressions.

While optimal control of epidemiological models has been well-studied, a limiting factor for implementing modeling and optimization concepts is that obtaining accurate estimates of key model parameters can be challenging [7]. Optimal control theory provides a valuable tool to begin to assess the trade-offs between vaccination and treatment strategies [5]. As the optimal control, the model predictive control needs accurate estimates of
model parameters. However, the design of a state estimator for nonlinear systems is non-trivial [16].

Quarantine and isolation can control the COVID-19 outbreak because they are two measures by which exposed or infectious individuals are removed from the population to prevent further spread of the infection. During the SARS epidemic of 2002–2003 [3], some countries used quarantine.

In the work of Kiesha Prem et al. [1], the authors conclude that non-pharmaceutical interventions based on sustained physical distancing have a strong potential to reduce the magnitude of the epidemic peak of COVID-19 and lead to a smaller number of overall cases. According to Johannes Köhler et al. [8], social distancing is an effective way to contain the spread of a contagious disease, particularly when little is known about the virus and no vaccines or other pharmaceutical interventions are available.

However, removing individuals from the population needs an adequate criterion because to impose restrictions on the individuals can result in the reduction of economic activity.

Cameron Nowzari et al. [17] had a survey about the analysis and control of epidemics. In Nowzari work, authors highlight as main research challenges: “all control methods discussed so far have been for deterministic models”; “all control methods discussed so far have admitted centralized solutions”; “all control methods discussed so far have assumed there are no uncertainties”; “more general epidemic models are needed”.

The importance of research challenges highlighted by Cameron Nowzari is that centralized solutions, simple models to describe the epidemic diseases, and uncertainties in the parameters of these models can impose a vast restriction on the individuals and, in the majority of times, this restriction is higher than necessary. On the other hand, define this restriction below the adequate level can result in the collapse of the health system. In a realistic scenario of an epidemic disease, the available social and medical resources to treat diseases or to prevent their spreading are usually limited [18].

Person-to-person transmission is mostly driven by who interacts with whom, which can vary by age and location of the contact [1]. In the Prem work, the authors used an age-group model because the social mixing patterns vary by contexts, including households, workplaces, schools, and others [1]. Models that assess the effectiveness of physical distancing interventions, such as school closure, need to account for social structures and heterogeneities in the mixing of individuals [1,19].

Contributions. Based on the challenges shown and the characteristics of the COVID-19 outbreak, the focus of this work is to propose a decentralized control law, based on the adjustment of the social distancing level, to keep an epidemic outbreak that occurs in a country below a defined limit. Proposed control law can be applied to epidemic disease when there is no vaccination and treatment. Additionally, this work proposes a more general epidemic model. The new model will be group-structured to allow the incorporation of the social mixing patterns, and the novelty is their format.

The paper is structured as follows. The group-structured SIR model is proposed in Section 2 to describe an epidemic disease. In Section 3, the proposed controller, considering the uncertainties of the process, will be presented as a decentralized solution for the group-structured SIR model. Section 4 contains some numerical simulations of the theory, and Section 5 provides some concluding comments.

2. Group-structured SIR model

Kermack and McKendrick formulate the Susceptible–Infectious–Recovered (SIR) model in 1927. The SIR model describes the dynamics of epidemic diseases. The SIR model is adequate for epidemics having a relatively short duration (few months). The SIR model is modified to describe several situations. The variations of the SIR model is called the SIR-type models. There are SIR models to temporary immunity, with demography, with vaccination, with a latent period, age-structured, with time-varying parameters, vector-borne diseases, multi-regions, among others. The model presented in this work is to epidemic diseases with no treatment and vaccination.

When subdivides the population by characteristics other than those that are disease-related such as risk status or age, a further complication arises [20]. Nowadays, the conventional mathematical models of spread disease have usually ignored or overlooked the spatial dynamics [21].

This work proposes a group-structured SIR model to describe an epidemic disease in groups with different characteristics. The group can mean ages, regions, population density, and among others.

Let the proposed group-structured SIR model as

\[
\begin{aligned}
\frac{d s(t)}{dt} &= - (B(t) i(t)) \odot s(t) \\
\frac{d i(t)}{dt} &= (B(t) i(t)) \odot s(t) - y \odot i(t) \\
\frac{d r(t)}{dt} &= y \odot i(t)
\end{aligned}
\]

where \( B(t) \) is a matrix with the proportion coefficient of the disease transmission rate to each group, \( n \) is the number of the groups, \( s(t) \in \mathbb{R}^n \) is the vector with the number of susceptible individuals to each group, \( i(t) \in \mathbb{R}^n \) is the vector with the number of infected individuals to each group, \( r(t) \in \mathbb{R}^n \) is the vector with the number of recovered individuals to each group, and \( y \in \mathbb{R}^n \) is the vector of the recovery rate. The Hadamard operator \( \odot \) represents pointwise multiplication. Let \( A = (a_{ij}) \in \mathbb{R}^{n \times n} \) and \( P = (p_{ij}) \in \mathbb{R}^{n \times n} \), the Hadamard product of \( A \) and \( P \) is \( A \odot P = (a_{ij} p_{ij}) \).

The proportional coefficient of the disease transmission rate for each matrix element is

\[
\beta_{x,j}(t) = \frac{k_{x,j}(t)\gamma_j}{N_j(t)}, \quad z = 1, 2, \ldots, n, \quad j = 1, 2, \ldots, n
\]

where \( k_{x,j}(t) \) is proportional to the number of contacts that an infected individual has per unit time of each group, and \( N_j(t) \) is the number of individuals in each group.

The matrix with the basic reproductive number is \( R_0 \in \mathbb{R}^{n \times n} \) (see Appendix B for more details) and their elements, when \( z = j \), are

\[
R_{x,j} = \frac{k_{0,x,j} \gamma_j}{\gamma_j}, \quad z = j = 1, 2, \ldots, n
\]

and, when \( z \neq j \), they are

\[
R_{x,j} = \frac{k_{0,x,j} \gamma_j N_j}{\gamma_j N_j}, \quad z = 1, 2, \ldots, n, \quad j = 1, 2, \ldots, n
\]

where \( k_{0,x,j} \) represents the mean value of the number of contacts that an infected individual has per unit time of each group in normal conditions. \( \gamma_j \) represents the mean value of the probability that contact with a susceptible individual results in transmission.

The fundamental characteristics of the model (1) are:

- **C1.** the number of susceptible individuals of all elements of \( s(t) \) must be greater than zero;
C2. the number of infected individuals of at least one element of \( i(t) \) must be greater than zero;

C3. the value of the number of contacts that an infected individual has per unit time must be greater than zero (\( \kappa_{x,j}(t) > 0 \));

C4. the probability of the contact between a susceptible individual and an infected individual results in the transmission must be greater than zero (\( \tau(t) > 0 \));

C5. the recovery rate of all elements of \( \gamma \) is greater than zero;

The proposed model considers the different behaviors that can exist between groups. For example, to a big city, the groups can represent different ages, neighborhoods, and among others.

The proposed model is a better option than the standard SIR model to simulate an epidemic outbreak that occurs in big cities.

Although other models in the literature are capable of considering different behavior, the proposed model does this in compact form, which allows the expansion of the proposed model to several situations and simplifies the mathematical analysis about it.

According to Calvin Tsay et al. [7], more complex epidemiological models can, ultimately, help decision-makers to improve and optimize their policies to mitigate the spread of epidemics while keeping the toll on society and the economy low.

3. Proposed controller

Consider

\[
B(t) = \rho(t) \odot B_0,
\]

where \( \rho(t) \in \mathbb{R}^{n \times n} \) is the matrix with the control signal, \( B_0 \) is a constant matrix, and their elements are

\[
\beta_{x,j} = \frac{\gamma R_{x,j}}{N_j}, \quad z = 1, 2, \ldots, n, \quad j = 1, 2, \ldots, n,
\]

where the number of individuals in the each group \( N_j \) is considered a constant value.

In physical meaning, the element of the main diagonal of matrix \( B_0 \) represents the average disease transmission rate of that group. The out of main diagonal elements of matrix \( B_0 \) means the average disease transmission rate between the groups. The \( \rho(t) \) main diagonal represents the level of social distancing or other non-pharmaceutical measures to be applied to the population.

The purpose is to find a control law \( \rho(t) \), to the system (1) with the change of Eq. (6), such that each component of the output error vector

\[
e(t) = h_d - h(t) = [e_1(t) \ e_2(t) \ \cdots \ e_n(t)]^T,
\]

tends to zero when \( t \to \infty \), where \( h_d \in \mathbb{R}^n \) indicates the vector of the maximum desired infected individuals for each group.

\[
h(t) = H \cdot i(t)
\]

is the vector with the number of hospitalized infected individuals for each group, and \( H > 0 \) is proportional to the number of infected individuals that need hospitalization.

Assuming the following assumptions:

A1. the maximum desired infected individuals of each group are assumed to be a step greater than zero;

A2. the number of individuals in the population is assumed constant to each group;

Considering

\[
D_0 = 1_{n \times n} + (I \odot (B_0 - 1_{n \times n})),
\]

where \( 1_{n \times n} \in \mathbb{R}^{n \times n} \) is the matrix with all elements equals one and \( I \in \mathbb{R}^{n \times n} \) is the identity matrix.

Let

\[
\rho(t) = \text{diag}(\psi(t)) \odot (|s(t)|h^T(t) \odot B_0)^{(y-1)},
\]

where

\[
\psi(t) = [\psi_{a,1}(t) \ \psi_{a,2}(t) \ \cdots \ \psi_{a,n}(t)]^T,
\]

with

\[
\psi_{c,2} = \psi_{12} e(z) + \psi_{22} \int_0^t e(z) dt, \quad z = 1, 2, \ldots, n
\]

where \( \psi_{12}, \psi_{22} \) are nonnegative constants chosen to adjust the error dynamics of each group. \text{diag}(.) represents an operation that transforms a vector into a matrix with its main diagonal equals to the elements of the vector and the Hadamard inverse \((.)^{-1} \) represents pointwise inversion operation. Let \( A = (a_{ij}) \in \mathbb{R}^{n \times n} \), the Hadamard inverse of \( A \) is \( A^{(y-1)} = (a_{ij})^{-1} \).

Theorem 1. Consider the system of Eq. (1), the error Eq. (8), and the control law (11). Whenever all assumptions (A4)–(A5) are satisfied, the vector of errors \( e(t) \) will converge to zero when time tends to infinity.

Proof. The result of applying the control signal of Eq. (11) in the differential equation of \( i(t) \) of the system (1) is

\[
\frac{d \hat{i}(t)}{dt} = \frac{\psi(t)}{H} + \delta_b - \gamma \odot \hat{i}(t),
\]

which indicates that each element of \( \hat{i}(t) \) is independent and

\[
\delta_b = ((1_{n \times n} - I) \odot B_0) \cdot 1_{n \times 1},
\]

where \( \delta_b \) is constant.

The derivative of \( e(t) \) is

\[
\frac{d e(t)}{dt} = - \frac{d h(t)}{dt} = -H \frac{d \hat{i}(t)}{dt} = \psi(t) + H\delta_b - \gamma \odot H\hat{i}(t),
\]

where \( \hat{h}_d = 0 \), and the second derivative of \( e(t) \) is

\[
\ddot{e}(t) = \psi(t) - \gamma \odot \dot{e}(t) = -\psi_1 \odot \dot{e} - \psi_2 \odot e - \gamma \odot \dot{e}(t),
\]

where \( H\delta_b = 0 \).

Consider the Lyapunov function

\[
V(\dot{e}, e) = \frac{\dot{e}^T \dot{e}}{2} + \frac{(\psi_2 \odot \dot{e})^2}{2}
\]

which is possible because the elements of each vector \( \dot{e} \) and \( e \) are independents.

By applying (16) in (18), \( V(\dot{e}, e) \) results in

\[
V(\dot{e}, e) = -\psi_1 \odot \dot{e} - \psi_2 \odot e + \gamma \odot \dot{e}(t) + (\psi_2 \odot \dot{e})^T \dot{e}
\]

\[
= -\psi_1 \odot \dot{e} - \gamma \odot \dot{e}(t)^T \dot{e},
\]

where \( \psi_1 \) is the vector made by \( \psi_{12} \) elements to \( z = 1, 2, \ldots, n \). For the system to maintain \( V(\dot{e}, e) = 0 \) condition, the trajectory must be confined to line \( \dot{e} = 0 \). Using the system dynamics (8) yields:

\[
\dot{e} \equiv 0 \Rightarrow \ddot{e} \equiv 0 \Rightarrow -\psi_2 \odot e \equiv 0 \Rightarrow e \equiv 0,
\]

which according to LaSalle’s theorem the origin is globally asymptotically stable (GAS).

Now, consider that there are uncertainties \( \hat{B}_0 = (\Delta_2 \odot B_0) \) and \( \Delta_\gamma \) in the parameters \( B_0 \) and \( \gamma \), respectively, where each element
of \( \Delta_y \) and \( \Delta_y \) can assume any value between 0.5 and 1.5. The control law will update to
\[
\rho(t) = \text{diag}(\hat{\Psi}(t)) \odot (s(t)\hat{h}(t)\odot \hat{D}_0)^{\gamma(-1)},
\]
where \( \hat{D}_0 = I_{n \times n} + (I \odot (\hat{B}_0 - I_{n \times n})) \).

\[ \text{(20)} \]

Corollary 1.1. Consider the system of Eq. (1), the error Eq. (8), the control law (19), and that there are uncertainties in the matrix \( B_0 \) and the vector \( y \). Whenever all assumptions (A4)-(A5) are satisfied, the value of error \( e(t) \) will converge to zero when time tends to infinity.

\[ \text{Proof.} \] The result of applying the control signal of Eq. (19) in the differential equation of \( \hat{h}(t) \) of the system (1) is
\[
\frac{d\hat{h}(t)}{dt} = \delta_y \odot H^{-1} \hat{\Psi}(t) + \delta_y - (\Delta_y \odot y) \odot \hat{h}(t),
\]
which indicates that each element of \( \hat{h}(t) \) is independent and
\[
\delta_y = (I \odot (\hat{B}_0 - B_0))I_{n \times n}. \]

Consider the Lyapunov function
\[
V(\hat{e}, e) = \frac{\hat{e}^T \hat{e}}{2} + \frac{(\delta_y \odot \hat{\Psi}_2) \odot e)^T e}{2}.
\]

Then, the time derivative of \( V(\hat{e}, e) \) will be
\[
\dot{V}(\hat{e}, e) = \hat{e}^T \hat{e} + (\delta_y \odot \hat{\Psi}_2) \odot e)^T e \leq 0.
\]

\[ \text{(24)} \]

For the system to maintain \( V(\hat{e}, e) = 0 \) condition, the trajectory must be confined to line \( \hat{e} = 0 \). Using the system dynamics (8) yields:
\[
\hat{e} \equiv 0 \Rightarrow \hat{e} \equiv 0 \Rightarrow -(\delta_y \odot \hat{\Psi}_2) \odot e \equiv 0 \Rightarrow e \equiv 0,
\]
which by LaSalle’s theorem the origin is globally asymptotically stable (GAS). \( \square \)

To the real epidemic disease without treatment, the control law will update to
\[
\rho(t) = \max(0, \min(1, \text{diag}(\hat{\Psi}(t)) \odot (s(t)\hat{h}(t)\odot D_0)^{\gamma(-1)})).
\]

\[ \text{(26)} \]

Corollary 1.2. Consider the system of Eq. (1), the error Eq. (8), and the control law (26). Whenever all assumptions (A4)-(A5) are satisfied, the vector of errors \( e(t) \) will converge to a value greater than zero when time tends to infinity.

\[ \text{Proof.} \] Consider the system (1) when the control law (26) is applied
\[
\frac{d s(t)}{dt} = -(\rho(t) \odot B_0)\hat{h}(t) \odot s(t)
\]
\[
\frac{d \hat{h}(t)}{dt} = ((\rho(t) \odot B_0)\hat{h}(t) \odot s(t) - y \odot \hat{h}(t)).
\]

Based on the characteristics (C6-C10) of the system (1), consider the Lyapunov function
\[
V(s, i) = I_{n \times n}(s + i).
\]

Then, the time derivative of \( V(s, i) \) will be
\[
\dot{V}(s, i) = I_{n \times n}(-y \odot i).
\]

Therefore \( \dot{V}(s, i) \leq 0 \), which implies that the system is stable. Based on mathematical properties of the SIR model (see [3]) \( \lim_{t \to \infty} \hat{h}(t) = 0 \) (because the infected individuals of each group go to zero) and this implies \( e(t) \rightarrow i_d > 0 \) when \( t \to \infty \). \( \square \)

Remark. Always that the control signal is saturated, the computation of the integral term of Eq. (13) stop.

3.1. Control law using the estimates of \( h(t) \)

During epidemic disease outbreaks, the number of confirmed cases generally is the most reliable measurement. To epidemic disease, based on the group-structured SIR model (Eq. (1)), where all individuals of the population are susceptible, the dynamic of the number of confirmed cases is given by
\[
\frac{d s(t)}{dt} = (B(t)h(t)) \odot s(t).
\]

Based on Eq. (27), it is possible to consider
\[
s_c(t) = N_\text{t} - c(t) = z_1, z_2, \ldots, n.
\]

The estimates of \( \hat{h}(t) \) and \( \hat{h}(t) \) are
\[
\hat{h}(t) = (B(t))^{-1} \left( \frac{d s(t)}{dt} \odot s(t) \right) = (\rho(t) \odot B_0)^{-1} \left( \frac{d s(t)}{dt} \odot s(t) \right)_1
\]

\[ \text{(29)} \]

and
\[
\hat{h}(t) = H \cdot \hat{h}(t).
\]

\[ \text{(30)} \]

respectively.

4. Numerical simulations

The main idea behind using a group-structured SIR model was to develop a more general epidemic model for cases with groups that have different basic reproductive number \( R_0 \) between the groups. The groups can represent ages, demography, regions, and among others.

To the simulations of this work, the group-structured SIR model represents a fictitious state with three different zones with different population density. All zones have the same number of citizens. However, each zone has its basic reproductive number.

In this section, the proposed controller will be simulated and their results are presented and analyzed. Some important considerations about all simulations are that the numerical method for solving ordinary differential equations was the Euler method, the integration step was one day, all simulations have 365 days and the proportion of hospitalized individuals is 10% of infected individuals, which means \( H = 0.1 \). Other considerations are defined per group as the number of individuals in the population \( N_\text{t} \), the recovery rate \( \gamma [1,22,23] \), the initial condition of the susceptible individuals \( s(0) \), the initial condition of the infected individuals \( i(0) \), the initial condition of the recovered individual \( r(0) \), and the gains \( (\hat{\Psi}_1, \hat{\Psi}_2) \) of proposed controller (see table 1).

The value \( h_0 = 1800 \) is equivalent to 1.8 Hospital beds per 1000 population (value equals 72% of the total Hospital beds per 1000 population of the United Kingdom in 2019 according to OECD – Organization for Economic Co-Operation and Development). The uncertainties considered were +5% and -20% in the values of \( R_0 \) and \( \gamma \), respectively.

The matrix with basic reproductive number is
\[
R_0 = \begin{bmatrix}
1.4 & 0.0002 & 0.0002 \\
0.0002 & 1.9 & 0.0002 \\
0.0002 & 0.0002 & 2.0
\end{bmatrix}.
\]
The main diagonal represents the basic reproductive number \( R_0 \) between the individuals of the same group, and the other elements of the matrix represent the basic reproductive number between the individuals of the different groups. In this scenario, the values 1.4, 1.9, and 2.0 represent the basic reproductive number of groups 1, 2, and 3, respectively. The value 0.0002 represents the basic reproductive number between the individuals of the different groups. Although the proposed controller can operate with any positive value for the elements of the matrix \( R_0 \), the choice was small values to off-diagonal to represent three distinct regions with a low flow of citizens between them. This choice allows us to demonstrate an inappropriate situation to the standard SIR model.

This work presents simulations according to three scenarios. The first scenario (Fig. 1) shows a simulation where the proposed controller is not applied. The second scenario (Figs. 2, 4(a), and 5) presents a simulation using the proposed controller as a centralized controller to control all groups together. The third scenario (Figs. 3, 4(b), and 5) presents the result of three different simulations where the proposed controller is applied to control each group separately. Furthermore, there is a simulation with no uncertainties in the parameters and no estimator, there is a simulation with uncertainties and no estimator, and there is a simulation with uncertainties and estimator.

The simulation of Fig. 1 shows the behavior of the global number of infected individuals that will need health care without a controller to control the epidemic outbreak. In this simulation, it is possible to see that an epidemic disease without a controller can result in a large number of hospitalized individuals, and this occurs in a few days. Another important aspect is that the behavior of the global value of infected individuals from the group-structured model and the standard SIR model is different (just one peak). This difference occurs because the basic reproductive number is not homogeneous, and the epidemic disease begins in a specific group and spread out to other groups. Finally, the last aspect is that the accumulated number of infected individuals is nearly 70% of the global population.

The simulation of Fig. 2 shows the behaviors of the number of infected individuals that will need health care when the proposed controller is applied to adjust the social distancing level to all group together (centralized approach). In this simulation, it is possible to see that the number of hospitalized individuals keeps lower than the desired number of hospitalized individuals in each group. However, the number of hospitalized individuals per group exceeds the maximum value desired \( (h_d) \) for each group, which means that it is necessary to transport some infected individuals to another region to them receive medical care.

The simulation of Fig. 3 shows the behaviors of the number of infected individuals that will need health care when the proposed controller is applied to control each group separately (decentralized approach). In this simulation, it is possible to see that the social distancing of each situation presents different behavior even though the three groups, to both simulations, have the same population size.

The simulation of Fig. 5 shows the behavior of the accumulated number of infected individuals in three situations: without a controller, with the proposed controller applied to control all groups together and to control each group separately. Based on this simulation, it is possible to see that the proposed controller in any approach reduces the value of the accumulated number of infected individuals and that this number is smallest when the proposed controller is applied to control each group separately.

### 4.1. Discussion

The proposed controller calculates the social distancing level to keep the COVID-19 outbreak controlled and to guarantee the number of hospitalized individuals below the desired value per group during the outbreak. This technique can reduce the economic problems of social distancing and keeps the health care system working.

Another relevant result using the proposed controller in an epidemic outbreak is that the total number of infected individuals is less than the number without mitigation (see Fig. 5). If the total number of infected individuals decreases, this implies that the number of hospitalized and dead individuals will decrease.

Based on Fig. 5, it is possible to observe that the proposed controller applied to control each group separately reduces the
total number of infected individuals from 70% to 55%, when compared to the situation without a controller, and 62% to 55%, when compared to the situation with proposed controller applied to control all group together. Considering a population with 1 million individuals, this means a reduction of 150 and 70 thousand infected individuals for the first and second situations, respectively.

The value of the gains ($\psi_1$, $\psi_2$) of the proposed controller has the following logic: $\psi_1$ is related to how fast the error ($e(t)$) goes to nearly of zero, and $\psi_2$ is related to how softly the error ($e(t)$) converges to zero. This procedure is similar to adjust a PI controller to a first-order plant.

The quality of $\hat{h}(t)$ depends on the estimator technique and knowledge of the value of $B_0$.

The necessary time to finish the social distancing depends on the capacity of the health care system to attend the infected individuals.

To summarize, our key benefits are the following:

- We develop a control law with a simple adjustment. To each homogeneous group are designed just two parameters.

5. Conclusions

During the outbreak of COVID-19, all nations determined lockdown to huge areas, which imposes severe restrictions on the population and cause immense economic losses. They are using lockdown because not exist control strategies to mitigate the effects of the COVID-19 outbreak.

This work proposed a decentralized control law to keep the number of infected individuals during the COVID-19 outbreak below the desired number, and it does this only by adjusting the social distancing level. This control law is robust to uncertainties in the parameters, can be applied to a spreadsheet, and only need the measurements of traditional parameters of the pandemic outbreak. The proposed controller is simple and can be applied to help in the reopening of a region as a decentralized solution.

For example, to control an epidemic outbreak that occurs in a country, it just necessary one spreadsheet by county. The mayor of each county will keep the level of the social distancing that is necessary during the correct period calculated to their country.

This work also proposed a group-structured SIR model, which considers the heterogeneities in contact networks, to simulate the behavior of the COVID-19 outbreak. This model is a more general epidemic model.

Simulation results, to show as the control law works, were presented. In all simulations, the proposed control law reaches its objectives. The controller developed in this work was robust
Fig. 3. Simulation of the group-structured SIR model using the proposed controller to control each group separately (decentralized version), where the blue line is the number of hospitalized individuals. The dashed blue line is the number of hospitalized individuals when there are uncertainties in the parameters. The dotted blue line is the number of hospitalized individuals when there are uncertainties in the parameters, and the controller is using the estimator of $h(t)$. The orange dotted line is the desired number of hospitalized individuals, and the orange region indicates the range of $\pm 5\%$ of the $h_d$ value. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Fig. 4. Simulation of group-structured SIR model using the proposed controller, where the red line is the percent of the social distancing required. The dashed red line is the percent of the social distancing required when there are uncertainties in the parameters. The dotted red line is the percent of the social distancing required when there are uncertainties in the parameters, and the controller is using the estimator of $h(t)$.

A simulation, considering that only the accumulative number of infected individuals is measured, was done, and the proposed controller kept the number of infected individuals below the upper limit of the desired number of infected individuals.

To conclude, this work proposes solutions for three of the four main research challenges\(^2\) of the analysis and control of epidemics.

\(^2\) The four main research challenges, according to Nowzari [17].
Fig. 5. Simulations of the group-structured SIR model applied to three different situations. The dashed green line is the accumulated percent of infected individuals when there is no controller. The dotted green line is the accumulated percent of infected individuals when the proposed controller is applied to control all groups together. The green region represents the upper and lower accumulated percent of infected individuals when the proposed controller is applied to control each group separately. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

**Declaration of competing interest**

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

**Appendix A. The group-structured-SIR model mathematical properties**

Consider \( x \) the \( z \)th element of the vector \( x \) and

\[
\begin{align*}
    zs(0) &= 0 < z s_0 < N \\
z i(0) &= z i_0 \\
z r(0) &= z r_0, \quad z = 1, \ldots, n
\end{align*}
\]

the initial conditions of the vectors \( s, i, \) and \( r, \) respectively.

To the model (1), consider

\[
\frac{dzs}{dt}(t) = -\left( \sum_{j=1}^{n} \beta_{z,j} zi(t) \right) z s(t). \tag{A.2}
\]

Solving differential equation (A.2), the result is

\[
z s(t) = z s_0 \exp(\int_{0}^{t} \sum_{j=1}^{n} \beta_{z,j}^i d(\int_{0}^{z} s(w) \, dw)) \tag{A.3}
\]

which implies in

\[
z s_0 > 0 \implies z s(t) > 0, \forall t. \tag{A.4}
\]

To the model (1), consider

\[
\frac{dzi}{dt} = \left( \sum_{j=1}^{n} \beta_{z,j} zi(t) \right) \frac{dzs}{dt}(t) + (\beta_{z,z} z s(t) - z \gamma) z i(t). \tag{A.5}
\]

Solving differential equation (A.5), the result is

\[
z i(t) = z i_0 \exp(\int_{0}^{t} (\exp(k_2) \sum_{j=1}^{n} \beta_{z,j} zi(w)) \, dw), \tag{A.6}
\]

where

\[
k_1 = \int_{0}^{t} (\beta_{z,z} z s(w) - z \gamma) \, dw
\]

and

\[
k_2 = \int_{0}^{t} (\beta_{z,z} z s(w) - z \gamma) \, dw,
\]

which implies in

\[
z i_0 > 0 \implies z i(t) > 0, \forall t \implies z s(t) \leq 0, \forall t. \tag{A.7}
\]

To the model (1), consider

\[
z i(t) = z \gamma z s(t) = z \gamma z t(N - s(t) - r(t)). \tag{A.8}
\]

Solving differential equation (A.8), the result is

\[
z r(t) = z r_0 \exp(k_1) + \int_{0}^{t} \exp(k_2) (N - s(w)) \, dw,
\]

where

\[
k_1 = \int_{0}^{t} z \gamma \, dw
\]

and

\[
k_2 = \int_{0}^{t} z \gamma \, dw,
\]

which implies in

\[
z r_0 > 0 \implies z r(t) > 0, \forall t. \tag{A.9}
\]

Based on (A.4) and (A.7), the limit \( t \to \infty \) \( z s(t) \) is in

\[
0 < \lim_{t \to \infty} z s(t) < z s_0. \tag{A.10}
\]

Consider

\[
z s_\infty = \lim_{t \to \infty} z s(t). \tag{A.11}
\]

The integration of (A.2) results in

\[
\int_{0}^{\infty} z s(t) \, dt = -\int_{0}^{\infty} \left( \sum_{j=1}^{n} \beta_{z,j}^i z s(t) \right) z s(t) \, dt. \tag{A.12}
\]

Applying (A.11) in (A.12), the result is

\[
z s_\infty - z s_0 = -\int_{0}^{\infty} \left( \sum_{j=1}^{n} \beta_{z,j}^i z s(t) \right) z s(t) \, dt
\]

\[
z s_0 - z s_\infty = \int_{0}^{\infty} \left( \sum_{j=1}^{n} \beta_{z,j}^i z s(t) \right) z s(t) \, dt. \tag{A.13}
\]

The last inequality, considering (A.7), implies that

\[
\sum_{j=1}^{n} \beta_{z,j}^i z i(t) \tag{A.14}
\]

is integrable on \([0, \infty)\). Hence,

\[
\lim_{t \to \infty} z i(t) = 0. \tag{A.15}
\]

28
Appendix B. The basic reproduction number

In the Group-structured SIR model (1), an epidemic occurs if the number of infected individuals increases, i.e.,
\[ \max_{z \in \{1, \ldots, n\}} \frac{d i_z(t)}{dt} > 0. \]  
(B.1)

Thus, for the equation
\[ \frac{d i_z(t)}{dt} = (B(t)i(t)) \otimes s(t) - \gamma \otimes i(t), \]  
(B.2)
the equilibria are the solutions of the equation
\[ (B(t)i(t)) \otimes s(t) - \gamma \otimes i(t) = 0. \]  
(B.3)

According with the properties of the Group-structured SIR model Appendix A, Eq. (B.3) has two solutions:

1. when \( i(t) = 0_{1 \times n} \). This case is referred to as a disease-free equilibrium (the disease is not present in the population);
2. when \( i(t) \neq 0_{1 \times n} \). This case is referred to as an endemic equilibrium (the disease is present in the population).

Considering the following assumptions:

A3. the number of individuals in the population is assumed constant to each group, \( N_z \);
A4. the mean value of the number of contacts that an infected individual has per unit time of each group is assumed constant, \( k_{zj} \);
A5. the mean value of the probability that contact with a susceptible individual result in the transmission is assumed constant, \( \gamma_j \);
A6. the recovery rate is assumed constant to each group, \( \gamma_j \);
A7. each group has a well-mixed population.

Assuming A3–A7, we rewrite Eq. (B.3) as
\[ (B(t)i(t)) \otimes s(t) = \gamma \otimes i(t), \]  
(B.4)
and manipulating it in the appropriate way we have
\[ ((B(t)i(t)) \otimes s(t)) \otimes (\gamma \otimes i(t)) = 1_{n \times 1}, \]  
(B.5)
where
\[ \mathcal{R}_z = ((B(t)i(t)) \otimes s(t)) \otimes (\gamma \otimes i(t)). \]  
(B.6)

is the effective reproductive vector.

At the outset of an epidemic, nearly everyone (except the index case) is susceptible. So we can say that
\[ s_z(0) \approx N_z \quad \text{and} \quad i_z(0) \gg i_z(0), \quad \forall z \in \{1, \ldots, n\}. \]  
(B.7)

and
\[ i_z(0) \approx i_{z+1}(0), \quad \forall z \in \{1, \ldots, n-1\}. \]  
(B.8)

Applying (B.7) and (B.8) in (B.5), we have
\[ ((B \cdot 1_{n \times 1}) \otimes N) \otimes \gamma = 1_{n \times 1}, \]  
(B.9)
where
\[ \mathcal{R}_0 = ((B \cdot 1_{n \times 1}) \otimes N) \otimes \gamma \]  
(B.10)
is the basic reproductive vector.

We say that the disease-free equilibrium is globally stable when \( \|\mathcal{R}_0\|_{\infty} < 1 \) and we say that the endemic equilibrium is globally stable when \( \|\mathcal{R}_0\|_{\infty} > 1 \).

The matrix \( \mathcal{R}_0 \) with the basic reproductive number is
\[ \mathcal{R}_0 \cdot 1_{n \times 1} = \mathcal{R}_0 \]  
(B.11)
where their elements, when \( z = j \), are
\[ R_{z,j} = \frac{k_{0z,j}N_z}{\gamma_j}, \quad z = j = 1, 2, \ldots, n, \]  
(B.12)
and, when \( z \neq j \), they are
\[ R_{z,j} = \left( \frac{k_{0z,j}N_z}{\gamma_jN_j} \right), \quad z = 1, 2, \ldots, n, \quad j = 1, 2, \ldots, n. \]  
(B.13)

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