Systems and Control Approaches for Modeling the Coevolution of Epidemics and Behavioral Response: A COVID-19 Case Study

Systems and control approaches for modeling the coevolution of epidemics and behavioral response: A COVID-19 case study

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Abstract The COVID-19 health crisis has highlighted how the collective behavioral response of a population —for instance, the decision on whether to take up appropriate self-protective behaviors— plays a critical role in shaping the outcome of an epidemic outbreak. Here, we will briefly survey the seminal works and the state-of-the-art models proposed to capture and predict the complex and intertwined coevolutionary dynamics of epidemics and behavioral response, with a special focus on the methodological approaches proposed in the systems and control community. Then, we present and discuss a general framework to model such a coevolutionary dynamics and we illustrate its potentialities. Finally, we validate our modeling approach by presenting an implementation of the proposed framework tailored to capture the collective behavioral response to COVID-19, and we use such a parameterized model to discuss different what/if scenarios concerning the implementation of nonpharmaceutical interventions.

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1 Introduction

In the past few years, we have witnessed the importance of mathematical models of epidemic diseases. Indeed, developing accurate mathematical models for the spread of epidemic diseases enabled the scientific community to accurately predict the evolution of an epidemic outbreak, ultimately helping public authorities in their complex decisions during an epidemic emergency. The history of mathematical modeling of epidemics can be traced back to second half of the 18th century, with the the pioneering work of the Swiss mathematician Daniel Bernoulli on evaluating the effectiveness of inoculation of the smallpox virus using statistical tools [6]. A second, giant leap in the mathematical modeling of epidemics has been made at the beginning of the 20th century, when dynamical systems theory has started being applied to model the spread of epidemic diseases. In these regards, we have to mention the seminal work [40], who first proposed the use of compartmental models to predict the spread of epidemic diseases —i.e, the well-known susceptible–infected–recovered (SIR) and susceptible–infected–susceptible (SIS) model.

In the last century, several researchers have built on such seminal work, incorporating important features of real-world epidemic spreading into these mathematical framework. Among these advances, we mention some that are key, especially for the scope of this chapter. A first, key extension consists in embedding the compartmental models onto a network structure, to account for the heterogeneity in the pattern of social interactions through which the disease spreads [44, 50, 59]. Second, while in their original incarnation, epidemic models account only for individuals who are either susceptible to the disease, or infected, of recovered and thus either immunized or susceptible again, more complex models have been proposed toward better capturing the real-world progression of the disease (e.g., by including latency periods between contagion and development of symptoms, or different severity or symptomatic of the illness). This is the case, e.g., of the models developed in the past few years to capture the characteristics of COVID-19 [10, 30]. Third, several researchers have incorporated control actions within their mathematical models, including either nonpharmaceutical interventions (e.g., those acting on the network of social interactions) and pharmaceutical ones (e.g., distribution of drugs or vaccination), in order to gain model-based insights into the effectiveness of different interventions and design optimal intervention polices to mitigate or eradicate an epidemic outbreak [14, 32, 53, 66]. Also these approaches have been successfully adopted in the case of COVID-19 [12, 23, 43, 55, 56]. More details on epidemic models on networks and their control can be found in some recent review papers [50, 53, 58, 92].

These extensions of the original SIS and SIR frameworks have proven to be effective in accurately capturing important features of real-world epidemics and their relative intervention policies. However, there is a key aspect of epidemics that often has been overlooked in mathematical models of epidemic diseases: human behavioral response. In fact, most epidemic diseases spread via physical interactions between individuals (e.g., sexual encounters for sexually-transmitted diseases, or close-distance interactions for airborne diseases). Hence, to reduce the risk of transmission of the disease, humans may decide to change their behavior (e.g., by

using personal protection equipment, or avoiding close-distance interactions). Consequently, it is evident that human behavioral response to an epidemic outbreak has a key role in shaping the course of the outbreak, and neglecting such an aspect may lead to erroneous predictions, as observed during the COVID-19 pandemic [5, 79]. Similar, also the success of intervention policies is strongly impacted by humans' decisions, e.g, on whether to comply with the guidelines suggested or enforced by public authorities [42], or on whether to register for a vaccination campaign [81].

In this chapter, we aim to shed the light on this important aspect, by discussing the mathematical modeling of the coevolution of epidemics and behavioral response. Specifically, the main contribution is threefold. First, we provide a brief survey on the state-of-the-art approaches to incorporate the behavioral response into mathematical models of epidemic diseases. Specifically, after presenting the simplest implementations in which human behavior is implicitly incorporated within the model parameters, we introduce more sophisticated modeling approaches, which we shall call *behavioral–epidemic models*, in which human behavior is explicitly represented by a dynamical process that coevolves with the epidemic spreading. Second, we extensively present a mathematical framework, originally proposed for the simple cases of an SIS and SIR model in [87], which is able to capture the simultaneous spread of an epidemic disease and the corresponding behavioral response. In such framework, humans' decisions are modeled using a game-theoretic approach that can account for a range of factors that affect real-world behavioral responses over the whole course of an epidemic, i.e., perceived infection risk [64], social influence [77], accumulating fatigue and socio-economic costs [52], limited rationality in humans' decisions [76], and the implementation of policies and actions by public health authorities and governments [62]. Third, we demonstrate the validity of our framework in a case study, which is calibrated using publicly available data on COVID-19 in Italy. Through this model, we prove the effectiveness of the proposed approach in capturing the emergent behavior of the epidemic spreading, including the nonmonotonic evolution of the epidemic outbreak, which shows multiple waves. Then, we illustrate how the calibrated model can be used to investigate different what/if scenarios concerning the implementation of nonpharmaceutical interventions, toward determining their effectiveness accounting for the behavioral response of the population.

The rest of the chapter is organized as follows. In Section 2, we present some mathematical preliminaries. In Section 3, we survey the main existing approaches to behavioral–epidemics models. In Section 4, we present our general game-theoretic framework. In Section 5, we discuss the case study based on the COVID-19 pandemic in Italy. Section 6 concludes the chapter and outlines perspectives and directions for future research.

2 Mathematical Preliminaries

Before presenting behavioral–epidemics models, we need to introduce some preliminary notions on the mathematical formulation of compartmental models and on

network epidemic models. We start presenting compartmental models as a mathematical formulation used to describe how a disease evolve, both spontaneously and via interactions with other individuals. Then, we present a discrete-time stochastic formulation of network epidemic models, and we illustrate how to derive the equations associated with a specific compartmental model in the considered scenario.

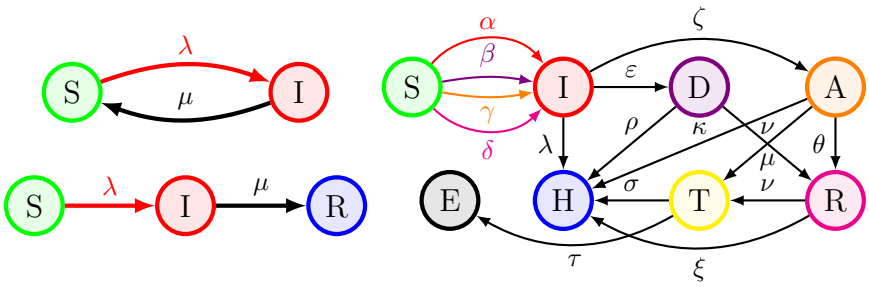
2.1 Compartmental models

Here, we present a general formulation of the compartmental models used in epidemiology to capture the progression of a disease. Specifically, compartmental models describe the possible health states that an individual can assume, and how such health states evolve either spontaneously or triggered by interactions with other individuals. Hence, compartmental models are characterized by the following three terms:

1. A (finite) set of *compartments* C, which represent all health states that an individual can have. These health states may include, e.g., being susceptible to the disease, being infected and infectious, or being recovered. Moreover, further states can be used to provide a detailed description of the state of the individual including, e.g., information on whether the individual is aware of being infected or on their behavior.
2. A matrix of *spontaneous transitions* $\mathbf{A} \in [0,1]^{C\times C}$, which describes how the health state of an individual changes spontaneously, due to the natural progression of the disease. In particular, its generic entry A_{ab} is the probability for individual who has health state $a \in C$ to transitions (spontaneously) to $b \in C$. Clearly, $A_{ab} = 0$ implies that no spontaneous transition from a to b can occur
3. A set of *interaction-driven transitions* $\mathbf{B^c} \in [0,1]^{C\times C}$, $c \in C$, which describes how the health state of an individual changes due to interactions with other individuals. In particular, its generic entry B^c_{ab} is equal to the probability at which an individual who has health state $a \in C$ transitions to $b \in C$, after an interaction with an individual with state $c \in C$.

A compartmental model can be conveniently represented by its *epidemic progression graph*, which is a multi-layer weighted graph with node set C and $|C| + 1$ layers. The first layer captures spontaneous transitions, with a link from $a \in C$ to $b \in C$ if and only if $A_{ab} > 0$ and weight equal to A_{ab}. The other layers capture interaction-driven transitions, with a link from $a \in C$ to $b \in C$ on layer $c \in C$ if and only if $B^c_{ab} > 0$ and weight equal to B_{ab}. We present here three illustrative examples.

Example 1 (SIS model) In the SIS model, individuals can either be susceptible to the disease or infected (and infectious). Consequently, the set of compartments is $C = \{S, I\}$. The health state of an individual evolves in a probabilistic fashion according to two contrasting mechanisms: susceptible individuals become infected after an interaction with an infected individual with probability $\lambda \in [0,1]$; while infected individuals spontaneously recover with probability $\mu \in [0,1]$, becoming



(a) SIS and SIR models

(b) SIDARTHE model

Fig. 1: Epidemic progression graphs for different epidemic models. Colors are used to represent the different layers: black is associated with spontaneous transitions, while edges on layer $a \in C$ are denoted with the color associated with compartment a.

again susceptible to the disease. Consequently, the only nonzero entries of the transition matrices are $A_{IS} = \mu$ and $B^I_{SI} = \lambda$. A visual representation of the SIS model by means of the epidemic progression graph is reported in Fig. 1a (above).

Example 2 (SIR model) The SIR model is a variation of the SIS model in which individuals become permanently immune after recovery. To account for such phenomenon, an additional health state is included for recovered individuals, i.e., $C = \{S, I, R\}$, and the only nonzero entries of the spontaneous transition matrix becomes $A_{IR} = \mu$. A visual representation of the SIR model by means of the epidemic progression graph is reported in Fig. 1a (below).

Example 3 (SIDARTHE model) The SIDARTHE model, proposed in [30] for COVID-19, is an extension of the SIR model in which the compartment for infected individuals is refined into five different compartments (I, D, A, R, T), to account for different stages of the disease, with not all of them being infectious (namely, treated individuals are isolated and thus not infectious). Despite the complexity of transition matrices due to the presence of many health states and transitions, the epidemic progression graph still offers an immediate visual interpretation of the model, as one can observe in Fig. 1b.

2.2 Network epidemic models

We present a discrete-time stochastic formulation of network epidemic models [8]. For the sake of simplicity, we will present such mathematical paradigm using the simple SIS model, which will be adopted in the next chapter as baseline for our mathematical framework.

We consider a (possibly time-varying) network $\mathcal{G}(t) = (\mathcal{V}, \mathcal{E}(t))$, where the node set $\mathcal{V} = \{1, \dots, n\}$ represents the n individuals of the population and the (possibly time-varying) edge set $\mathcal{E}(t) \subseteq \mathcal{V} \times \mathcal{V}$ represents physical interactions between individuals at discrete time $t \in \mathbb{Z}_+$: the presence of an (undirected) link $(i, j) \in \mathcal{E}(t)$ means that i and j have a physical interaction at time t, i.e., that the infectious disease could potentially spread from i to j or vice versa. The discrete time unit $t \in \mathbb{Z}_+$ is the time resolution of the model. Its choice may depend on the specific characteristics of the disease under exam and on the temporal granularity of available data that are used to calibrate the model, which can typically vary from one day to one week. For instance, in the case study considered in this chapter, we set the time resolution equal to 1 day, consistent with the temporal granularity of available data [67].

Each individual $i \in \mathcal{V}$ is characterized by a health state $x_i(t)$ in a set of compartments $\mathcal{C}$ (see Section 2.1), i.e., $x_i(t) \in \mathcal{C}$. Such health state evolves in time according to the spontaneous and interaction-driven transitions defined in the corresponding compartmental model. For instance, in the case of the SIS and SIR models, $x_i(t)$ evolves according to the two contrasting mechanisms of contagion and (spontaneous) recovery described in Examples 1 and 2, yielding the dynamical system described by the following equations.

Example 4 (Network SIS model) In a network SIS model, each individual $i \in \mathcal{V}$ can be either susceptible ($x_i(t) = S$) or infected ($x_i(t) = I$) at discrete time-step t. Transmission occurs for each interaction with an infected individual with probability λ (independent of others). Consequently, if we denote by

$$N_i(t) := \big|\{j \in \mathcal{V} : (i, j) \in \mathcal{E}(t),\, x_j(t) = I\}\big| \tag{1}$$

the number of infected individuals with whom i interacts at time t, we have that each of such $N_i(t)$ interaction can transmit the disease to individual i with probability $B^I_{SI} = \lambda$, independent of the others. Consequently, the probability that i becomes infected at the following time-step is derived using elementary probability theory tools, obtaining the nonlinear term:

$$\mathbb{P}[x_i(t+1) = I \,|\, x_i(t) = S] = 1 - (1 - \lambda)^{N_i(t)}. \tag{2}$$

Besides the contagion, at each time-step t, every infected individual i recovers with probability $A_{IS} = \mu \in (0, 1]$, independent of the others and of the past, becoming susceptible again to the disease. Consequently, we obtain

$$\mathbb{P}[x_i(t+1) = S \,|\, x_i(t) = I] = \mu. \tag{3}$$

Example 5 (Network SIR model) In a network SIR model, each individual $i \in \mathcal{V}$ can have health state $x_i(t) \in \{S, I, R\}$. Contagion follows the same mechanism as in the SIS model, yielding the same Eq. (2). Concerning recovery, instead, recovered individuals transition to state R, yielding

$$\mathbb{P}[x_i(t+1) = R \,|\, x_i(t) = I] = \mu. \tag{4}$$

Following a similar approach, one can derive the equations associated with any compartmental model, including complex epidemic progressions like the one of the SIDARTHE model presented in Example 3.

Typically, it is assumed that the network $\mathcal{G}(t)$ is generated according to a Markov process. This encompasses the simpler case of a time-invariant networks [50, 53], as well as many models of time-varying networks such as the temporal-switching [78], activity-driven [61, 89, 90], and edge-markovian models [17]. Under this assumption, the whole process determined by Eq. (2), Eq. (3), and the possible network formation process is a Markov chain, which can be analytically studied. The emergent behavior of this epidemic process is characterized by two possible regimes: either the epidemic outbreak is quickly eradicated (reaching the disease-free equilibrium in which all individuals are susceptible in a time that grows only logaritmically in the population size) or the disease remains endemic in the population for a long time (precisely, for a time that grows exponentially large with the population size). Such emergent behavior is fully determined by the parameters —the infection probability λ and the recovery probability μ— and the network $\mathcal{G}(t)$. For more details, see [53, 92].

In the above, we discussed the implementation of an SIS model on a network. Similar, any compartmental model defined by means of an epidemic progression graph (see Section 2.1) can be embedded onto a (fixed or time-varying) network structure and, under the same markovianity assumptions on the network formation process, one can guarantee that the overall network epidemic model obtained yields an analytically treatable Markov chain.

3 Survey on Behavioral–Epidemics Models

In this section, we briefly review the state-of-the-art approaches that have been proposed in the literature to incorporate the behavioral response into mathematical models of epidemic diseases. We start our survey by discussing classical models, in which the behavioral response is implicitly incorporated within time-varying parameters of the model. The simplicity of such approach allows for an easier treatment, but it fails to capture the complexity of real-world behavioral responses. To address such limitation, more complex models that involve an explicit modeling of human behavior have been proposed. The common idea is to expand the epidemic model by adding additional compartments or co-evolving dynamics to account for the evolution of human behavior. Here, we present some of the main mathematical approaches used to capture such coevolution, which will be categorized into two main classes: i) reactive responses driven by the spread of information or awareness of the disease, and ii) rational responses, in which individuals actively take decisions on their behavior accounting for several factors. For the second class, we discuss some different mathematical approaches that have been proposed to model rational decision-making, including opinion dynamics and game-theoretic models.

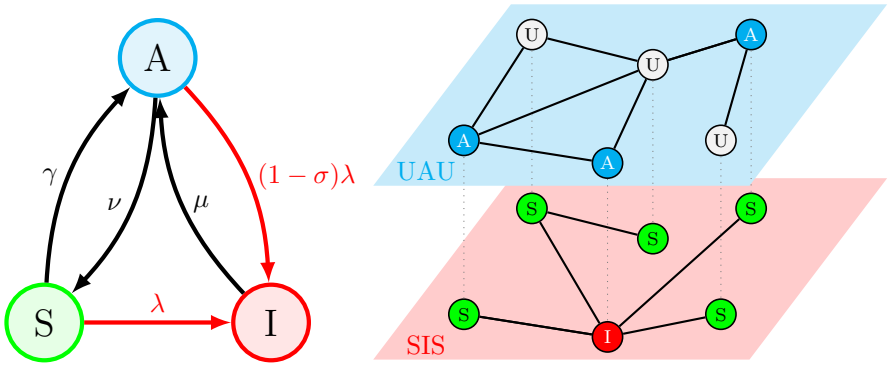
3.1 Implicit modeling of human behavior

Classical network epidemic models, however, do not explicitly account for the behavioral response of the population and its impact on the epidemic spreading. A first approach that has been used to account for such response is to assume that the infection and recovery parameters are time varying, i.e., λ and μ are substituted by $\lambda(t)$ and $\mu(t)$, and they may vary across the population. Of particular interest is the case in which such parameters (in particular, the contagion rate) are defined as feedback functions of the spreading process, either measured at the global or at the local level [26, 63]. This allows to capture the impact of policies and law-enforced actions, e.g., total or partial lockdown policies when the number of infection trespasses a certain threshold. This modeling approach was originally proposed in [11], with the inclusion of a saturation function in the infection parameter. Expanding on this methodology, several approaches have been proposed to implicitly incorporate human behavior into the term $\lambda(t)$. See, e.g., [2, 27], where such approach has been implemented on an SIS and an SIR model, respectively. Similarly, information-induced behavioral changes have been modeled implicitly by defining time-varying transition rates through some auxiliary variables [24]. These methods have been successfully used in real-world scenarios, e.g., to study the role of citizen compliance with mitigation measures during the COVID-19 pandemic [9]. Moreover, other researchers proposed the use of adaptive time-varying network, in which individuals who are infected reduce (or completely avoid) having interactions with others [70].

Despite useful to capture some features of behavioral responses, the lack of an explicit modeling of human behavior hinders the possibility for such approaches to fully capture the real-world complexity. In fact, these approaches rely on the basis assumption that it is known a priori how the behavioral response shapes the epidemic spreading (either as a function of time or of the spread). However, in the real-world, this is rarely the case, since the behavioral response is driven by complex human decision-making, which cannot be straightforwardly modeled through a direct correspondence between human behavior and law-enforced prescriptions. In fact, different social, psychological, and economical factors may modulate the adoption of laws and regulations, yielding to complex, bounded-rational, behavior [79]. Such complexity calls for the explicit modeling of human behavior as a variable of the system, as we shall see in the rest of this section.

3.2 Awareness-driven models

Awareness-driven models rely on the assumption that the behavioral response is purely reactive to the spread of information —or awareness— on the epidemic outbreak. The first implementation of such a paradigm is the susceptible–alert–infected–susceptible (SAIS) model, originally proposed originally proposed by Sahneh et al. in [19], and further extended and studied in [65, 71, 91]. This model relies on the inclusion of an additional compartment to account for individuals who are aware of



(a) SAIS model (b) UAU-SIS model

Fig. 2: Schematic illustrating the two main approaches for awareness-driven modeling of behavioral responses.

the disease and alert, i.e., $C = \{S, A, I\}$. At each time step, susceptible individuals become alerted with a spontaneous transition probability $A_{SA} = \gamma \in [0, 1]$, which can be interpreted as the effectiveness of awareness campaigns and intervention policies (and thus a possible control input of the system, as in [91]). Individuals who are alert adopt self-protections to reduce transmission, thus their infection probability is reduced by a parameter $\sigma \in [0, 1]$. However, alert individuals may spontaneously stop adopting such behaviors, becoming again fully susceptible; this occur with probability $A_{AS} = \nu \in [0, 1]$ at each time step. These transitions are illustrated in the epidemic progression graph in Fig. 2a. The transition probabilities of the network SAIS model are obtained similar to Eq. (2) and Eq. (3), yielding:

$$\begin{aligned}
&\mathbb{P}[x_i(t+1) = I \,|\, x_i(t) = S] = 1 - (1-\lambda)^{N_i(t)} \\
&\mathbb{P}[x_i(t+1) = I \,|\, x_i(t) = A] = (1-\sigma)\big(1 - (1-\lambda)^{N_i(t)}\big), \\
&\mathbb{P}[x_i(t+1) = A \,|\, x_i(t) = I] = \mu, \\
&\mathbb{P}[x_i(t+1) = S \,|\, x_i(t) = A] = \nu, \\
&\mathbb{P}[x_i(t+1) = A \,|\, x_i(t) = S] = \gamma.
\end{aligned} \tag{5}$$

Building on a the same assumptions, Granell et al. proposed the aware–unaware–aware SIS (UAU-SIS) model to investigate the competing spreading of awareness and the epidemic dynamics [33,34]. The main difference with the SAIS model is that, here, the spread of awareness is captured by an additional variable $y_i(t) \in \{U, A\}$, that captures whether individual i at time t is unaware of the epidemics ($y_i(t) = U$) or if i is aware and thus adopts self-protections to reduce transmission ($y_i(t) = A$), which is captured by reducing the infection probability by a factor $\sigma \in [0, 1]$, similar to the SAIS model. Hence, the state of individual i at time t is captured by a two-dimensional variable $z_i(t) = (x_i(t), y_i(t))$. In its original incarnation, it was assumed that the disease is symptomatic, and thus infected individuals are aware of

the disease. In such a scenario, the variable $z_i(t)$ can have three possible states:

$$z_i(t) = \begin{cases} (S,U), \text{ if } i \text{ is susceptible and unaware of the disease,} \\ (S,A), \text{ if } i \text{ is susceptible and aware of the disease,} \\ (I,A), \text{ if } i \text{ is infected and aware of the disease.} \end{cases} \tag{6}$$

Otherwise, an additional fourth state $z_i(t) = (I,U)$ can be included to represent individuals who are infected by the disease but unaware of its existence.

Such modeling approach allows for including an explicit dynamics of the spreading of information/awareness. In fact, unlike the SAIS model, where the transition to the "alert" state is independent of others and fully determined by the transition probability γ, in the UAU-SIS model, individuals can become aware either due to infection (i.e., if they transition to $x_i = I$ then they also transition to $y_i = A$), or as a consequence of social interactions with aware individuals on a a social network (with a mechanism similar to the contagion), which may in general differ from the physical contact network through which the disease spread. Such a distinction between social and physical interactions can be captured by a bilayer network $\mathcal{G}(t) = (\mathcal{V}, \mathcal{E}_C(t), \mathcal{E}_I(t))$, where $\mathcal{E}_C(t)$ and $\mathcal{E}_I(t)$ are two (possibly different and possibly time-varying) layers representing physical contacts and social interactions, respectively. See, e.g., the schematic illustrated in Fig. 2b. Hence, the nonzero transition probabilities of the two-dimensional variable $z_i(t) = (x_i(t), y_i(t))$ are:

$$\begin{aligned} &\mathbb{P}[z_i(t+1) = (I,A) \,|\, z_i(t) = (S,A)] = (1-\sigma)\big(1-(1-\lambda)^{N_i(t)}\big), \\ &\mathbb{P}[z_i(t+1) = (I,A) \,|\, z_i(t) = (S,U)] = 1-(1-\lambda)^{N_i(t)}, \\ &\mathbb{P}[z_i(t+1) = (S,A) \,|\, z_i(t) = (S,U)] = (1-\lambda)^{N_i(t)}\big(1-(1-\gamma)^{\tilde{N}_i(t)}\big), \\ &\mathbb{P}[z_i(t+1) = (S,A) \,|\, z_i(t) = (I,A)] = \mu, \end{aligned} \tag{7}$$

where $N_i(t) := |\{j : (i,j) \in \mathcal{E}_C(t),\, x_j(t) = I\}|$ is the number of physical interactions that i has at time t with infected individuals, $\tilde{N}_i(t) := |\{j : (i,j) \in \mathcal{E}_I(t),\, y_j(t) = A\}|$ is the number of social interactions that i has at time t with individuals who are aware, and $\gamma \in [0,1]$ is the "social contagion" probability. Additionally, one could add a forgetting process, through which individuals who are susceptible and aware spontaneously returns to the unaware state, similar to the transition from A to S in the SAIS model.

While awareness-driven models have demonstrated effectiveness in capturing the immediate behavioral response to an epidemic outbreak, allowing researchers to establish theoretical results on how the spreading of awareness shapes the epidemic outbreak [80, 82], their have some critical limitations. In fact, by assuming fully rational, purely instantaneous, and reactive decision-making in the population, they neglect all the other factors that affect real-world behavioral responses over the entire course of an epidemic, i.e., once the entire population is aware of the disease. The behavioral responses observed during the COVID-19 pandemic and their heterogeneity across different countries is indeed a clear example that awareness is not the only key factor in shaping human behavior during the entire course of a pandemic [42], calling for the development of more refined modeling approaches.

3.3 Model based on opinion dynamics

Mathematical opinion dynamics models have been developed from the second part of the last century [20] to represent and study opinion formation processes in social networks. Such modeling paradigm relies on the assumption that the opinion of an individual on a specific topic is influenced by those shared by others on a social network. Such influence is typically assumed to act on individuals' opinion in a linear fashion, yielding linear averaging dynamics, which are amenable to analytical treatment. See [3,68] for a detailed review on seminal and recent advances on opinion dynamics.

Within the framework of behavioral–epidemic models, opinion dynamics have been recently adopted to model the behavioral response during an epidemic outbreak. Specifically, similar to awareness-driven model, an additional behavioral variable $y_i(t)$ is incorporated in the model. However, such variable is typically assumed to take a continuous value (typically in the range $y_i(t) \in [0, 1]$) that captures the opinion of individual i on the severity of the disease. Such variable evolves according to some classical opinion dynamics models, whereby individuals revise their opinion by averaging their current opinion with the ones of their neighbors on a social interaction network, possibly under the influence of some external factors and of some characteristics of the individual (e.g., in this application, their health status) [3, 68]. Then, individuals adopt self-protections proportionally to their opinion, thereby reducing their infection probability similar to the UAU-SIS model, but in a continuous fashion, so that the infection probability of an individual with opinion $y_i(t)$ is re-scaled by multiplying the term in Eq. (2) by the factor $1 - \sigma y_i(t)$, with $\sigma \in [0, 1]$ modeling the effectiveness of self-protections, ultimately obtaining

$$\mathbb{P}[x_i(t+1) = I \,|\, x_i(t) = S] = \big(1 - \sigma y_i(t)\big)\big(1 - (1-\lambda)^{N_i(t)}\big). \tag{8}$$

Such modeling approach, adopted in [7,60,85], allows to capture in an analytically-tractable model the key role of social influence in shaping the behavioral response [77], and can be extended to account, e.g., for the presence of cooperative and antagonistic interactions in the social network [75]. See, [74] for more details. However, besides just focusing on social influence, this approach rely on another critical assumption, i.e., that the behavioral response is fundamentally a linear phenomenon. However, it is worth of mentioning that one of the main difficulty that scientists have always found in modeling and predicting human behavior is in its inherent nonlinearity [37]. Therefore, despite indeed useful to shed light into the impact of social influence, the assumption that the entire behavioral response is the result of linear dynamics could be too restrictive to have an accurate representation of human behavior during epidemic spreading.

3.4 Game-theoretic models

With the goal of overcoming this limitation, game-theoretic models have recently emerged as a powerful tool to capture the complexity and nonlinearity of human decision-making in many applications [88], supported by empirically-validated studies [86]. Within behavioral–epidemic models, the first application of game-theoretic decision-making concerns the behavioral response to voluntary vaccination campaigns. In their seminal paper, Bauch et al. [4] proposed to model the individuals' decision on whether to take part in the vaccination campaign or not using a game-theoretic formalism. Specifically, it is assumed that each individual can choose between two possible actions (to join the campaign or not), captured by a binary variable $y_i(t) \in \{0, 1\}$. Each individual's decision is taken in a (fully or partially) rational way, by comparing two payoff functions that quantifies the (perceived) risk associated with vaccination and with infection, respectively. The latter is clearly function of i) the level of herd immunity of the population already reached (due to natural or vaccine-induced immunization) and ii) the number of infected individuals in the population. Hence, it ultimately depends on the decisions of other individuals, consistent with the game-theoretic formulation [88]. Then, the infection probability of an individual is re-scaled, according to their decision, using Eq. (8). Such modeling approach has been extended to incorporate additional features, e.g., bounded rationality in human decisions, imperfect vaccines, and different time-scales of vaccination and contagion [16, 21, 29], and has been validated on real-world case studies [84].

Recently, the use of a game-theoretic formalism has been extended to model not only the behavioral response to a vaccination campaign, but also the one to the an epidemic spreading. In such a framework, it is assumed that an individual $i \in \mathcal{V}$ decides whether to adopt self-protective measures or not as the result of a (possibly bounded) rational decision-making process, based on comparing two payoff functions, associated with adopting or not adopting protections. Such formalism is extremely flexible, as it allows to incorporate within the two payoff functions all the different factors that may influence the behavioral response. Specifically, besides (perceived) infection risk, awareness, and social influence (already considered in the approaches presented in the above), one can also include in the payoff functions, e.g., the effect accumulating fatigue and socio-economic costs, limited rationality in humans' decisions, and the role of interventions.

Several researchers have recently adopted such a modeling formalism to explore different aspects of the complex interplay between the epidemic spreading and its behavioral response, by conveniently defined the payoff functions. In [35], the payoff functions focus on the contrasts between the possible risk of becoming infected and the social and economic impact associated with avoiding close-distance interactions. Extensions of this work accounts for more complex decision-making processes and epidemic progression models [47, 73]. In [49], the payoff functions are defined to investigate the optimal design of intervention policies. In [87], further terms are added to the payoff function to account for social influence and accumulating fatigue. In [25], a more complex decision is considered, which accounts not only

for activation, but also on migration, depending on the state of the epidemics. Interestingly, different studies have proved that such a complex framework lead to a rich and nontrivial range of emergent behaviors in the epidemic spreading, with possibly bifurcation phenomena, with the emergence of multiple waves and periodic outbreaks [28, 41, 46, 54], consistent with real-world observations. For more details on the use of game-theory in epidemic models, we will refer to some recent review papers [15, 36].

4 Our Mathematical Framework

In this section, we present a general formulation of the game-theoretic behavioral–epidemic framework we originally proposed in [87]. In its original incarnation, the framework was proposed for the SIS and SIR models. Here, we extend it to more general compartmental models, such as those that capture the epidemic progression of COVID-19.

Similar to a UAU-SIS model, we consider a population of n individuals, who interacts on a bilayer network $\mathcal{G}(t) = (\mathcal{V}, \mathcal{E}_C(t), \mathcal{E}_I(t))$, where $\mathcal{E}_C(t)$ is the time-varying edge set of physical contacts at time t (through which the disease is transmitted) and $\mathcal{E}_I(t)$ is a (possibly time-varying) edge set of social interactions at time t, which capture social influence between individuals.

Each individual $i \in \mathcal{V}$ is characterized by a two-dimensional state variable: the health state $x_i(t) \in \mathcal{S}$, where $\mathcal{S}$ is the (finite) set of possible health states (see Section 2.1), and the behavior $y_i(t) \in \{0, 1\}$, which captures whether individual i adopts self-protections at time t ($y_i(t) = 1$) or not ($y_i(t) = 0$). The health state and the behavior are updated at each time step synchronously, as detailed in the following and illustrated in Fig. 3.

4.1 Health state update

We consider a generic compartmental model (see Section 2.1), and we introduce a parameter $\sigma \in [0, 1]$, which captures the efficacy of the adoption of self-self-protective behaviors in reducing the risk of contagion. Specifically, $\sigma = 0$ means that protections have no impact on contagion, while $\sigma = 1$ models the ideal scenario where protections are 100% effective in preventing contagion. Then, if an individual $i \in \mathcal{V}$ adopts self-protections at time t (i.e., $y_i(t) = 1$), then all the transition probabilities associated with a contagion for individual i are reduced by the multiplicative factor σ. This can be captured mathematically by multiplying all the transition probabilities associated with a contagion by a factor $(1 - \sigma y_i(t))$. For instance, in the network SIS model presented in Section 2.2, the infection probability at time t in Eq. (2) is replaced by Eq. (8).

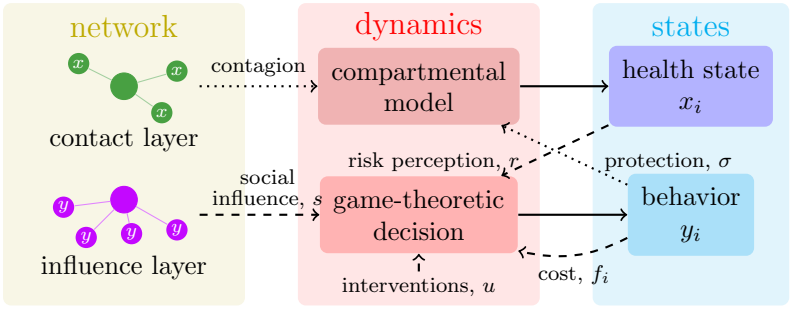


Fig. 3: Schematic of the coevolutionary framework presented in Section 4.

4.2 Behavior update

Individuals' behavioral update is modeled via a game-theoretic formalism. Specifically, we introduce two payoff functions associated with the (perceived) reward that individual i would receive for adopting each possible action (i.e., either adopt or not adopt self-protective measures), which are defined as the sum of four different contributions described below, obtaining

$$\pi_i^0(t) := s_i^0(\mathbf{y}(t)) + f_i(t), \quad \pi_i^1(t) := s_i^1(\mathbf{y}(t)) + r(\mathbf{x}(t)) + u(t). \tag{9}$$

The four different contributions capture different factors that affect individual's behavioral response during the spread of an epidemic disease and their impact is supported by empirical studies, as well as theoretical research in the social-psychology literature. Specifically, the contributions are the following.

Social influence. The first terms in both payoffs in Eq. (9) capture social influence of neighboring individuals and individual i's desire to coordinate with them on the behavioral response [77]. A classical implementation of such term, grounded on network coordination games [39], is to set

$$s_i^a(t) := \frac{1}{d_i(t)} \left| \{ j : (i,j) \in \mathcal{E}_I(t), y_j(t) = a \} \right|, \tag{10}$$

for $a \in \{0,1\}$, where $d_i(t) := |\{j : (i,j) \in \mathcal{E}_I(t)\}|$ is the degree of node i on the influence layer at time t, whereby an increased payoff is provided for conforming to the behavior adopted by the majority of individual i's neighbors.

Cost of self-protective behavior. Adopting self-protections has clearly an immediate social, psychological, and economic cost related, e.g., to the inability to socialize, work from the office, enjoy public spaces. Moreover, fatigue, stress, and economic losses tend to accumulate over time, exacerbating the contribution, as observed in several studies during the COVID-19 pandemic [52, 69]. These aspects are captured by the function $f_i(t)$, which can be defined as the sum of a fixed term $c \geq 0$ associated with the immediate cost for adopting self-protections, and an

accumulation term, which is discounted by a factor $\gamma \in [0, 1]$, obtaining

$$f_i(t) = c + \sum_{s=1}^{t} \gamma^s c y_i(t-s); \tag{11}$$

the larger the γ, the higher the impact of past decisions on the payoff.

Risk perception. The term $r(\mathbf{x}(t))$ accounts for risk perception, capturing an incentive to adopt self-protective behavior in response to the fear of becoming infected as the disease spreads. This term is inspired by awareness-based models. To model such phenomenon, we introduce the detected prevalence $z(t) = z(\mathbf{x}(t))$ as the fraction of detected infections at time t (in a simple scenario for an SIS/SIR model, one can simply assume $z(t) = \frac{1}{n}|\{i \in \mathcal{V} : x_i(t) = I\}|$), and we assume that the risk-perception depends on $\mathbf{x}(t)$ through $z(t)$, and that $r(z) : [0, 1] \to \mathbb{R}_{\geq 0}$ is monotonically nondecreasing in z. The simplest scenario, which will be used in Section 5, is to assume the risk perception function linear in the detected prevalence, i.e.,

$$r(z(t)) := \kappa z(t). \tag{12}$$

Policy interventions. The term $u(t)$ is a function that captures the interventions enforced by public authorities at time t to encourage people adopt self-protective behaviors. The value of such parameter is proportional to the severity of the policies implemented, and can vary in time, as public authorities may change their policies during the course of the epidemic outbreak. From the perspective of public health authorities and governments, the function $u(t)$ is the input that they can use to control human behavior and, ultimately, the spread of the epidemic disease, since behavior and epidemics are coupled by Eq. (8). This will be discussed for the COVID-19 case study in our numerical simulations in Section 5.3.

At each time-step, each individual i decides whether to revise their behavior $y_i(t) \in \{0, 1\}$ using an evolutionary game-theoretic revision protocol [72]. Such protocols typically account for the nonlinearity of human behavior and for individuals' bounded rationality in decision making. For instance, they can adopt a logit learning [72], whereby individual i will adopt self-protective behaviors at the following time-step with a probability equal to

$$\mathbb{P}[y_i(t+1) = 1] = \frac{\exp\{\beta_i \pi_i^1(t)\}}{\exp\{\beta \pi_i^0(t)\} + \exp\{\beta_i \pi_i^1(t)\}}, \tag{13}$$

and not adopt them otherwise. Here, the parameter $\beta_i \in [0, \infty)$ measures an individual's *rationality* in the decision-making process; the larger is β, the higher the probability that the individual chooses the behavior that maximizes their perceived reward. Other revision protocols, such as imitation rules can be used as well (see, e.g., the theoretical results obtained with a mean-field approach for an SIS model developed within our framework [28], or the kinetic-equation approach in [22]). Note that the stochasticity and nonlinearity of Eq. (13), combined with the nonlinearity

of Eq. (8) implies that the policy interventions $u(t)$ may have ultimately a nontrivial and nonlinear impact on the emergent behavior of the system.

In [87], some preliminary simulations of our game-theoretic epidemic–behavioral model were performed, in order to illustrate the wide range of possible emergent behaviors that our model is able to reproduce, and the impact of the behavioral parameters on the epidemic spreading. In particular, by coupling the model with simple SIS and SIR compartmental models, the model were able to reproduce complex realistic epidemic patterns, including recurrent outbreaks and multiple waves. In the next section, we present a case study, in which we tailor our mathematical framework to reproduce the spread of COVID-19 in Italy, and we use such a case study to validate the proposed model, and to illustrate how this novel framework can be used to help assist public health authorities, investigating different what/if scenarios for the implementation of intervention policies.

5 Case Study: COVID-19 in Italy

In this section, we present and discuss a case study, in which we adopt the epidemic-behavioral mathematical framework proposed in Section 4 to reproduce the first wave of the COVID-19 epidemic outbreak in Italy and to illustrate how such mathematical framework, after being tailored to a specific scenario and calibrated using real-world data, can be used to test different intervention policies, toward help assist public health authorities in their decisions.

5.1 COVID-19 Model

We start by introducing the compartmental model we use to capture the spread of COVID-19. For the sake of simplicity, we decided to adopt a parsimonious SIRD model, where $C = \{S, I, R, D\}$, representing susceptible, infected, recovered, and dead individuals. The dynamics is built from the SIR model presented in Example 2, assuming that a fraction $\delta \in [0, 1]$ of the infected individuals die instead of recovering, and that immunity is not permanent, and wanes with probability ψ at each time step. Hence, the three nonzero entries of the spontaneous transition matrix are $A_{IR} = \mu(1-\delta)$, $A_{ID} = \mu\delta$, and $A_{RS} = \psi$. A schematic of the epidemic progression is illustrated in Fig. 4a. Hence, the equations that govern the epidemic dynamics comprise Eq. (8) for the contagion process, coupled with the following equations associated with spontaneous transitions:

$$\begin{aligned} &\mathbb{P}[x_i(t+1) = R \,|\, x_i(t) = I] = \mu(1-\delta), \\ &\mathbb{P}[x_i(t+1) = D \,|\, x_i(t) = I] = \mu\delta, \\ &\mathbb{P}[x_i(t+1) = S \,|\, x_i(t) = R] = \psi. \end{aligned} \tag{14}$$

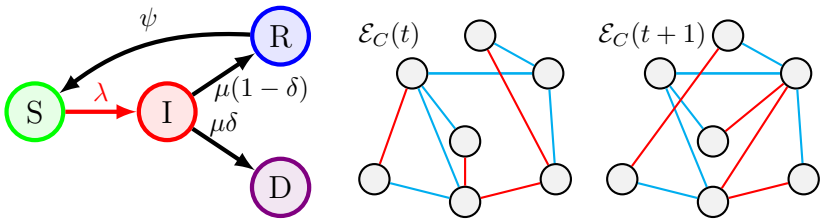
Importantly, a key element of the payoff functions that determine the decision-making process in the behavior update is the risk perception, which depends on the detected prevalence $z(t)$. In the case of COVID-19, it is not reasonable to assume that all cases are detected, due to the high prevalence of asymptotic cases [13]. Moreover, especially in the early phases of the outbreak, information about the number of cases was subject to delays due to the time needed to process swabs [13]. Consistent with these observations, we introduced two parameters: the detection rate $\nu \in [0, 1]$ and the detection delay $\tau \in \mathbb{Z}_+$, representing respectively the fraction of infected individuals that are actually detected and the number of time-units of delay between infection and reporting. Hence, we write

$$z(t) := \frac{1}{n} \cdot \nu \cdot |\{i \in \mathcal{V} : x_i(t - \tau) = I\}| . \tag{15}$$

Notice that an alternative to the introduction of these two parameters can be the use of a more complex compartmental model, such as the SIDARTHE model in Example 3. However, such an increased in complexity would have introduced more than just two additional parameters to the model. Hence, we preferred to keep the compartmental model a simple as possible and to define the detected prevalence $z(t)$ using Eq. (15).

In the modeling framework, the population $\mathcal{V}$ is connected through a bilayer time-varying network $\mathcal{G} = (\mathcal{V}, \mathcal{E}_I(t), \mathcal{E}_C(t))$, where $\mathcal{E}_I(t)$ is the social influence layer and $\mathcal{E}_C(t)$ the contact layer through which the disease is transmitted. Here, we assume that the influence layer is fixed during the time-window of interest (few months, during the first wave of COVID-19 in Italy). Towards representing real-world social interaction networks as faithfully as possible, we generate such a layer as a Watts–Strogatz small-world network [83] with average degree 10 and rewiring probability 0.15, consistently with empirical observations on real-world social networks [51]. Note that the network is undirected, since it makes sense to assume that social influence is a mutual phenomenon, so that if a first individual is influenced by the behavior of a second, also the second is affected by the behavior of the first one.

Then, we set the time-unit to be equal to one day, and we generate the time-varying layer of social contact $\mathcal{E}_C(t)$ as follows. According to the empirical study in [51], almost half of the physical contacts one individual has during a day are with family members, close friends, and colleagues (i.e., the neighbors on the influence layer $\mathcal{E}_I$). The remaining contacts are temporary and unusual interactions, for instance, those occurring at the supermarket, in the elevator, or in public transport. Consistently, at each time-step $t \in \mathbb{Z}_+$, we generate the layer $\mathcal{E}_C(t)$ by adding to the influence layer $\mathcal{E}_I$ a set of (temporary) randomly generated links, which we generate using an algorithm inspired by activity-driven networks [61,89]. Specifically, for each individual $i \in \mathcal{V}$, we generate five undirected links by sampling a 5-uple of nodes uniformly at random in the network and generates undirected links from i to each node belonging to this 5-uple. At the following time step, $t + 1$, links generated at time t are removed, and a new set of links is sampled to be added to $\mathcal{E}_I$, generating $\mathcal{E}_C(t + 1)$. In such a way, we generate a time-varying layer $\mathcal{E}_C(t)$ whose average degree is equal to 20



(a) SIRD model (b) Time-varying contact layer

Fig. 4: Specialization of the modeling framework to COVID-19: (a) Epidemic progression graphs and (b) two consecutive time instances of the contact layer $\mathcal{E}_C(t)$ and $\mathcal{E}_C(t+1)$; links belonging to the influence layer $\mathcal{E}_I$ (present in all time instances) are in cyan, temporary links are in red.

(on average, 10 links come from the influence layer, 10 are generated at random), consistent with [51], where it is empirically observed that an heterogeneous sample of Italian people results to have on average 19.77 contacts per day. An illustration of the network formation process is offered in Fig. 4b.

5.2 Parameter Identification

Now, we discuss how we use empirical data to identify the parameters of the epidemic-behavioral model. Specifically, we set some parameters, using available data, and we identify the remaining ones. The parameters set from available data are the effectiveness of self-protections, set to $\sigma = 0.5$, people's rationality, set to $\beta = 6$ following [87], reporting rate $\nu = 0.07$ and reporting delay $\tau = 14$ days, based on empirical studies for the first COVID-19 epidemic wave in Italy [13], and we neglect waning immunity ($\psi = 0$) since the time-horizon used in the simulations ($T = 100$ days) is within the average duration of natural immunity [31]. Additionally, we set the policy interventions, which remained mostly constant during the initial lockdown phase and were then partially uplifted at the end of the simulation horizon, to the value $u(t) = 0.5$. The identified parameters are thus the infection probability λ, the recovery probability μ, the death probability δ, the self-protective behavior cost c, the scaling factor of the accumulation term γ, and the risk-perception parameter κ. For all these parameters, we define a tuning range in terms of a minimal and maximal value, consistent with the literature and with common sense. The tuning ranges used are reported in Table 1.

To identify these parameter, we re-scale the Italian population of approximately 58, 851 million individuals [38] to $n = 1,000$ agents, in order to perform Monte Carlo simulations, which are required due to the inherent stochasticity of the epidemic model. Considering the tuning ranges for the six unknown parameters, we generate a 6-dimensional grid $\mathcal{T}$. For each point of the grid $\mathbf{p} = (\lambda, \mu, \delta, c, \gamma, \kappa)$, representing

a configuration of the six parameters, we generate $M = 200$ independent runs of the epidemic–behavioral model using that set of parameters, obtaining a Monte Carlo estimation of the number of cases and deaths over time $\hat{I}_{\mathbf{p}}(t)$ and $\hat{D}_{\mathbf{p}}(t)$, by averaging the outcomes of the simulations over the M independent runs. Such quantities are compared with the real Italian data from February 24, 2020, for $T = 100$ days, using the officially reported cases from [67]. Specifically, we compute the SSE (sum of square errors) between the simulated curves $\hat{I}_{\mathbf{p}}(t)$ and $\hat{D}_{\mathbf{p}}(t)$ and the real one $I(t)$ and $D(t)$ in order to optimize its tuning parameters. Data are adjusted to account for delay and limited detection, similar to the expression in Eq. (15), using reporting rate $\nu = 0.07$ and reporting delay $\tau = 14$ days [13]. In summary, the optimal set of parameters is determined as

$$\hat{\mathbf{p}} := \text{argmin}_{p \in \mathcal{T}} \sum_{t=1}^{T} \left(I(t) - \hat{I}_{\mathbf{p}}(t)\right)^2 + \sum_{t=1}^{T} \left(D(t) - \hat{D}_{\mathbf{p}}(t)\right)^2 . \tag{16}$$

Once the optimal set of parameters $\hat{\mathbf{p}}$ is found by solving Eq. (16), the procedure is re-iterated with denser 6-dimensional grids in the neighborhood of the optimal node, in order to refine the estimate while keeping a reasonable computational complexity of the method —in fact, the entire identification procedure entails the evaluation of approximately 5,000 different parameter configurations and takes approximately 83 hours on a laptop with a 4-core 10th Generation Intel i7-10510U processor. Following such a procedure, we obtain the optimal value of the parameters as: $\lambda = 0.007$, $\mu = 0.14$, $\delta = 0.003$, $c = 0.5$, $\gamma = 0.25$, and $k = 12$, as reported in Table 1. The average gap between the data and the fitted model is of 140 active infected individuals and 2.3 deaths, over the entire Italian population of 58, 851 million individuals.

parameter	symbol	initial tuning range	identified value
per-contact infection probability	λ	$[0.001, 0.1]$	0.007
recovery probability	μ	$[0.01, 0.5]$	0.14
death probability	δ	$[0.001, 0.1]$	0.003
immediate cost	c	$[0, 3]$	0.5
accumulation factor	γ	$[0, 1]$	0.25
risk perception	κ	$[0, 20]$	12

Table 1: Summary of the model parameters identified from epidemiological data: tuning ranges and identified values.

5.3 Simulations

In this section, we illustrate how the epidemic-behavioral model, once the parameters are calibrated to reproduce a real-world scenario, can be used in order to assess the

effectiveness of different intervention policies. Specifically, intervention policies are captured by the time-varying function $u(t)$, which represents the intensity of the policies enacted at time step t. Here, we investigate different what/if scenarios towards optimally defining the control function $u(t)$ to decrease the total number of infections and deaths. Specifically, our primary goal is to reduce the epidemic's peak, easing the strain on overwhelmed healthcare systems.

We start by considering constant policies, i.e., policies in which we set

$$u(t) = u_h, \tag{17}$$

for all $t \geq 0$, and we test different values of the constant u_h. Simulation results are illustrated in Fig. 5, with our quantitative findings summarized in Table 2 (averaged over 50 independent runs). Specifically, our simulations show how the control policy $u(t) = u_h$ affects the fraction of population adopting self-protective measures (average behavior $Y_{\text{avg}} := \frac{1}{nT} \sum_{t=1}^{T} \sum_{i \in \mathcal{V}} y_i(t) = 1$) and, ultimately, the average and peak fraction of infections (let $I(t) := \frac{1}{n} |\{i \in \mathcal{V} : x_i(t) = \text{I}\}|$, we define $I_{\text{avg}} := \frac{1}{T} \sum_{t=1}^{T} I(t)$ and $I_{\text{max}} := \max_{t \in [0,T]} I(t)$, respectively) and on the total deaths ($D_{\text{tot}} := \frac{1}{n} |\{i \in \mathcal{V} : x_i(T) = \text{D}\}|$).

u_h	Y_{avg} (avg behavior)	I_{avg} (avg infections)	I_{max} (epidemic peak)	D_{tot} (total deaths)
0.45	0.3059	0.0186	0.0270	9.2×10^{-4}
0.50	0.4182	0.0169	0.0249	8.2×10^{-4}
0.55	0.4951	0.0155	0.0230	6.8×10^{-4}
0.60	0.5973	0.0141	0.0210	6.6×10^{-4}
0.65	0.6750	0.0132	0.0203	6.2×10^{-4}
0.70	0.7762	0.0123	0.0176	6.4×10^{-4}
0.75	0.8215	0.0119	0.0172	5.2×10^{-4}
0.80	0.8651	0.0112	0.0133	6.2×10^{-4}
0.85	0.9035	0.0110	0.0134	3.2×10^{-4}
0.90	0.9250	0.0109	0.0126	4.8×10^{-4}

Table 2: Outcome of the Monte Carlo simulations with different (costant) values of the control action $u(t) = u_h$.

Predictably, increasing u_h reduces the fraction of infected individuals, as can be observed in Fig. 5a. However, we observe that such impact decreases in magnitude as u_h increases, becoming eventually marginal beyond $u(t) = 0.8$. Thus, we conclude that $u(t) = 0.8$ (representing a 60% increase in NPI intensity with respect to the baseline case used in the parameter identification) is a reasonable choice for enacting severe measures, which effectively flattens the epidemic curve without resorting to unnecessary economic-damaging lockdown.

However, maintaining such high value of control for an extended period may still pose challenges for the population mental health and the national economy, potentially yielding limited benefits. To investigate whether such severe policies may be uplifted, mirroring real-world strategies, we propose to reduce the strength

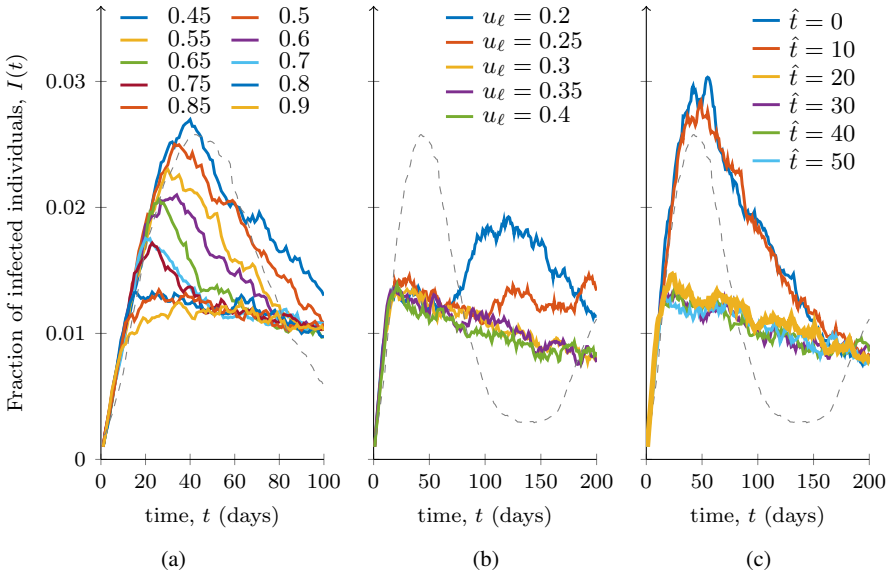


Fig. 5: Simulations of the what/if scenarios with the epidemic-behavioral model calibrated to COVID-19. In (a), we consider a constant control policy $u(t) = u_h$ with different values of u_h. In (b,c), we consider a piece-wise constant policy of the form Eq. (18), with (b) different values of u_ℓ and (c) different changing points $\hat{t}$. In all the plots, the real infection curve is reported using a gray dashed curve.

of interventions after a specific date $\tilde{t}$, defining $u(t)$ as a piecewise constant function

$$u(t) = \begin{cases} 0.8, & t \le \tilde{t}, \\ u_\ell, & t > \tilde{t}. \end{cases} \tag{18}$$

The goal now is to find the best values for u_ℓ and $\tilde{t}$. To perform such study, we test different values in the range $\tilde{t} \in [0, 100]$ and $u_\ell \in [0, 0.8)$ in a Monte Carlo fashion, extending the numerical simulations up to $T = 200$ days to assess the short- and medium-term impact of such change of control action. The results of our numerical study, reported in Fig. 5 illustrates the epidemic curves obtained fixing one of the two parameters and changing the other. Specifically, Fig. 5b suggest that decreasing u_ℓ below 0.3 would not be sufficient to prevent a steady increase in the number of cases and a second epidemic wave. On the other hand, increasing u_ℓ above the value 0.3 has a negligible positive impact on the course of the outbreak. In Fig. 5c, instead, we observe that setting the changing point to $\tilde{t} = 20$ would be optimal, since a shorter duration of the severe lockdown seems not sufficient to curb the epidemic curve (blue curve), while longer a duration seems not beneficial. Our conclusion is that it seems that the optimal solution could have been to implement sliglty more

severe policies during the first three weeks and then, at $\tilde{t} = 20$ partially uplift them, setting $u_\ell = 0.3$. Such a strategy is able to reduce the peak by more than 50% and the average number of infections by more than 40% with respect to the real one, keeping then the figures under control even when the policies are partially uplifted, as one can see from the thicker yellow curve in Fig. 5c.

6 Conclusion and Perspectives

In this chapter, we have first briefly surveyed the recent developments in the mathematical approaches to realistically reproduce the spread of epidemic diseases in structured populations, accounting for human behavioral response to the epidemics. Building on these efforts, we have built a game-theoretic modeling framework to accurately predict the complex interplay between epidemic spreading and behavioral response during an epidemic outbreak. The proposed framework incorporates many factors that play a key role in shaping human behavior, such as risk sensitivity, social pressure, accumulation of frustration, and the implementation of intervention policies, as empirically observed in the social psychology literature. Then, we have tested our framework on a real-world case study. Specifically, we have demonstrated its effectiveness by illustrating how the model, tailored to COVID-19, is able to reproduce the 2020 first wave of COVID-19 in Italy, and we have illustrated how such model, calibrated with real-world data, can be used by public health authorities to explore the effectiveness of different intervention policies.

The growing interest of the scientific community in mathematical models of epidemic spreading that are able to account for human behavioral response paves the way for several future lines of research. First, efforts should be placed in expanding the theoretical treatment of the model presented in this chapter, in particular toward developing comprehensive techniques to identify model parameters, estimate the exact state of the system, and study the design of optimal intervention policies, by employing control-theoretic methods, expanding the tools developed for classical epidemic models [1, 57], along the lines, e.g., of [53, 92]. In fact, our behavioral-epidemic model allows us to predict the complex behavioral response determined by the implementation of a specific control action (i.e, a specific choice of the function $u(t)$) by utilizing a game-theoretic framework. Hence, the growing theory of control of game-theoretic dynamics and the new methodologies developed in that field [18, 45, 48, 93] could be leveraged in order to develop new control-theoretic methods for the control of epidemic outbreaks in realistic settings.

Second, inspired by the work in Section 5, further empirical studies should be performed, not only to validate the different epidemic-behavioral frameworks, but also to establish some rigorous methodological routines to identify model parameters, also utilizing available data on human behavior, e.g., tracking the evolution of human mobility. Third, the lesson learned in mathematical epidemiology on the key role than human decision-making plays in shaping the emergent behavior of epidemic outbreak should lay the foundation for a novel holistic approach to mathematical

modeling for complex socio-technical systems, where human behavior could play a key role but has often been neglected, being the case, for instance, of energy systems.

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References

1. Alamo, T., G. Reina, D., Millán Gata, P., Preciado, V.M., Giordano, G.: Data-driven methods for present and future pandemics: Monitoring, modelling and managing. Annu. Rev. Control **52**, 448–464 (2021). https://doi.org/10.1016/j.arcontrol.2021.05.003
2. Alutto, M., Como, G., Fagnani, F.: On SIR epidemic models with feedback-controlled interactions and network effects. In: 60th IEEE Conf. Decis. Control. pp. 5562–5567 (2021). https://doi.org/10.1109/CDC45484.2021.9683007
3. Anderson, B.D.O., Ye, M.: Recent Advances in the Modelling and Analysis of Opinion dynamics on Influence Networks. Int. J. Autom. Comput. **16**(2), 129–149 (2019). https://doi.org/10.1007/s11633-019-1169-8
4. Bauch, C.T., Earn, D.J.D.: Vaccination and the theory of games. Proc. Natl. Acad. Sci. USA **101**(36), 13391–13394 (2004). https://doi.org/10.1073/pnas.0403823101
5. Bedson, J., et al.: A review and agenda for integrated disease models including social and behavioural factors. Nat. Hum. Behav. **5**(7), 834–846 (2021). https://doi.org/10.1038/s41562-021-01136-2
6. Bernoulli, D.: Essai d'une nouvelle analyse de la mortalité causée par la petite vérole et des avantages de l'inoculation pour la prévenir. Memoirs of the Royal Mathematical and Physical Academy of Sciences pp. 1–45 (1760)
7. Bhowmick, S., Panja, S.: Analysis of epidemic spreading with opinion evolution in multiplex network. IEEE Trans. Circuits Syst. II Express Briefs **70**(2), 695–699 (2023). https://doi.org/10.1109/TCSII.2022.3212268
8. Brauer, F., Castillo-Chavez, C.: Mathematical Models in Population Biology and Epidemiology. Springer, New York NY, US (2011)
9. Buonomo, B., Della Marca, R.: Effects of information-induced behavioural changes during the COVID-19 lockdowns: the case of Italy. R. Soc. Open Sci. **7**(10), 201635 (2020). https://doi.org/10.1098/rsos.201635
10. Calafiore, G.C., Novara, C., Possieri, C.: A time-varying SIRD model for the COVID-19 contagion in Italy. Annu. Rev. Control **50**, 361–372 (2020). https://doi.org/10.1016/j.arcontrol.2020.10.005
11. Capasso, V., Serio, G.: A generalization of the kermack-mckendrick deterministic epidemic model. Math. Biosci. **42**(1–2), 43–61 (1978). https://doi.org/10.1016/0025-5564(78)90006-8
12. Carli, R., Cavone, G., Epicoco, N., Scarabaggio, P., Dotoli, M.: Model predictive control to mitigate the COVID-19 outbreak in a multi-region scenario. Annu. Rev. Control **50**, 373 – 393 (2020). https://doi.org/10.1016/j.arcontrol.2020.09.005
13. Català, M., Pino, D., Marchena, M., Palacios, P., Urdiales, T., Cardona, P.J., Alonso, S., López-Codina, D., Prats, C., Alvarez-Lacalle, E.: Robust estimation of diagnostic rate and real incidence of COVID-19 for european policymakers. PLOS ONE **16**(1), 1–26 (2021). https://doi.org/10.1371/journal.pone.0243701
14. Cenedese, C., Zino, L., Cucuzzella, M., Cao, M.: Optimal policy design to mitigate epidemics on networks using an SIS model. In: 60nd IEEE Conf. Decis. Control. IEEE (2021). https://doi.org/10.1109/cdc45484.2021.9683737
15. Chang, S.L., Piraveenan, M., Pattison, P., Prokopenko, M.: Game theoretic modelling of infectious disease dynamics and intervention methods: a review. J. Biol. Dyn. **14**(1), 57–89 (2020). https://doi.org/10.1080/17513758.2020.1720322

16. Chen, X., Fu, F.: Imperfect vaccine and hysteresis. Proc. Royal Soc. B **286**(1894), 20182406 (2019). https://doi.org/10.1098/rspb.2018.2406
17. Clementi, A.E.F., Macci, C., Monti, A., Pasquale, F., Silvestri, R.: Flooding Time of Edge-Markovian Evolving Graphs. SIAM J. Discrete Math. **24**(4), 1694–1712 (2010). https://doi.org/10.1137/090756053
18. Como, G., Durand, S., Fagnani, F.: Optimal targeting in super-modular games. IEEE Trans. Autom. Control. **67**(12), 6366–6380 (2022). https://doi.org/10.1109/TAC.2021.3129733
19. Darabi Sahneh, F., Scoglio, C.: Epidemic spread in human networks. In: 51st IEEE Conf. Decis. Control. pp. 3008–3013 (2011). https://doi.org/10.1109/CDC.2011.6161529
20. DeGroot, M.H.: Reaching a Consensus. J. Am. Stat. Assoc. **69**(345), 118–121 (1974). https://doi.org/10.2307/2285509
21. Della Marca, R., d'Onofrio, A., Sensi, M., Sottile, S.: A geometric analysis of the impact of large but finite switching rates on vaccination evolutionary games. Nonlinear Anal. Real World Appl. **75**, 103986 (2024). https://doi.org/10.1016/j.nonrwa.2023.103986
22. Della Marca, R., Loy, N., Menale, M.: Intransigent vs. volatile opinions in a kinetic epidemic model with imitation game dynamics. Math. Med. Biol. **40**(2), 111–140 (2022). https://doi.org/10.1093/imammb/dqac018
23. Della Rossa, F., Salzano, D., Di Meglio, A., De Lellis, F., Coraggio, M., Calabrese, C., Guarino, A., Cardona-Rivera, R., De Lellis, P., Liuzza, D., Lo Iudice, F., Russo, G., di Bernardo, M.: A network model of Italy shows that intermittent regional strategies can alleviate the COVID-19 epidemic. Nat. Commun. **11**(1) (2020). https://doi.org/10.1038/s41467-020-18827-5
24. d'Onofrio, A., Manfredi, P., Salinelli, E.: Vaccinating behaviour, information, and the dynamics of SIR vaccine preventable diseases. Theor. Popul. Biol. **71**(3), 301–317 (2007). https://doi.org/10.1016/j.tpb.2007.01.001
25. Elokda, E., Bolognani, S., Hota, A.R.: A dynamic population model of strategic interaction and migration under epidemic risk. In: 60th IEEE Conf. Decis. Control. pp. 2085–2091 (2021). https://doi.org/10.1109/CDC45484.2021.9683739
26. Fenichel, E.P., Castillo-Chavez, C., Ceddia, M.G., Chowell, G., Parra, P.A.G., Hickling, G.J., Holloway, G., Horan, R., Morin, B., Perrings, C., Springborn, M., Velazquez, L., Villalobos, C.: Adaptive human behavior in epidemiological models. Proc. Natl. Acad. Sci. USA **108**(15), 6306–6311 (2011). https://doi.org/10.1073/pnas.1011250108
27. Franco, E.: A feedback SIR (fSIR) model highlights advantages and limitations of infection-dependent mitigation strategies. ArXiv preprint: 2004.13216 (2020). https://doi.org/10.48550/arXiv.2004.13216
28. Frieswijk, K., Zino, L., Ye, M., Rizzo, A., Cao, M.: A mean-field analysis of a network behavioral–epidemic model. IEEE Control Syst. Lett. **6**, 2533–2538 (2022). https://doi.org/10.1109/LCSYS.2022.3168260
29. Fu, F., Rosenbloom, D.I., Wang, L., Nowak, M.A.: Imitation dynamics of vaccination behaviour on social networks. Proc. Royal Soc. B **278**(1702), 42–49 (2011). https://doi.org/10.1098/rspb.2010.1107
30. Giordano, G., Blanchini, F., Bruno, R., Colaneri, P., Di Filippo, A., Di Matteo, A., Colaneri, M.: Modelling the COVID-19 epidemic and implementation of population-wide interventions in Italy. Nat. Med. **26**(6), 855–860 (2020). https://doi.org/10.1038/s41591-020-0883-7
31. Goldberg, Y., Mandel, M., Bar-On, Y.M., Bodenheimer, O., Freedman, L.S., Ash, N., Alroy-Preis, S., Huppert, A., Milo, R.: Protection and waning of natural and hybrid immunity to SARS-CoV-2. N. Engl. J. Med. **386**(23), 2201–2212 (2022). https://doi.org/10.1056/NEJMoa2118946
32. Gracy, S., Pare, P.E., Sandberg, H., Johansson, K.H.: Analysis and distributed control of periodic epidemic processes. IEEE Trans. Control. Netw. Syst. **8**(1), 123–134 (2021). https://doi.org/10.1109/TCNS.2020.3017717
33. Granell, C., Gómez, S., Arenas, A.: Dynamical interplay between awareness and epidemic spreading in multiplex networks. Phys. Rev. Lett. **111**(12), 128701 (2013). https://doi.org/10.1103/PhysRevLett.111.128701
34. Granell, C., Gómez, S., Arenas, A.: Competing spreading processes on multiplex networks: Awareness and epidemics. Phys. Rev. E **90**(1) (2014). https://doi.org/10.1103/physreve.90.012808

35. Hota, A.R., Sneh, T., Gupta, K.: Impacts of game-theoretic activation on epidemic spread over dynamical networks. SIAM J. Control Optim. **60**(2), S92–S118 (2021). https://doi.org/10.1137/20m1376923
36. Huang, Y., Zhu, Q.: Game-theoretic frameworks for epidemic spreading and human decision-making: A review. Dyn. Games Appl. **12**(1), 7–48 (2022). https://doi.org/10.1007/s13235-022-00428-0
37. Huys, R., Jirsa, V.K. (eds.): Nonlinear Dynamics in Human Behavior. Springer Berlin Heidelberg (2011). https://doi.org/10.1007/978-3-642-16262-6
38. Italian National Institute of Statistics (ISTAT): Population and Households. Available at https://www.istat.it/en/population-and-households. (2023), last Accessed: August 22, 2025
39. Jackson, M.O., Zenou, Y.: Games on Networks. In: Handbook of Game Theory with Economic Applications, vol. 4, chap. 3, pp. 95–163. Elsevier (2015)
40. Kermack, W.O., McKendrick, A.G., Walker, G.T.: A contribution to the mathematical theory of epidemics. Proc. Roy. Soc. A **115**(772), 700–721 (1927). https://doi.org/10.1098/rspa.1927.0118
41. Khazaei, H., Paarporn, K., Garcia, A., Eksin, C.: Disease spread coupled with evolutionary social distancing dynamics can lead to growing oscillations. In: 60th IEEE Conf. Decis. Control. pp. 4280–4286 (2021). https://doi.org/10.1109/CDC45484.2021.9683594
42. Kleitman, S., et al.: To comply or not comply? a latent profile analysis of behaviours and attitudes during the COVID-19 pandemic. PLOS ONE **16**(7), 1–22 (2021). https://doi.org/10.1371/journal.pone.0255268
43. Köhler, J., Schwenkel, L., Koch, A., Berberich, J., Pauli, P., Allgöwer, F.: Robust and optimal predictive control of the COVID-19 outbreak. Annu. Rev. Control **51**, 525–539 (2021). https://doi.org/10.1016/j.arcontrol.2020.11.002
44. Lajmanovich, A., Yorke, J.A.: A Deterministic Model for Gonorrhea in a Nonhomogeneous Population. Math. Biosci. **28**(3), 221 – 236 (1976). https://doi.org/10.1016/0025-5564(76)90125-5
45. Liu, T., Wang, J., Zhang, X., Cheng, D.: Game theoretic control of multiagent systems. SIAM J. Control Optim. **57**(3), 1691–1709 (2019). https://doi.org/10.1137/18m1177615
46. Madeo, D., Mocenni, C.: Identification and control of game-based epidemic models. Games **13**(1), 10 (2022). https://doi.org/10.3390/g13010010
47. Maitra, U., Hota, A.R., Srivastava, V.: Sis epidemic propagation under strategic non-myopic protection: A dynamic population game approach. IEEE Control Syst. Lett. **7**, 1578–1583 (2023). https://doi.org/10.1109/LCSYS.2023.3273504
48. Marden, J.R., Shamma, J.S.: Game theory and control. Annu. Rev. Control Robot. Auton. Syst. **1**(1), 105–134 (2018). https://doi.org/10.1146/annurev-control-060117-105102
49. Martins, N.C., Certório, J., La, R.J.: Epidemic population games and evolutionary dynamics. Automatica **153**, 111016 (2023). https://doi.org/10.1016/j.automatica.2023.111016
50. Mei, W., Mohagheghi, S., Zampieri, S., Bullo, F.: On the dynamics of deterministic epidemic propagation over networks. Annu. Rev. Control **44**, 116–128 (2017). https://doi.org/10.1016/j.arcontrol.2017.09.002
51. Mossong, J., Hens, N., Jit, M., Beutels, P., Auranen, K., Mikolajczyk, R., Massari, M., Salmaso, S., Tomba, G.S., Wallinga, J., Heijne, J., Sadkowska-Todys, M., Rosinska, M., Edmunds, W.J.: Social contacts and mixing patterns relevant to the spread of infectious diseases. PLOS Med. **5**(3), e74 (2008). https://doi.org/10.1371/journal.pmed.0050074
52. Nicola, M., Alsafi, Z., Sohrabi, C., Kerwan, A., Al-Jabir, A., Iosifidis, C., Agha, M., Agha, R.: The socio-economic implications of the coronavirus pandemic (COVID-19): A review. Int. J. Surg. **78**, 185–193 (2020). https://doi.org/10.1016/j.ijsu.2020.04.018
53. Nowzari, C., Preciado, V.M., Pappas, G.J.: Analysis and Control of Epidemics: A Survey of Spreading Processes on Complex Networks. IEEE Control Syst. Mag. **36**(1), 26–46 (2016). https://doi.org/10.1109/MCS.2015.2495000
54. Paarporn, K., Eksin, C.: SIS epidemics coupled with evolutionary social distancing dynamics. In: 2023 Am. Control Conf. pp. 4308–4313 (2023). https://doi.org/10.23919/ACC55779.2023.10156026

55. Parino, F., Zino, L., Calafiore, G.C., Rizzo, A.: A model predictive control approach to optimally devise a two-dose vaccination rollout: A case study on COVID-19 in Italy. Internat. J. Robust Nonlinear Control **33**(9), 4808–4823 (2021). https://doi.org/10.1002/rnc.5728
56. Parino, F., Zino, L., Porfiri, M., Rizzo, A.: Modelling and predicting the effect of social distancing and travel restrictions on COVID-19 spreading. J. R. Soc. Interface **18**(175), 20200875 (2021). https://doi.org/10.1098/rsif.2020.0875
57. Paré, P.E., Liu, J., Beck, C.L., Kirwan, B.E., Başar, T.: Analysis, Estimation, and Validation of Discrete-Time Epidemic Processes. IEEE Transactions on Control Systems Technology **28**(1), 79–93 (2020)
58. Paré, P.E., Beck, C.L., Başar, T.: Modeling, estimation, and analysis of epidemics over networks: An overview. Annu. Rev. Control **50**, 345–360 (2020). https://doi.org/10.1016/j.arcontrol.2020.09.003
59. Pastor-Satorras, R., Castellano, C., Van Mieghem, P., Vespignani, A.: Epidemic processes in complex networks. Rev. Mod. Phys. **87**, 925–979 (2015). https://doi.org/10.1103/RevModPhys.87.925
60. Peng, K., Lu, Z., Lin, V., Lindstrom, M.R., Parkinson, C., Wang, C., Bertozzi, A.L., Porter, M.A.: A multilayer network model of the coevolution of the spread of a disease and competing opinions. Math. Models Methods Appl. Sci. **31**(12), 2455–2494 (2021). https://doi.org/10.1142/s0218202521500536
61. Perra, N., Gonçalves, B., Pastor-Satorras, R., Vespignani, A.: Activity driven modeling of time varying networks. Sci. Rep. **2**, 469 (2012). https://doi.org/10.1038/srep00469
62. Perra, N.: Non-pharmaceutical interventions during the COVID-19 pandemic: A review. Phys. Rep. (2021). https://doi.org/10.1016/j.physrep.2021.02.001
63. Perra, N., Balcan, D., Gonçalves, B., Vespignani, A.: Towards a Characterization of Behavior-Disease Models. PLOS ONE **6**(8) (08 2011). https://doi.org/10.1371/journal.pone.0023084
64. Poletti, P., Caprile, B., Ajelli, M., Pugliese, A., Merler, S.: Spontaneous behavioural changes in response to epidemics. J. Theor. Biol. **260**(1), 31–40 (2009). https://doi.org/10.1016/j.jtbi.2009.04.029
65. Preciado, V.M., Sahneh, F.D., Scoglio, C.: A convex framework for optimal investment on disease awareness in social networks. In: 2013 IEEE Glob. Conf. Signal Inf. Process. pp. 851–854 (2013). https://doi.org/10.1109/GlobalSIP.2013.6737025
66. Preciado, V.M., Zargham, M., Enyioha, C., Jadbabaie, A., Pappas, G.: Optimal vaccine allocation to control epidemic outbreaks in arbitrary networks. In: 52nd IEEE Conf. Decis. Control. pp. 7486–7491 (2013). https://doi.org/10.1109/CDC.2013.6761078
67. Presidenza del Consiglio dei Ministri — Dipartimento della Protezione Civile: COVID-19 Italia — Monitoraggio situazione. Available at https://github.com/pcm-dpc/COVID-19. (2023), last Accessed: August 22, 2025
68. Proskurnikov, A.V., Tempo, R.: A tutorial on modeling and analysis of dynamic social networks. Part I. Annu. Rev. Control **43**, 65–79 (2017). https://doi.org/10.1016/j.arcontrol.2017.03.002
69. Qiu, J., Shen, B., Zhao, M., Wang, Z., Xie, B., Xu, Y.: A nationwide survey of psychological distress among Chinese people in the COVID-19 epidemic: implications and policy recommendations. Gen. Psychiatr. **33**(2), e100213 (2020). https://doi.org/10.1136/gpsych-2020-100213
70. Rizzo, A., Frasca, M., Porfiri, M.: Effect of individual behavior on epidemic spreading in activity driven networks. Phys. Rev. E **90**, 042801 (2014). https://doi.org/10.1103/PhysRevE.90.042801
71. Sahneh, F.D., Chowdhury, F.N., Scoglio, C.M.: On the existence of a threshold for preventive behavioral responses to suppress epidemic spreading. Sci. Rep. **2**(1) (2012). https://doi.org/10.1038/srep00632
72. Sandholm, W.H.: Population Games and Evolutionary Dynamics. Cambridge University Press (2010)
73. Satapathi, A., Dhar, N.K., Hota, A.R., Srivastava, V.: Coupled evolutionary behavioral and disease dynamics under reinfection risk. IEEE Trans. Control. Netw. Syst. pp. 1–12 (2023). https://doi.org/10.1109/TCNS.2023.3312250

74. She, B., Gracy, S., Sundaram, S., Sandberg, H., Johansson, K.H., Paré, P.E.: Epidemics Spread Over Networks: Influence of Infrastructure and Opinions, chap. 16, pp. 429–456. John Wiley & Sons, Ltd (2023). https://doi.org/10.1002/9781119857433.ch16
75. She, B., Liu, J., Sundaram, S., Paré, P.E.: On a networked SIS epidemic model with cooperative and antagonistic opinion dynamics. IEEE Trans. Control. Netw. Syst. **9**(3), 1154–1165 (2022). https://doi.org/10.1109/TCNS.2022.3145748
76. Simon, H.A.: Bounded rationality in social science: Today and tomorrow. Mind Soc. **1**(1), 25–39 (2000). https://doi.org/10.1007/BF02512227
77. Tunçgenç, B., El Zein, M., Sulik, J., Newson, M., Zhao, Y., Dezecache, G., Deroy, O.: Social influence matters: We follow pandemic guidelines most when our close circle does. Br. J. Psychol. (2021). https://doi.org/10.1111/bjop.12491
78. Valdano, E., Ferreri, L., Poletto, C., Colizza, V.: Analytical computation of the epidemic threshold on temporal networks. Physical Review X **5**, 021005 (2015). https://doi.org/10.1103/PhysRevX.5.021005
79. Van Bavel, J.J., et al.: Using social and behavioural science to support COVID-19 pandemic response. Nat. Hum. Behav. **4**(5), 460–471 (2020). https://doi.org/10.1038/s41562-020-0884-z
80. Verelst, F., Willem, L., Beutels, P.: Behavioural change models for infectious disease transmission: a systematic review (2010-2015). J Roy. Soc. Interface **13**, 20160820 (2016). https://doi.org/10.1098/rsif.2016.0820
81. Viswanadham, R.V.: How behavioral economics can inform the next mass vaccination campaign: A narrative review. Prev. Med. Rep. **32**, 102118 (Apr 2023). https://doi.org/10.1016/j.pmedr.2023.102118
82. Wang, Z., Andrews, M.A., Wu, Z.X., Wang, L., Bauch, C.T.: Coupled disease–behavior dynamics on complex networks: A review. Phys. Life Rev. **15**, 1 – 29 (2015). https://doi.org/10.1016/j.plrev.2015.07.006
83. Watts, D.J., Strogatz, S.H.: Collective dynamics of 'small-world' networks. Nature **393**(6684), 440–442 (1998). https://doi.org/10.1038/30918
84. Wells, C.R., Huppert, A., Fitzpatrick, M.C., Pandey, A., Velan, B., Singer, B.H., Bauch, C.T., Galvani, A.P.: Prosocial polio vaccination in Israel. Proc. Natl. Acad. Sci. USA **117**(23), 13138–13144 (2020). https://doi.org/10.1073/pnas.1922746117
85. Xuan, W., Ren, R., Paré, P.E., Ye, M., Ruf, S., Liu, J.: On a network SIS model with opinion dynamics. In: 21st IFAC World Congress. vol. 53, pp. 2582–2587 (2020). https://doi.org/10.1016/j.ifacol.2020.12.305
86. Ye, M., Zino, L., Mlakar, Ž., Bolderdijk, J.W., Risselada, H., Fennis, B., Cao, M.: Collective patterns of social diffusion are shaped by individual inertia and trend-seeking. Nat. Comm. **12**, 5698 (2021). https://doi.org/10.1038/s41467-021-25953-1
87. Ye, M., Zino, L., Rizzo, A., Cao, M.: Game-theoretic modeling of collective decision making during epidemics. Phys. Rev. E **104**(2), 024314 (2021). https://doi.org/10.1103/PhysRevE.104.024314
88. Young, H.P., Zamir, S. (eds.): Handbook of Game Theory with Economic Applications, vol. 4. Elsevier (2015)
89. Zino, L., Rizzo, A., Porfiri, M.: Continuous-time discrete-distribution theory for activity-driven networks. Phys. Rev. Lett. **117**, 228302 (2016). https://doi.org/10.1103/PhysRevLett.117.228302
90. Zino, L., Rizzo, A., Porfiri, M.: Modeling Memory Effects in Activity-Driven Networks. SIAM J. Appl. Dyn. Syst. **17**(4), 2830–2854 (2018). https://doi.org/10.1137/18M1171485
91. Zino, L., Rizzo, A., Porfiri, M.: On Assessing Control Actions for Epidemic Models on Temporal Networks. IEEE Control Syst. Lett. **4**(4), 797–802 (2020). https://doi.org/10.1109/LCSYS.2020.2993104
92. Zino, L., Cao, M.: Analysis, prediction, and control of epidemics: A survey from scalar to dynamic network models. IEEE Circuits Syst. Mag. **21**(4), 4–23 (2021). https://doi.org/10.1109/MCAS.2021.3118100
93. Zino, L., Ye, M., Rizzo, A., Calafiore, G.C.: On adaptive-gain control of replicator dynamics in population games. In: 62nd IEEE Conf. Decis. Control. pp. 7486–7491 (2023). https://doi.org/10.1109/CDC49753.2023.10383983