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Dynamic Control of Modern, Network-Based Epidemic Models*

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Abstract. In this paper we make the first steps to bridge the gap between classic control theory and modern, network-based epidemic models. In particular, we apply nonlinear model predictive control (NMPC) to a pairwise ODE model which we use to model a susceptible-infectious-susceptible (SIS) epidemic on nontrivial contact structures. While classic control of epidemics concentrates on aspects such as vaccination, quarantine, and fast diagnosis, our novel setup allows us to deliver control by altering the contact network within the population. Moreover, the ideal outcome of control is to eradicate the disease while keeping the network well connected. The paper gives a thorough and detailed numerical investigation of the impact and interaction of system and control parameters on the controllability of the system. For a certain combination of parameters, we used our method to identify the critical control bounds above which the system is controllable. We foresee that our approach can be extended to even more realistic or simulation-based models with the aim of applying these to real-world situations.

Key words. SIS epidemic, pairwise model, adaptive network, nonlinear model predictive control

AMS subject classifications. 34H20, 05C82, 37N25, 92D30

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1. Introduction.

1.1. Background.

Being able to control a process or a system can prove to be highly beneficial as it allows the user to tune it or operate it in a planned or ideal regime [6, 18]. Hence, *control theory* is a subject area on its own at the interface of subjects ranging from engineering and mathematics to biology [6, 8, 18]. Mathematical models of disease transmission, be they of simple compartmental type [2] or more modern network-type models [7, 13], have been and are being developed with the ultimate aim of making predictions about our capability to *control* outbreaks. An epidemiological model, describing disease transmission within a population, that is correctly developed and parametrized, offers important insight into understanding which control mechanisms under what circumstances can lead to a reduction in the prevalence of infection or its complete eradication. For many models, this problem is well understood, especially in terms of vaccination [2], quarantine, and contact tracing [14]. However, in all these cases control is of a static nature and does not always evolve in parallel with the epidemic spread. For example, vaccinating a proportion of the population in order to develop herd

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immunity is a preemptive step toward minimizing the impact of the disease. A somewhat more interactive control is achieved by the use of contact tracing, where the critical tracing rate for sexually transmitted infections (STIs) can differentiate between the disease-free and endemic states.

Control, in the general sense, is dynamic in nature, where via an external input or perturbation to the system the users are able to tune it toward a desired outcome. This process, in many cases, is dynamic where the challenge is to determine the optimal external input across time in order to reach a target or to minimize a cost function. In terms of epidemics, such questions have been investigated in order to determine, for example, the optimal time-dependent vaccination in a susceptible-infected/infectious-recovered model under minimizing a cost function that measures the cumulative number of infected and vaccinated people [17]. More recently, and by considering somewhat more complex control, but still in the context of compartmental ODE models, Hansen and Day [9] have considered optimal control in the presence of limited resources using isolation, vaccination, or mixed control strategies for the susceptible-infected-recovered (SIR) dynamics. Earlier on, Clancy and Piunovskiy [3] considered a variation of the classical SIR model with nonlinear infection rate function and have analytically computed optimal control based on isolation. Moreover, the authors considered how control computed based on the ODE model compared to control of the true stochastic process.

It is now evident that modern epidemiological models are amenable to accounting for and incorporating network structure which aims to mimic to some degree a more realistic contact pattern amongst members of a population. Pairwise models proved to be quite successful in this modeling endeavor as they provide a relatively simple representation of epidemics unfolding on a network as opposed to the homogeneous random mixing assumption of the classic compartmental models. In this paper we wish to bridge the gap between modern disease transmission models [11, 13] and control of epidemics, where the focus is on controlling the network rather than the recovery time or the proportion of immunized individuals. This opportunity to extend the scope of control arises naturally since the network of contacts is explicitly modeled and, thus, controllable. For example, in [15], Liu, Slotine, and Barabási studied the controllability of complex directed networks. For a deterministic, but not a stochastic, epidemic model, they investigated how the structure of the network influences its controllability. Their aim was to identify special vertices in the network, the so-called driver nodes, such that the system can be controlled completely through these nodes. By controllability the authors referred to structural controllability, which means that the system can be controlled for almost all control values. This is a generic property of the network which can be rephrased in terms of graph theory. By these tools the authors developed a method to find the minimal number of driver nodes in directed networks. Then this method was applied to real networks to study how the degree distribution of the network determines the minimal number of driver nodes.

Topological properties of nodes and edges have been exploited in order to address the problem of controlling networks. Namely, it is well known that isolating or immunizing highly connected nodes, cutting edges, or links with high betweenness centrality is far more efficient than selecting nodes and edges at random [1, 10]. When global information is poor, acquaintance immunization [5] provides an effective way to significantly reduce the spread of an epidemic. While extremely useful, none of the above arise directly from a classically or

mathematically rigorously formulated control problem. This observation together with the existence of a number of network-based mean-field models open up new opportunities to consider control of networks from a new or different perspective, and this is what we wish to elaborate on in this paper.

1.2. The problem. The aim of the paper is to investigate the control of a susceptible-infectious-susceptible (SIS) epidemic on a network, using pairwise equations, by controlling the creation and deletion of edges of certain types. The classic pairwise model augmented with the control elements leads to the following system of equations:

$$\dot{[I]} = \tau[SI] - \gamma[I], \tag{1a}$$

$$\dot{[SI]} = \gamma([II] - [SI]) + \tau([SSI] - [ISI] - [SI]) - u_1 \cdot f_1([SI]), \tag{1b}$$

$$\dot{[II]} = -2\gamma[II] + 2\tau([ISI] + [SI]), \tag{1c}$$

$$\dot{[SS]} = 2\gamma[SI] - 2\tau[SSI] + u_2 \cdot f_2([S], [SS]), \tag{1d}$$

where the $[\cdot]$ brackets denote expected number of singles and pairs of different types. For example, $[SI]$ denotes the expected value of the number of SI edges, which amounts effectively to counting on labeled networks. The evolution equations follow naturally by observing that singles depend on pairs, and pairs depend on triples. The precise derivation of these equations is discussed in detail in [12]. In the system above, the control parameters are u_1, the rate of cutting SI edges, and u_2, the rate of creating/deleting SS edges. The functions f_1 and f_2 will be specified later, but in general these will be linear or quadratic functions describing the precise rewiring mechanisms. The parameter τ is the per-contact infection rate, and γ is the rate of recovery. The desired outcome of our control problem is to eradicate the epidemic while keeping the network well connected, i.e., drive the system to $[I](T) = 0$, $n(T) = n_0$ for some final time $T > 0$, where N is the population size and $n(t) = ([SS] + 2[SI] + [II])/N$ is the average connectivity in the network.

1.3. The structure of the paper. The paper is organized as follows. First we consider in detail the problem of *constant control*, where the problem is effectively equivalent to a dynamic or adaptive network problem, since the epidemic dynamics and the dynamics of the network influence each other. Here, we will provide a classic bifurcation-type analysis, and we show that there are three qualitatively different regimes: (1) stable disease-free steady state, (2) stable endemic state, and, finally, (3) stable oscillations in both epidemic dynamics and the network's average connectivity. This is followed by the *dynamic control* case, where we use the nonlinear model predictive control (NMPC) method to determine if controllability is possible. In this control scheme the number of edges to be deleted and created is determined by minimizing an objective functional on a prediction horizon which is moved forward after each control step. Since finite time controllability, i.e., driving the system to the target set in finite time, is generally too much to expect from such a method, we use a different notion of controllability. We allow an error term ε, and we are interested in whether the values $[I](T)$ and $n(T)$ differ from their desired setpoint at most by ε. We study how successful control depends on parameters such as infection rate, control bounds, the frequency of intervention, and damping parameters in the control's target function. In many cases we give a substantial

treatment and identify controllable and uncontrollable situations. Finally, we discuss links to classic control and outlook toward the problem of controlling individual-based network models.

2. Constant control. In this section we make an attempt to control the epidemic by finding suitable values for u_1 and u_2 which stay constant until the end of the control period. We consider positive values for these parameters, so the control removes SI edges while creating new SS edges. The control should delete no more edges than the existing SI edges, so we take $f_1([SI]) = [SI]$, and the control should make no more SS connections than the total number of unconnected $S - S$ pairs, so we take $f_2([S], [SS]) = [S]([S] - 1) - [SS]$. By substituting $[S] = N - [I]$, system (1a)–(1d) takes the following form:

$$\dot{[I]} = \tau[SI] - \gamma[I], \tag{2a}$$

$$\dot{[SI]} = \gamma([II] - [SI]) + \tau([SSI] - [ISI] - [SI]) - u_1[SI], \tag{2b}$$

$$\dot{[II]} = -2\gamma[II] + 2\tau([ISI] + [SI]), \tag{2c}$$

$$\dot{[SS]} = 2\gamma[SI] - 2\tau[SSI] + u_2((N - [I])(N - [I] - 1) - [SS]). \tag{2d}$$

Now, instead of the variables $[SSI]$ and $[ISI]$, we are going to use the following approximations or closures [12]:

$$[SSI] \approx \frac{n-1}{n} \cdot \frac{[SS][SI]}{[S]} = \frac{n-1}{n} \cdot \frac{[SS][SI]}{N - [I]},$$

$$[ISI] \approx \frac{n-1}{n} \cdot \frac{[SI]^2}{[S]} = \frac{n-1}{n} \cdot \frac{[SI]^2}{N - [I]},$$

where $n(t)$ is the current mean degree of the network,

$$n(t) = \frac{2[SI] + [SS] + [II]}{N}.$$

Substituting these into the set of differential equations above, we obtain the following approximation:

$$\dot{[I]} = \tau[SI] - \gamma[I], \tag{3a}$$

$$\begin{aligned}\dot{[SI]} = {} & \gamma([II] - [SI]) + \tau\left(\frac{n-1}{n}\right)\frac{[SS][SI]}{N - [I]} \\ & - \tau\left(\frac{n-1}{n}\right)\frac{[SI]^2}{N - [I]} - (\tau + u_1)[SI],\end{aligned} \tag{3b}$$

$$\dot{[II]} = -2\gamma[II] + 2\tau\left(\left(\frac{n-1}{n}\right)\frac{[SI]^2}{N - [I]} + [SI]\right), \tag{3c}$$

$$\begin{aligned}\dot{[SS]} = {} & 2\gamma[SI] - 2\tau\left(\frac{n-1}{n}\right)\frac{[SS][SI]}{N - [I]} \\ & + u_2((N - [I])(N - [I] - 1) - [SS]).\end{aligned} \tag{3d}$$

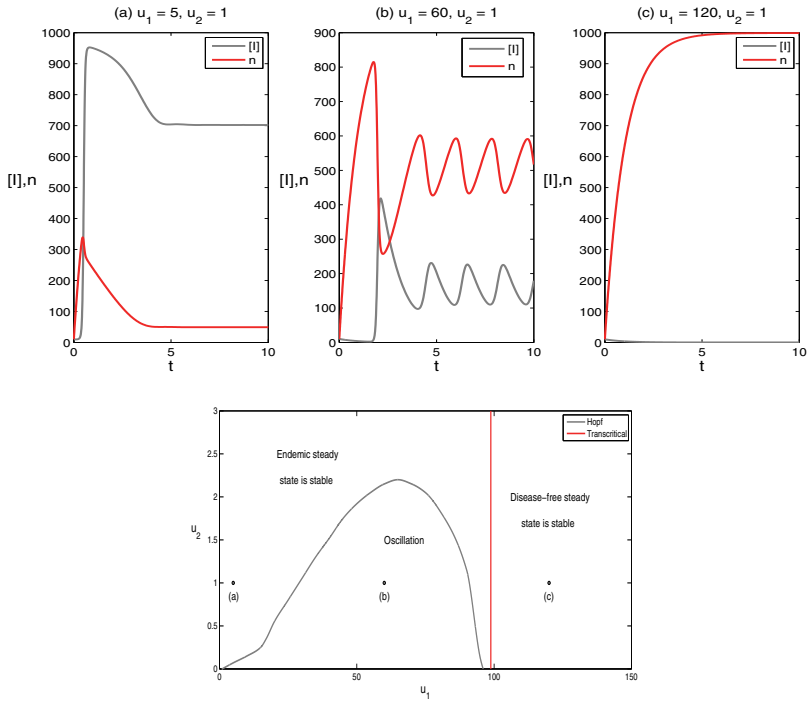

Figure 1. *Typical system behaviors (top row) and bifurcation diagram (bottom panel) for* $N = 1000$, $n(0) = 10$, $\tau = 0.1$, $\gamma = 1$, *and* $I(0) = 10$.

2.1. Dynamical behavior. In the appendix, we show that the system has two steady states, (1) the disease-free steady state and (2) the endemic steady state, and the system also exhibits a stable limit cycle as it undergoes a Hopf bifurcation; see the top panel of Figure 1. In the appendix we also give the detailed calculations corresponding to the stability analysis. The system is characterized by three main behaviors, as illustrated in Figure 1, in the (u_1, u_2) parameter space; see the bottom panel. The first case, from left to right, is when the endemic steady state is stable. In this case, after a short period of damped oscillations, the system settles to the endemic steady state, for which $[I](T) \neq 0$. The second case is when both the endemic and the disease-free steady states are unstable. In this case, the system variables exhibit stable oscillations, which fail to damp due to the instability of both steady states. Finally, in the third case, when the disease-free steady state is stable, the infection eventually disappears from the system, and due to the accumulation of the SS edges the network will become fully connected. Hence, the final state of the system is a complete network with every node in state S. The curve of transcritical bifurcation is given by (12) (i.e., $u_1 = \tau(N-2)-\gamma$), and the Hopf bifurcation set is defined as in (14). Obviously, varying parameters such as τ will not alter the qualitative behavior, but the stability regions of the various steady states change.

A key ingredient in considering such models is the relation between the dynamics of the epidemic and the network. The current system can be considered as an adaptive or dynamic network model [19], where the epidemic affects link deletion and creation, since these are

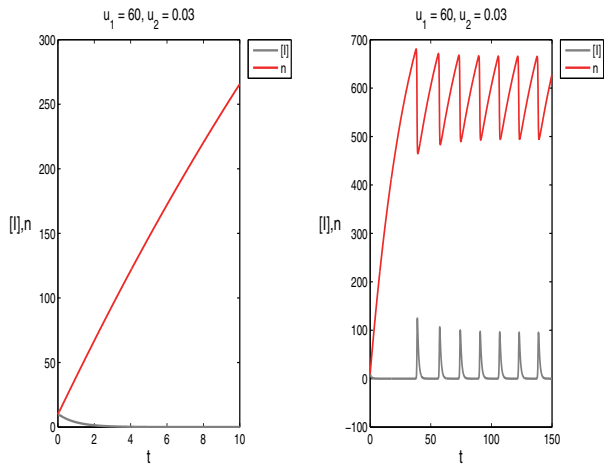

Figure 2. *Time evolution of prevalence and network connectivity for* $N = 1000$, $n(0) = 10$, $\tau = 0.1$, $\gamma = 1$, *and* $I(0) = 10$.

type-dependent, and at the same time, link activation deletion can favor or hinder epidemic spread, respectively. The impact of this interaction is maximal if both processes operate on a comparable time scale. When this is not the case, the system can exhibit a seemingly surprising behavior. In the case of small values for τ and u_2 and a comparably large value for u_1, such that the disease-free steady state is still unstable (i.e., $u_1 < \tau(N-2)-\gamma$), a seemingly eradicated epidemic reappears at a significant level; see Figure 2. This can be explained as follows. The low rate of infection combined with a low rate of link creation, but a high rate of SI edge cutting, pushes the system close to the disease-free steady state; infection slowly disappears from the system. But when the system gets close to the disease-free state, the cutting of the SI edges is less significant as there are few such edges, and in the meantime the number of SS edges is slowly building up. Hence, a network that becomes better connected with a very small seed of infection can spark an epidemic outbreak. Obviously, in a stochastic model it may not be feasible for the system to visit states of very low prevalence without the epidemic becoming extinct. If we would like to control a system in this way, it could be quite effective (infection could almost be removed from the system), but it is crucial to stop or alter the control at the right time, before another epidemic can start.

Concluding our analysis of constant control, we note that there is a wide range of parameter value combinations that lead to the eradication of the disease. Usually, this requires the deletion of SI edges at a fast rate at the expense of a dramatic drop in the mean degree of the network. The system then compensates by connecting susceptible individuals, and in the successful control case the network becomes completely connected, which is also a dramatic change. Trivially, we can delete SI edges at a very fast rate and then wait for the infecteds to recover without creating extra SS links followed by the creation of SS edges in order to reach the desired target connectivity in the network. To achieve this type or a similar type of control, in the next section we consider dynamic control using the NMPC algorithm.

3. Time-dependent control. We have seen in the previous section that constant control is not an effective way to control the mean degree of the network, and it is a very costly way to control the infection itself. In particular, cutting infection by breaking the network down is an extreme measure which in reality would correspond to a major quarantine at population level. This is obviously not feasible, and while the cutting of some potentially risky links in response to an epidemic is possible, in general individuals will aim to maintain some form of social connectedness. Hence, a realistic control should be able to eradicate the disease without leading to a heavily fragmented population. So in this section we introduce a more sophisticated form of control, i.e., time-dependent control.

The basic idea of time-dependent control is that we can update the control signal from time to time according to the current state of the system and our goals. So in this case the control signals u_1, u_2 will be piecewise constant functions. These functions should be bounded by some realistic values. We want u_1 to be positive, since creation of links between infected and susceptible individuals would hinder control. But this time we want to admit negative values for u_2 since deleting SS edges will prove useful in controlling the mean degree. There should exist constants M_1, M_2 such that $u_1 \le M_1$ and $|u_2| \le M_2$. We introduce a step size Δt for how often we can intervene and change the amount of control. In order to motivate our concept of controllability, it is worth recalling some existing notions of controllability in classical control theory. The property of being able to drive the system (possibly with some convergence rate) to the target as $t \to \infty$ is called asymptotic stability. In some cases this might be too restrictive and can be relaxed to practical asymptotic stability where only the possibility of driving the system in some neighborhood of the target is required as $t \to \infty$ (see [8]). However, in our case asymptotic properties are not relevant since the disease needs to be eradicated in a relatively short period of time. Therefore, we introduce a constant T which will mark the total length of the control period and is assumed to be some multiple of the infectious period of the disease. We will set a target value for the two variables we wish to control: $[I^*]$ for the number of infected individuals and n^* for the mean degree. Of course, controllability in finite time would be too restrictive, and so, motivated by the concept of practical asymptotic stability, we allow an error term ε and relax the finite-time controllability to our notion of controllability.

Definition 1. *The system is ε-controllable in time T with step size Δt and with control bounds M_1, M_2 to the targets $[I^*]$, n^* if there are piecewise constant functions $u_1, u_2 : [0, T] \to \mathbb{R}$ such that*

- $0 \le u_1(t) \le M_1$, $|u_2(t)| \le M_2$ *for all* $t \in [0, T]$,
- u_1 *and* u_2 *are constants in the intervals* $[(k-1)\Delta t, k\Delta t)$ *for all* $k = 1, 2, \dots, [T/\Delta t]$,
- $|[I](T) - [I^*]| \le \varepsilon$, *and* $|n(T) - n^*| \le \varepsilon$.

Naturally, for larger values of T one could demand smaller error terms, and as $T \to \infty$ asymptotic stability is expected. However, as we have already mentioned, we are not interested in asymptotic properties since the disease needs to be eradicated in a relatively short period of time.

We can group the parameters in the following way: the *system parameters* are N, τ, γ, $[I](0)$, the *control parameters* are $T, \Delta t, M_1, M_2$, the *targets* are $[I^*], n^*$, and the error term is ε; see Table 1. Our aim is to investigate how the controllability of the system depends on these parameters.

Table 1

Table summarizing system and control parameters as well the ideal outcome or target of control. With the applicability in mind, we work with the average infectious period D. Based on this, the time to the end of control is set as $T = QD$, and the number of interventions are also per average length of infection, $\Delta t = D/U$. The control bounds, M_1, M_2, and Q, can also be interpreted as parameters and will be treated as such.

System parameters		
N	size of population	1000
τ	rate of infection across a contact	
γ	rate of recovery	1
D	average length of the infectious period	$1/\gamma$
$[I](0)$	number of infecteds at $t = 0$	10
$[SS](0), [SI](0), [II](0)$	link types at $t = 0$	
Control parameters		
u_1	rate of cutting SI links	$0 \le u_1 \le M_1$
u_2	rate of creating/cutting SS links	$\lvert u_2\rvert \le M_2$
T	time to end of control	DQ
U	number of interventions during D	
Δt	step size for control adjustment	D/U
ε	error term	
Targets to achieve		
$[I^*]$	number of infecteds at T	0
n^*	average connectivity at T	$n(0)$
Damping parameters		
λ_1	controlling level of infection	
λ_2	controlling jumps in u_1	
λ_3	controlling average connectivity	
λ_4	controlling jumps in u_2	

We will fix some of the parameters, such as $N = 1000$ and $[I](0) = 0.01N = 10$. Let D be the length of the epidemic, and so the recovery rate is $\gamma = \frac{1}{D}$. Let $Q > 0$ be a constant such that $T = D \cdot Q$, meaning that we can set control over many generations/waves of infection. We also make the frequency of intervention of control depend on D, and we set U to be the parameter for how many times we will intervene during an average infectious period, so for the step size for control we use $\Delta t = \frac{D}{U}$. For simulation purposes we used $D = 1$, $Q = 10$, and $U = 5$. This means a control period of $T = 10$ and a step size of $\Delta t = 0.2$. While these are in arbitrary units, these values translate to seeing an intervention every day or every week for disease with a typical average infectious period of five days or five weeks, respectively. We also have to provide some reasonable values for M_1 and M_2. For example, if

$$u_1 \cdot \Delta t = 0.2,$$

then this corresponds to deleting 20% of the SI edges in Δt time. This is quite a considerable amount, and, hence, the maximum value M_1 for u_1 is set to $\frac{0.2}{\Delta t} = \frac{0.2 \cdot U}{D}$, which for our simulation parameters equates to 1. Similarly, an appropriate value for u_2 is $\frac{u_1}{N} = 0.001$, since u_2 has a quadratic multiplier in terms of N in the system of equations (3a)–(3d), while the multiplier of u_1 is linear in N.

Our targets will be $[I^*] = 0$ and $n^* = n(0)$, describing our goal of finding and applying a control which eradicates infection while keeping the network connected. In this case, without

loss of generality we set the target average connectivity to its value at time $t = 0$. Finally, the error will be acceptable if it is lower than 0.1, but ideally it should be of much smaller magnitude than this value. Nonetheless, we say the control is effective if $\varepsilon \leq 0.1$.

3.1. Nonlinear model predictive control. Here, for the reader's convenience, a brief introduction to nonlinear model predictive control (NMPC) is provided. NMPC is a control strategy which is suited for constrained, multivariable problems. The main idea of the method is as follows. At each step of the NMPC algorithm a sequence of optimal control signals is calculated along a prediction horizon of fixed length by minimizing an objective functional which includes predicted future outputs of the system. This optimization is a nonlinear programming problem which is solved subject to some constraints imposed on the input and output signals. Only the first control of the obtained sequence of optimal signals is applied to the system; then the prediction horizon is moved one step forward, and the next control signal is calculated the same way. Due to this moving horizon technique, the NMPC is also called receding horizon control. There are many applications of NMPC, such controlling drug dosing, industrial plants, or automobiles; see the collection of survey papers [16]. For further theoretical details on NMPC, we refer the reader to the monograph [8].

Our aim is now to apply the NMPC method to control epidemic spread. We use a system that is slightly different from those in the previous sections:

$$[\dot{I}] = \tau[SI] - \gamma[I], \tag{4}$$

$$[\dot{SI}] = \gamma([II] - [SI]) + \tau([SSI] - [ISI] - [SI]) - u_1[SI], \tag{5}$$

$$[\dot{II}] = -2\gamma[II] + 2\tau([ISI] + [SI]), \tag{6}$$

$$\begin{aligned}[\dot{SS}] = {} & \gamma[SI] - 2\tau[SSI] + \max\{u_2, 0\} \cdot ((N - [I])(N - [I] - 1) - [SS]) \\ & + \min\{u_2, 0\} \cdot [SS].\end{aligned} \tag{7}$$

We now admit the algorithm to assign negative values to u_2, so that it can also delete SS edges; (7) is adjusted accordingly. The vector of state variables and control variables will be $x = ([I], [IS], [II], [SS])$ and $u = (u_1, u_2)$, respectively. The output variables are the number of infected individuals and the mean degree, and so $y = ([I], n)$. The ith coordinate of x and y will be denoted by x_i and y_i, respectively, e.g., $y_1 = [I]$ and $y_2 = n$.

In order to apply the NMPC algorithm, the system given by (4)–(7) is first discretized. A time step Δt is fixed, and the system is observed only at instants $t = k\Delta t$, where $k \in \mathbb{Z}$. For simplicity, Δt is omitted, and $x(k)$, $y(k)$ are used, which means that x and y are evaluated at time instant $k\Delta t$. It is assumed that the control variables u_1 and u_2 are held constant along the intervals $[k\Delta t, (k+1)\Delta t)$ $(k \in \mathbb{Z})$; in other words, they are piecewise constant functions. With these conventions, (4)–(7) lead to the following discretized system:

$$x(k+1) = F(x(k), u(k)), \tag{8}$$

$$y(k) = h(x(k)), \tag{9}$$

where $x(k) \in \mathbb{R}^4$ is the vector of state variables, $u \in \mathbb{R}^2$ is the vector of input (control) signals, and $y \in \mathbb{R}^2$ is the vector of output signals. Furthermore, the function F denotes the numerical solution of the ODE system given by (4)–(7) on the interval $[k\Delta t, (k+1)\Delta t]$

and $h(x_1, x_2, x_3, x_4) = (x_1, (2x_2 + x_3 + x_4)/N)$. Due to practical limitations, the following constraints are imposed on the control signals:

$$0 \le u_1(k) \le M_1, \quad -M_2 \le u_2(k) \le M_2.$$

Now, the control action u at time k is computed as follows. A prediction horizon of P steps is set, and a nonlinear optimization procedure is applied over the admissible set of future control actions as described below. Let $u_i(k+j|k)$ $(i = 1, 2)$ denote an arbitrary admissible future control action at time $k + j$ chosen at time k. If the admissible sequences of future control actions $u_i(k|k), u_i(k+1|k), \dots, u_i(k+P-1|k)$ $(i = 1, 2)$ are chosen, then these controls yield predicted future outputs $y_1(k + j|k)$, $y_2(k + j|k)$ $(j = 1, 2, \dots, P - 1)$, where the notation means that $y_i(k + j|k)$ is a predicted output at time $k + j$ calculated at instant k. More specifically,

$$\begin{aligned} x(k+j|k) &= F\big(x(k+j-1|k), (u_1(k+j-1|k), u_2(k+j-1|k))\big), \\ y(k+j|k) &= h(x(k+j|k)), \qquad j = 0, 1, 2, \dots, P-1, \end{aligned}$$

where $x(k+j|k)$ denotes the predicted state at $k+j$ calculated at instant k. The setpoints for the output signals are $y_{1_s} = [I^*] = 0$ and $y_{2_s} = n^* = n(0)$; therefore, the objective functional $J\colon \mathbb{R}^{2P} \to \mathbb{R}$ to be minimized has the form

$$\begin{aligned} J\big(u(k|k), \dots, u(k+P-1|k)\big) = \sum_{j=0}^{P-1} &\lambda_1 (y_1(k+j|k))^2 + \lambda_2 (\Delta u_1(k+j|k))^2 \\ &+ \lambda_3 (y_2(k+j|k) - n(0))^2 + \lambda_4 (\Delta u_2(k+j|k))^2 \end{aligned} \tag{10}$$

with parameters $\lambda_1, \dots, \lambda_4$, where $\Delta u_i(k+j|k) = u_i(k+j|k) - u_i(k+j-1|k)$ is the predicted control effort at instant $k + j$ calculated at time k. The evaluation of the functional J requires the numerical solution of the ODE system given by (4)–(7). Clearly, by adjusting the parameters, the penalty for large control efforts or departures from the control target can be adjusted as needed. For example, λ_1 penalizes small departures from the no-epidemic state, while λ_2 penalizes large changes in rewiring rates. Now, solving the nonlinear optimization problem given above, a sequence of optimal controls $u_i(k|k), u_i(k+1|k), \dots, u_i(k+P-1|k)$ $(i = 1, 2)$ is obtained. This can be done by using a nonlinear optimization routine, such as *lsqnonlin* in MATLAB, with the quadratic functional J as an input. Then only $u(k) := (u_1(k|k), u_2(k|k))$ is applied to the system, and the prediction horizon is translated one step forward. The same optimization procedure is implemented to calculate the next control.

It is intuitively clear that dynamical control is more effective than constant control. However, the number of parameters involved in setting up or specifying dynamic control makes it nontrivial to understand which combinations of factors and what parameter values will make the system controllable. In the next section, we will numerically explore in detail the impact of system, control, and damping parameters; see Table 1.

3.2. The interplay between the infection rate and control bounds. First, let us analyze the prevalence level, i.e., the number of infected individuals at time $T = 10$, $[I](T)$, and the mean degree at time T in the uncontrolled system $(u_1, u_2 \equiv 0)$ for different values of τ. As

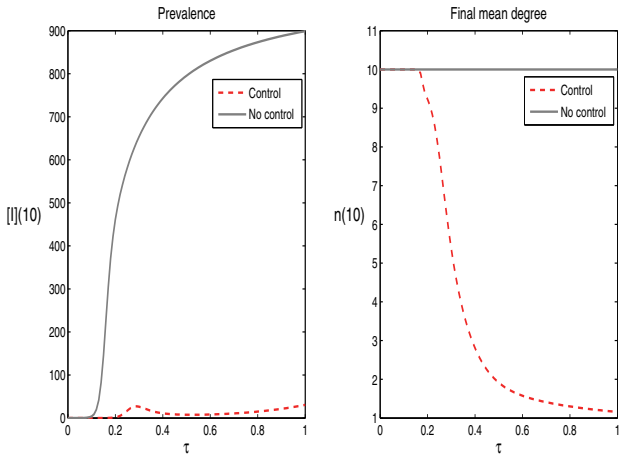

Figure 3. *The value of prevalence and network connectivity at the end of the control at time* $T = 10$, $[I](T)$ *and* $n(T)$, *as a function of the transmission parameter* τ *for* $\gamma = 1$, $N = 1000$, $I(0) = 10$, $I^* = 0$, $n(0) = n^* = 10$, $M_1 = 1$, $M_2 = 0.001$, $\Delta t = 0.1$, $\lambda_1 = 10^4$, *and* $\lambda_2 = \lambda_3 = \lambda_4 = 1$.

expected, in Figure 3 we can see that for very small values of τ (for about $\tau < 0.05$) the infection disappears from the system even without control. However, for higher values of τ the disease becomes more widespread, and the prevalence level converges toward the full population size. When no control is applied, the mean degree of the system remains unchanged in each step, and so naturally the final value of the mean degree is the initial value $n(0) = 10$ for each τ.

Now, using the NMPC method introduced above, the case of dynamic control is studied. Initially, we consider a set of fixed control parameters, M_1 and M_2, and a varying value of τ. Naturally, it is easier to control the infection when the infection rate is low and impossible within the given control bounds if the infection rate is high. In Figure 3, the prevalence and the mean degree at time T for different values of τ are plotted. The figure shows that for approximately $\tau > 0.15$ the control is ineffective; the values of both $[I](T)$ and $n(T)$ visibly differ from their target at the end of the control period. For about $\tau > 0.25$, the final number of $[I]$ is greater than $[I](0) = 10$, so in this case the control failed to decrease the initial number of infected individuals. For even higher values of τ, the control has little effect on $[I]$ or n, and so if $\tau \to \infty$, the final values of these variables converge to the final values of an uncontrolled system. The final mean degree of 10 can be attained for some small vales of τ, which cannot be said in the uncontrolled case. However, the value of $n(10)$ becomes much lower for higher values of τ despite the control.

This behavior is due to the strict bounds on the values of the control parameters: limiting the cutting rate of SI edges, u_1, throughout the entire control period makes the control inefficient for higher values of τ. For high infection rates even SS edges are cut, but again with a limited strength and making little difference. In fact, this results only in the drop of the mean degree, since in this case u_2 has no capacity to make new connections. To shed some light on the precise dependency of successful control on the bounds of the rewiring rates for different values of τ, a detailed numerical exploration is carried out. To carry out this

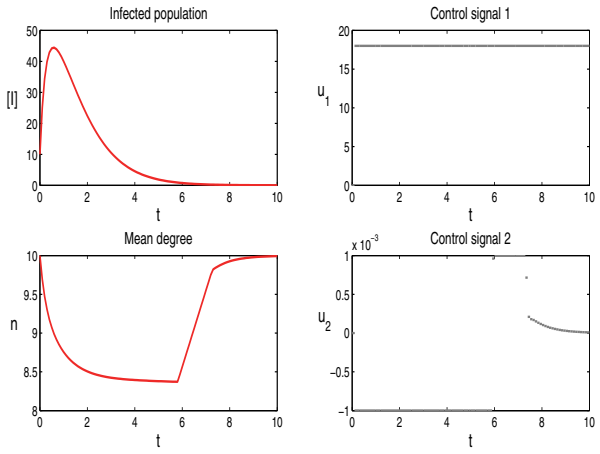

Figure 4. *Time evolution of prevalence, network connectivity, and control signals, u_1 and u_2, for $\tau = 2$, $\gamma = 1$, $N = 1000$, $I(0) = 10$, $I^* = 0$, $n(0) = n^* = 10$, $M_1 = 18$, $M_2 = 0.001$, $\Delta t = 0.1$, $\lambda_1 = 10^4$, and $\lambda_2 = \lambda_3 = \lambda_4 = 1$.*

exploration, we fix the damping parameters as follows: $\lambda_1 = 10^4$ and $\lambda_2 = \lambda_3 = \lambda_4 = 1$. This choice penalizes even a small departure from the ultimate target of disease eradication. While we fix these, the damping parameters themselves will impact the controllability of the system, and this is considered in the next subsection.

First, we will investigate the effect of M_1's magnitude with fixed values for τ and M_2. A value of $\tau = 2$ is a good starting point given that with the previous bounds for link rewiring, $M_1 = 1$ and $M_2 = 0.001$, control was not successful even for $\tau = 1$; see Figure 3. If we wish to keep $M_2 = 0.001$, we should increase the value of M_1. Figure 4 shows that $M_1 = 18$ makes the system controllable.

Extensive numerical simulations suggest that for a fixed value of τ and M_2, there is a critical value M_1^c such that if M_1 is lower than M_1^c, the control is not effective. However, if M_1 is larger than the critical value, then control is effective in T units of time. The higher the value of M_1, the less time is needed to control the system. But choosing a high value for M_1 implies that control is more severe or drastic. Hence, if our aim is to control our system in T units of time by using the least invasive control, it is optimal to choose M_1^c as the bound for M_1. This critical value is the strictest bound admissible. In Figure 5 (left panel), the critical value M_1^c for three different values of M_2 is plotted as τ is varied. These curves in fact define the strictest possible bounds, and, hence, one can use these to identify (M_1, M_2) pairs that can deliver a successful control. Moreover, the same figure shows that higher values of M_2 have negligible effect on the critical M_1 curve, since the fast creation of SS does not help to control the epidemic. In Figure 5 (right panel) the critical value of M_1 is plotted for a range of M_2 values and different infection rates. The situation here is the same as that in the left panel of the figure: choosing bounds below this curve will not result in an effective control. Choosing a pair (M_1, M_2) belonging to these curves is in some sense optimal, since these represent the strictest bounds.

The above results are expected heuristically; however, it is less obvious to what extent

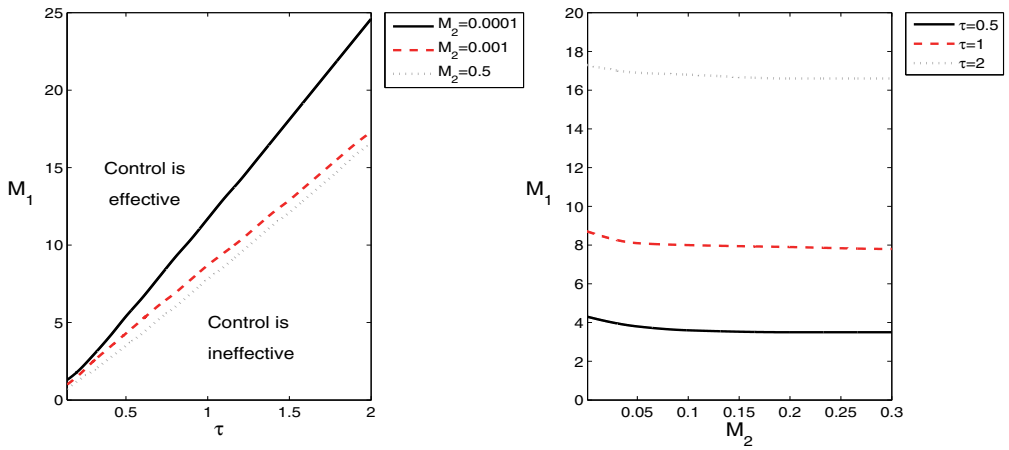

Figure 5. *Threshold plots illustrating the relation between system (τ) and control parameters (M_1 and M_2) for $\gamma = 1$, $N = 1000$, $I(0) = 10$, $I^* = 0$, $n(0) = n^* = 10$, $\Delta t = 0.1$, $\lambda_1 = 10^4$, and $\lambda_2 = \lambda_3 = \lambda_4 = 1$.*

Table 2

Critical value of M_1 for the cases $-M_2 \le u_2 \le M_2$ and $0 \le u_2 \le M_2$ with fixed $M_2 = 0.5$ and for $\gamma = 1$, $N = 1000$, $I(0) = 10$, $I^* = 0$, $n(0) = n^* = 10$, $\Delta t = 0.1$, $\lambda_1 = 10^4$, and $\lambda_2 = \lambda_3 = \lambda_4 = 1$.

τ	0.25	0.5	0.75	1	1.25	1.5	1.75	2
M_1	1.43	3.48	5.61	7.79	9.98	12.17	14.37	16.56
M_1 ($u_2 \ge 0$)	1.91	4.13	6.24	8.26	10.30	12.44	14.61	16.78

the controllability of the system changes when SS-type edges are not allowed to be deleted. (Note that the original idea of controlling the epidemic was to delete SI edges and keep the network connected by creating SS edges.) The bounds for u_2 were $-M_2 \le u_2 \le M_2$; i.e., SS edges were allowed to be cut. We carried out numerical experiments by using the restricted control bounds $0 \le u_2 \le M_2$; i.e., SS edges could only be created. For several values of τ and for $M_2 = 0.5$, we determined the critical value of M_1. In Table 2 this critical value is shown for both cases, i.e., for the cases when $-M_2 \le u_2 \le M_2$ and when $0 \le u_2 \le M_2$. We note that the numbers corresponding to the case $-M_2 \le u_2 \le M_2$ are the same as those shown in Figure 5 (left panel). One can observe that it is essential to delete SS edges in the first period of the control procedure; otherwise higher cutting rates have to be applied in order to achieve control.

3.3. Effects of Δt and the damping parameters on controllability. In this section we analyze how the value of the step size Δt and the damping parameters (i.e., λ_i $(i = 1, 2, 3, 4)$) in the cost functional affect system controllability. Let us first deal with the step size. A greater value for this means a slower reaction, and so as we increase it, controlling the system requires more radical changes in control, and the change in the mean degree during the control period could be quite drastic. However, we experienced that step sizes $\Delta t \le 5$ are effective, which means that $U = 0.2$ (i.e., $\Delta t = D/U = 1/0.2 = 5$) is not enough, but any larger U suffices (the parameter U marked the number of control actions during the average infectious

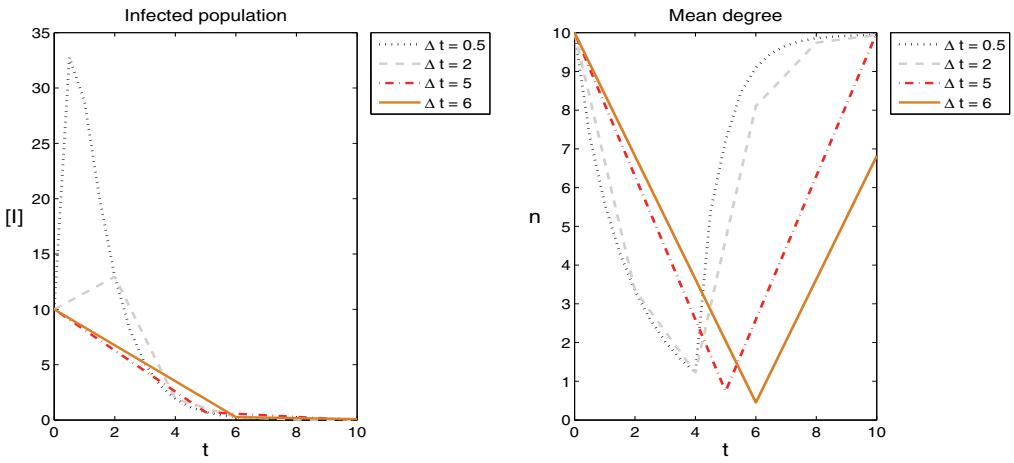

Figure 6. *The impact of intervention frequency in terms of the time evolution of prevalence and network connectivity for* $\tau = 1$, $\gamma = 1$, $N = 1000$, $I(0) = 10$, $I^* = 0$, $n(0) = n^* = 10$, $M_1 = 7.8$, $M_2 = 0.5$, $\lambda_1 = 10^4$, *and* $\lambda_2 = \lambda_3 = \lambda_4 = 1$.

period D, and U needs to take values less than 1 if one wants to investigate slow reactions in control). For a greater step size, the reaction of the control is not fast enough to control the system in $T = 10$ units of time. Figure 6 uses $M_2 = 0.5$, $\tau = 1$, and the critical value of $M_1^c = 7.8$. In Figure 6 the effect of control is shown for four different values of Δt. It is clear that the system is only controllable if time steps are small enough. While we do not separately investigate the effect of the control parameter T, we note that an increase in the control horizon is likely to make controllability possible.

Let us continue with the analysis of the damping parameters. The damping parameters assigned to Δu_1 and Δu_2 are λ_2 and λ_4, respectively. When both are large compared to λ_1 and λ_3, achieving the control target will be difficult due to small increments in the rewiring rates. The damping parameters λ_1 and λ_3, controlling the level of infection and average connectivity, have a strong effect on controllability. In order to show how to choose their values, we carried out an extensive study by running the NMPC procedure for (λ_1, λ_3) pairs varied on a lattice and determined how far $I(T)$ and $n(T)$ are from their target values. In Figure 7, the color denotes the value of the error, that is, $\max\{I(T), |n(T) - n^*|\}$, for the parameter range $\lambda_1, \lambda_3 \in [0, 100]$. One can see that there is only a narrow zone in the parameter domain where the error is less than $\varepsilon = 0.1$, in which case the control procedure was called effective. This explains why the value of λ_1 was chosen much larger than the values of the other damping parameters in the previous figures.

3.4. Control-bound-induced targets. Posing a controllability question usually involves establishing the control bounds for a given target. However, understanding what targets can be achieved with given control bounds is equally valuable, especially when these could be close to the ideal targets. We have seen in the previous sections that if we fix a value of the constraint (i.e., M_2) on u_2 and the infection rate τ, there exists a critical value for M_1 below which the system is not controllable. In many cases, the main difficulty was posed by reaching

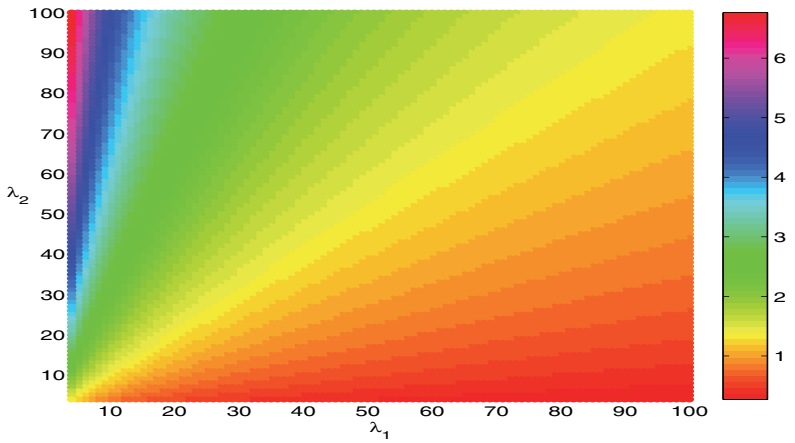

Figure 7. *Dependence of system's controllability on the damping parameters λ_1 and λ_3 for $\tau = 1$, $\gamma = 1$, $N = 1000$, $I(0) = 10$, $I^* = 0$, $n(0) = n^* = 10$, $M_1 = 8.5$, $M_2 = 0.5$, $\Delta t = 0.1$, and $\lambda_2 = \lambda_4 = 0$. The color denotes the value of the error $\max\{I(T), |n(T) - n^*|\}$. Note that the region with the error less than 0.1 is an extremely narrow area close to the horizontal axis; it is a triangle-shaped region with an acute angle.*

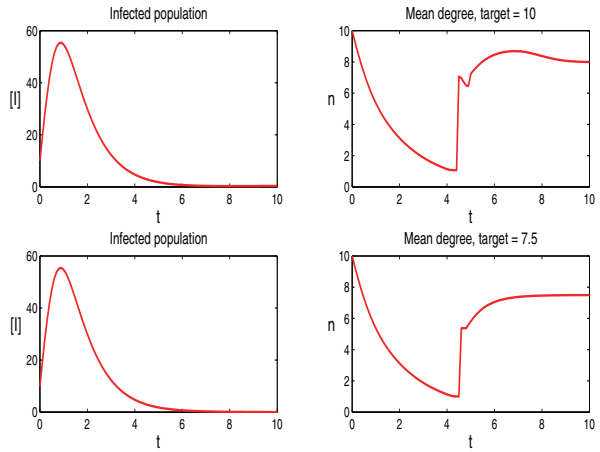

Figure 8. *The effect of adjusting the control targets in terms of the time evolution of prevalence and network connectivity for $\tau = 1$, $\gamma = 1$, $N = 1000$, $I(0) = 10$, $I^* = 0$, $n(0) = 10$, $M_1 = 6$, $M_2 = 0.5$, $\Delta t = 0.1$, $\lambda_1 = 10^4$, and $\lambda_2 = \lambda_3 = \lambda_4 = 1$.*

the target connectivity. More importantly, the infection is almost completely eradicated from the system in every case if the formerly fixed $\lambda_1 = 10^4$ and $\lambda_2 = \lambda_3 = \lambda_4 = 1$ damping parameters are used. So, for a weakened control, let us admit a decrease in the value of the target mean degree. For example, if we use the previously seen $M_2 = 0.5$, $\tau = 1$ parameters, we have seen that the critical value M_1^c was 7.8, and Figure 5 (left panel) shows that the system is not controllable for $M_1 = 6$. Now let us use the target value $n^* = 7.5$ admitting a 25% decrease in the mean degree. In Figure 8, it is clearly illustrated that $n^* = 10$ cannot be achieved; see top row. However, modifying the target to $n^* = 7.5$, the system becomes

Table 3

Table showing the achievable target n^* for different values of the control bound M_1.

M_1	n^*
7.8	10
7.5	9.2
7	8.6
6.5	8.2
6	7.6
5.5	7.2
5	6.6
4.5	6
4	5.2
3.5	4.4

controllable; see bottom row. Let us fix the parameters M_2 and τ above and analyze the highest possible achievable n^* for different values of M_1. Table 3 shows the results of some of our simulations.

3.5. Summary of results about time-dependent control. Our goal was to show that epidemics can be controlled by solely acting upon the individuals' contact pattern or network. This is in contrast to isolation and vaccination policies that act directly on individuals and only indirectly on links. Control of the network was achieved by introducing link deletion (u_1) and creation rates (u_2) into the pairwise ODE system (4)–(7). These control parameters are considered to be piecewise constant in time. In order to determine control, i.e., decide the rate at which edges are deleted or created at given times, we used the NMPC algorithm and showed that, in many cases, the system can be guided to the desired target once the values of the control parameters are carefully chosen. The algorithm uses several parameters as shown in Table 1. The main step in finding the actual values of u_1 and u_2 is to minimize the objective functional J given in (10). The success of the NMPC algorithm is highly sensitive to the value of the artificial damping parameters λ_is, which are direct entries of the objective functional J, as is shown in subsection 3.3. Figure 7 shows that the values of the damping parameters have to be chosen carefully, as randomly chosen parameter values lead to failure of control. The value of the step size Δt, determining how often we intervene, also plays an important role. We investigated how the number of control actions during the average infectious period needs to be chosen in order to have a successful control. Finally, the choice of the bounds M_1 and M_2 for the link deletion u_1 and link creation u_2 is dictated by the magnitude of the infection rate τ and by length of the control horizon T. It is heuristically obvious that for larger infection rates we need a larger control bound, M_1, for link deletion. However, it is less obvious how the control bound depends on τ. This was investigated in detail in subsection 3.2. Intuitively, SI edges have to be destroyed and SS edges have to be created in order to eradicate infection and keep the network well connected. However, for higher values of the

infection rate, this control strategy is not effective. Successful control, for this regime, requires that a proportion of SS links also be deleted early on in the epidemic. This will prevent the fast spread of the infection through the network. The NMPC algorithm found this control strategy, as it was explained in subsection 3.2. Moreover, in Table 2 we showed that control is less effective for $u_2 \in [0, M_2]$ compared to the $u_2 \in [-M_2, M_2]$ case.

4. Discussion. The novelty of our paper is twofold: (a) control is applied to the network of contacts leading to dynamic contact patterns, and (b) the NMPC technique, as opposed to more classical approaches, is used to compute the optimal control. The main purpose of the study was to contribute to highlighting the opportunities offered by linking modern network-based ODEs and control. For this reason, in designing the study, our natural instinct was to concentrate on controlling the network, as this could not be the subject of control when using classical compartmental models, since these assume homogeneous random mixing. The control in this paper does not appear in the form of what could be termed as classical control. More precisely, such problems in epidemiology involve the minimization of an integral or cost function. Here, we focus on the end target, and we identify the piecewise constant control that allows us to be as close as possible to the final target. Hence, within the setting of our control, we ignore costs of control and those induced by infection.

It is straightforward to apply the methodology presented here to more complicated settings involving costs and competing effects, such as the tradeoff in cost between isolation, vaccination, mixed methods, and network control. Namely, more vaccination increases the cost but results in fewer infectious cases, which in turn reduces cost. Similarly, cutting links involves a cost but results in fewer infectious cases, and hence a smaller cost. In our current setting, such tradeoffs were realized by aiming to control disease spread while maintaining social cohesion. Obviously, if the connectedness of the network is not required, control will produce a disconnected network where transmission is no longer possible. In real life this is not the case. For example, STIs persist due to the network being well connected with many concurrent partnerships, and it is reasonable to assume that control will need to be achieved without fragmenting the network into components.

The next steps will be (i) to incorporate control of classical type with an appropriate cost function and evaluate how network and nonnetwork control methods perform and (ii) to extend this approach to individual-based network simulations and work out to what extent the control predicted by network-based mean-field models would result in good/optimal control in the full stochastic model. Studies in this latter direction already exist, and the first signs are positive. Namely, control computed from mean-field models seems to translate well, at least for some cases, and provides a viable solution for the control of the stochastic/simulation counterpart [4, 20]. In general, the cost of computing control from ODEs is much smaller than the cost of working out control from stochastic models. Therefore, if ODE-inspired control applies to the stochastic setting, in principle it is possible to control complex systems without the additional complexity or cost. If it proves to apply more widely or be portable between different systems, such an approach could gain momentum and could lead to many worthwhile and important applications in the context of controlling real systems.

Appendix: Steady states and their stability for the constant control case. Let us calculate the steady states of system (3a)–(3d). These are the solutions of

$$0 = \tau[SI] - \gamma[I], \tag{11a}$$

$$0 = \gamma([II] - [SI]) + \tau\left(1 - \frac{N}{2\cdot[SI] + [II] + [SS]}\right)\frac{[SS][SI]}{N - [I]} - \tau\left(1 - \frac{N}{2\cdot[SI] + [II] + [SS]}\right)\frac{[SI]^2}{N - [I]} - (\tau + u_1)[SI], \tag{11b}$$

$$0 = -2\gamma[II] + 2\tau\left(\left(1 - \frac{N}{2\cdot[SI] + [II] + [SS]}\right)\frac{[SI]^2}{N - [I]} + [SI]\right), \tag{11c}$$

$$0 = 2\gamma[SI] - 2\tau\left(1 - \frac{N}{2\cdot[SI] + [II] + [SS]}\right)\frac{[SS][SI]}{N - [I]} + u_2((N - [I])(N - [I] - 1) - [SS]). \tag{11d}$$

By solution, we mean an all-real, all-positive solution. It is easy to see that the disease-free steady state of the system is

$$\begin{aligned}[I] &= 0,\\ [SI] &= 0,\\ [II] &= 0,\\ [SS] &= N(N-1).\end{aligned}$$

Denoting the disease-free steady state as E_d, the Jacobian at state E_d is

$$J(E_d) = \begin{pmatrix} -\gamma & \tau & 0 & 0\\ 0 & -\gamma + \tau(N-2) - (\tau + u_1) & \gamma & 0\\ 0 & 2\tau & -2\gamma & 0\\ -u_2(2N+1) & 2\gamma - 2\tau(N-2) & 0 & -u_2 \end{pmatrix}.$$

It is clear that $-\gamma$ and $-u_2$ are eigenvalues of the Jacobian, and these eigenvalues are always real and negative. So we only have to deal with the eigenvalues of the inner 2×2 submatrix:

$$\begin{pmatrix} -\gamma + \tau(N-2) - (\tau + u_1) & \gamma\\ 2\tau & -2\gamma \end{pmatrix}.$$

The determinant of this submatrix is $2\gamma(\gamma - \tau(N-2) + u_1)$, and its trace is $-3\gamma + \tau(N-3) - u_1$. For stability we need the eigenvalues to have negative real parts. For this the determinant has to be positive, and the trace has to be negative. So if $u_1 > \tau(N-2) - \gamma$ and $u_1 > \tau(N-3) - 3\gamma$, the disease-free steady state is stable. Note that the second condition bears no new information, and so we can exclude that. Thus our only criterion for the disease-free steady state to be stable is

$$u_1 > \tau(N-2) - \gamma. \tag{12}$$

Note that in the disease-free steady state, the mean degree is $n = \frac{N(N-1)}{N} = N - 1$, and so the network becomes fully connected.

To calculate the endemic steady state(s), we first express the variable $[SI]$ from (11a) to get

$$[SI] = \frac{\gamma}{\tau}[I].$$

Then we express $[SS]$ from (11b)–(11d):

$$[SS] = u_2(N - [I])(N - [I] - 1) - \frac{u_1}{u_2} \cdot \frac{\gamma}{\tau}[I].$$

We substitute these expressions of $[SI]$ and $[SS]$ into (11c). We obtain a quadratic equation for $[II]$, from which $[II]$ can also be expressed in terms of $[I]$:

$$[II] = \frac{1}{2}\frac{(A - B \cdot C + \sqrt{D})}{B},$$

where

$$\begin{aligned} A &= \frac{[SI] + N - [I]}{N[SI]}, \\ B &= \gamma \cdot \frac{N - [I]}{\tau N[SI]^2}, \\ C &= 2[SI] + [SS], \\ D &= (A - B \cdot C)^2 - 4B(1 - A \cdot C). \end{aligned}$$

Since we are looking for all-real solutions, if $D < 0$, we are left without a solution having a meaning to us. Otherwise, we substitute these expressions for $[SI]$, $[SS]$, and $[II]$ into (11b), and we get an equation containing only the unknown $[I]$. Due to its complexity, we refrain from writing it out in detail, but let us denote it as equation $(*)$. By solving $(*)$ we get the endemic steady states. Our numerical experiments show that there is always only one all-positive solution, and so we can conclude that the endemic steady state (if it exists) is unique. Let us denote this state by $E_e(u_1, u_2)$. The Jacobian is far more complicated this time; we exclude its concrete form. Substituting $E_e(u_1, u_2)$ into the Jacobian, we can see by numerical experiments that for some values of u_1 there exists a value u_2^* such that, for a lower value of u_2 than this u_2^*, the Jacobian at $E_e(u_1, u_2)$ has two real, negative eigenvalues and two imaginary eigenvalues with positive real parts. For $u_2 > u_2^*$, the real part of the two imaginary eigenvalues becomes negative. To calculate the exact value of this u_2^*, let us use the method introduced in [19] and write the characteristic polynomial of the Jacobian at E_e in the following form:

$$\lambda^4 - b_3\lambda^3 + b_2\lambda^2 - b_1\lambda + b_0,$$

such that $b_3 = \operatorname{Tr} J(E_e)$, $b_0 = \det J(E_e)$, and b_1, b_2 can be given as the sum of some subdeterminants of the Jacobian, the concrete form of which is not important at this moment. In the case of 4×4 matrices the necessary and sufficient conditions for the existence of pure imaginary eigenvalues are

$$b_0 b_3^2 = b_1(b_2 b_3 - b_1) \quad \text{and} \quad \operatorname{sign} b_1 = \operatorname{sign} b_3. \tag{13}$$

Thus the Hopf bifurcation set can be defined as

$$H = \{(u_1, u_2) \in \mathbb{R}^2_+ : \exists\ [I] \in [0, N] \text{ such that } (*),\ (13) \text{ hold}\}. \tag{14}$$

This is a simple curve in the (u_1, u_2)-parameter plane. E_e is stable above the curve and unstable below. There is notable oscillation in the value of $[I]$ according to time in the unstable region.

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