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 non-trivial 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. The analysis reveals, that for certain set parameters it is possible to identify 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 to apply these to real-world situations.

Keywords: SIS epidemic; pairwise model; adaptive network; nonlinear model predictive control

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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 \[4, 13\]. Hence, control theory is a subject area on its own at the interface of subjects ranging from engineering and mathematics to biology \[4, 5, 14\]. Mathematical models of disease transmission, be it of simple compartmental type \[1\] or more modern network-type models \[4, 9\], 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 parametrised, offers important insight into understanding which control mechanisms and 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 \[1\], quarantine and contact tracing \[10\]. However, in all these cases control does not form an integral part of the disease dynamics and often only comes in as the proportion of the population that needs to be vaccinated in order to develop herd immunity so that infection can be stopped or as the critical contact tracing rate, which for sexually transmitted infections (STIs) can differentiate between the disease-free and epidemic state.

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 towards 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 minimise 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 (SIR) model under minimising a cost function that measures the cumulative amount of infected and vaccinated people \[13\]. More recently, but still in the context of classic compartmental models, Hansen & Day \[6\] have considered optimal control in the presence of limited resources.

It is now evident that modern epidemiological models are amenable to account for and incorporate 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 modelling endeavour as they provide a relatively simple representation of epidemics unfolding on a network as opposed to the homogenous random mixing assumption of the classic compartmental models. In this paper we wish to bridge the gap between modern disease transmission models \[7, 9\] and control of epidemics, where the focus is on controlling the network and not so much disease parameters, such as recovery time or the widely used pre-emptive or reactive vaccination. This opportunity to broaden the control’s target arises naturally since the network of contact is explicitly modeled, and thus controllable. For example, in \[11\], Barabási et al. 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 completely be controlled 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.

1.2 The problem

The aim of the paper is to investigate the control of a susceptible-infected/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:

\[
\begin{align*}
\dot{I} &= \tau[SI] - \gamma[I], \\
\dot{SI} &= \gamma([II] - [SI]) + \tau([SSI] - [ISI] - [SI]) - u_1 \cdot f_1([SI]), \\
\dot{II} &= -2\gamma[II] + 2\tau([ISI] + [SI]), \\
\dot{SS} &= 2\gamma[SI] - 2\tau[SSI] + u_2 \cdot f_2([S], [SS]),
\end{align*}
\]

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 [8]. 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
function \(f_1\) and \(f_2\) will be specified later, but in general these will be linear or quadratic
functions describing the precise rewiring mechanisms. The parameter \(\tau\) is the per contact
infection rate and \(\gamma\) 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 organised as follows. First we consider in detail the problem of \textit{constant
control}, where the problem is effectively equivalent to a dynamic or adaptive network
problem, where the epidemic dynamics and the dynamics of the network impact on and
influence each other. Here, we will provide a classic bifurcation type analysis and we show
that there are three qualitatively different regimes: (1) disease-free steady state is stable,
(2) stable endemic state, and finally (3) stable oscillations in both epidemic dynamics and
network’s average connectivity. This is followed by the \textit{dynamic control case}, where we use
the Nonlinear Model Predictive Control Method to determine if controllability is possible
and 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 towards 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:

\[
\dot{I} = \tau[SI] - \gamma[I], \quad (2a)
\]
\[
\dot{SI} = \gamma([I]-[SI]) + \tau([SSI] - [ISI] - [SI]) - u_1[SI], \quad (2b)
\]
\[
\dot{II} = -2\gamma[II] + 2\tau([ISI] + [SI]), \quad (2c)
\]
\[
\dot{SS} = 2\gamma[SI] - 2\tau[SSI] + u_2((N-[I])(N-[I]-1) - [SS]). \quad (2d)
\]

Now instead of the variables \([SSI]\) and \([ISI]\) we are going to use the following approximations or closures [8]:

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

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:

\[
\dot{I} = \tau[SI] - \gamma[I], \quad (3a)
\]
\[
\dot{SI} = \gamma([I]-[SI]) + \tau\left( \frac{n-1}{n} \right) \left[ \frac{SS[SI]}{N-[I]} \right] - \tau \left( \frac{n-1}{n} \right) \left[ \frac{SI^2}{N-[I]} \right] - (\tau + u_1)[SI], \quad (3b)
\]
\[
\dot{II} = -2\gamma[II] + 2\tau\left( \frac{n-1}{n} \right) \left[ \frac{SI^2}{N-[I]} \right] + [SI], \quad (3c)
\]
\[
\dot{SS} = 2\gamma[SI] - 2\tau \left( \frac{n-1}{n} \right) \left[ \frac{SS[SI]}{N-[I]} \right] + u_2((N-[I])(N-[I]-1) - [SS]). \quad (3d)
\]

2.1 Dynamical behaviour

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 top panel of Fig. 1. In the Appendix we also give the detailed calculations corresponding to the stability analysis. The system is characterised
by three main behaviours as illustrated in Fig. 1 on the \((u_1, u_2)\) parameter plane, see 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 \([I](t_{final}) \neq 0\). 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, the third case is 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 being in state S. The curve of transcritical bifurcation is given by Eq. (11) (e.g. \(u_1 = \tau(N - 2) - \gamma\)) and the Hopf bifurcation set is defined in Eq. (13). Obviously, varying parameters such as \(\tau\) will not alter the qualitative behaviour, but the stability regions of the various steady states change.

A key ingredient to consider in such models is the relation between the dynamics of the epidemic and the network. The current system can be considered as an adaptive of dynamic network model [15], where the epidemic affects link deletion and creation, since these are type-dependent, and at the same time, link activation deletion can favour 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 behaviour. In the case of small values for \(\tau\) 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 re-appears at a significant level, see Fig. 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 mean time 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, 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. Obviously, this strategy requires extremely high cutting rates that are not feasible in practice, and it will not work in the constant control case and within the control horizon \(T\). To achieve this type or similar control, in the next section we consider dynamic control using the Nonlinear Model Predictive Control (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 down the network 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 potential 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 $u_1 \leq M_1$ and $|u_2| \leq M_2$.

We introduce a step size $\Delta t$ for how often we can intervene and change the amount of control, and a constant $T$ which will mark the total length of the control period. 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. Using these notations, we can define what we mean by controllability.

**Definition 1** The system is $\varepsilon$-controllable in time $T$ with step size $\Delta t$ 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

- $0 \leq u_1(t) \leq M_1$, $|u_2(t)| \leq M_2$ 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 $k = 1, 2, \ldots, \lfloor T/\Delta t \rfloor$,
- $||I|(T) - [I^*]| \leq \varepsilon$ and $|n(T) - n^*| \leq \varepsilon$.

Total controllability ($\varepsilon = 0$) would be ideal, but in most cases this is simply too much to expect from such a control scheme. In practice usually different forms of asymptotic controllability (termed asymptotic stability) is expected of NMPC algorithms, see [5] for a wide variety of examples. We study finite-time controllability, so our definition using the error term $\varepsilon$ is in good agreement with the notion of asymptotic controllability.

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

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, 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 to depend on $D$, and set $U$ to be the parameter for how many times we are to 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
| System parameters |  |
|-------------------|---|
| $N$               | size of population | 1000 |
| $\tau$           | rate of infection across a contact |  |
| $\gamma$         | 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 | $0 \leq u_1 \leq M_1$ |
| $u_2$              | rate of creating/cutting $SS$ links | $|u_2| \leq M_2$ |
| $T$               | time to end of control | $DQ$ |
| $U$              | number of intervention during $D$ |  |
| $\Delta t$        | step size for control adjustment | $D/U$ |
| $\varepsilon$     | error term |  |

| Targets to achieve |  |
|--------------------|---|
| $[I^*]$           | number of infecteds at $T$ | 0  |
| $n^*$              | average connectivity at $T$ | $n(0)$ |

| Damping parameters |  |
|--------------------|---|
| $\lambda_1$       | controlling level of infection |  |
| $\lambda_2$       | controlling jumps in $u_1$ |  |
| $\lambda_3$       | controlling average connectivity |  |
| $\lambda_4$       | controlling jumps in $u_2$ |  |

Table 1: Table summarising 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.
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 5 days or 5 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 $\Delta t$ time. This is quite a considerable amount, and hence, the maximum value $M_1$ for $u_1$ is set to $\frac{0.2}{\Delta t} = \frac{0.2}{0.4} = 0.5$, which for our simulation parameters equates to 1. Similarly, an appropriate value for $u_2$ is $\frac{0.01}{\Delta t} = 0.001$, since $u_2$ has a quadratic multiplier in terms of $N$ in the system of equations (3a)–(4), while the multiplier of $u_1$ is linear in $N$.

Our targets will be $[I^*] = 0$ and $n^* = n(0)$ describing our goal that we wish to find and apply 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 readers 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, for example controlling drug dosing, industrial plants or automobiles, see the collection of survey papers [12]. For further theoretical details on NMPC, we refer to the monograph [5].

Our aim is now to apply the NMPC method to control epidemics spread. We use a little different system than in the previous sections:

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

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

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

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

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

In order to apply the NMPC algorithm, we should first discretize the system (4)–(7). We fix a time step $\Delta t$ and observe the system only at instants $t = k\Delta t$ where $k \in \mathbb{Z}$. For
simplicity, we shall omit $\Delta t$ and write $x(k)$, $y(k)$ which means that $x$ and $y$ are evaluated at time instant $k\Delta t$. We suppose 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 we obtain the following discretized form of system \(4\)–\(7\):

\[
x(k + 1) = F(x(k), u(k)), \quad (8)
\]

\[
y(k + 1) = h(x(k + 1)), \quad (9)
\]

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 $F$ symbolizes that we solve the system of ODEs given by Eqs. \(4\)–\(7\) numerically on the interval $[k\Delta t, (k+1)\Delta t]$ and $h(x_1, x_2, x_3, x_4) = (x_1, (2x_2 + x_3 + x_4)/N)$. As we explained before, we impose the following constraints on the control signals:

\[
0 \leq u_1(k) \leq M_1, \\
-M_2 \leq u_2(k) \leq M_2.
\]

Now, the control action $u$ at time $k$ is computed as follows. We fix a prediction horizon of length $P$ steps and perform a nonlinear optimization procedure over the admissible set of future control actions as described below. Denote by $u_i(k+j|k)$ ($i = 1, 2$) an arbitrary admissible future control action at time $k + j$ chosen at time $k$. If we choose admissible sequences of future control actions $u_1(k|k), u_1(k+1|k), \ldots, u_1(k+P-1|k)$ ($i = 1, 2$), then these controls yield predicted future outputs $y_1(k+j|k), y_2(k+j|k)$ ($j = 1, 2, \ldots, P-1$), where the notation means that $y_i(k+j|k)$ is a predicted output at time $k + j$ calculated at instant $k$. More specifically,

\[
x(k+j|k) = F(x(k+j-1|k), (u_1(k+j-1|k), u_2(k+j-1|k))),
\]

\[
y(k+j|k) = h(x(k+j|k)), \quad j = 0, 1, 2, \ldots, P-1,
\]

where $x(k+j|k)$ denotes the predicted state at $k+j$ calculated at instant $k$. The setpoints for the output signals are $y_1^* = [1^*] = 0$ and $y_2^* = n^* = n(0)$, therefore, we choose the objective functional $J: \mathbb{R}^{2P} \rightarrow \mathbb{R}$ to be minimized to have the form

\[
J(u(k|k), \ldots, u(k+P-1|k)) = \sum_{j=0}^{P-1} \lambda_1(y_1(k+j|k))^2 + \lambda_2(\Delta u_1(k+j|k))^2 + \lambda_3(y_2(k+j|k) - n(0))^2 + \lambda_4(\Delta u_2(k+j|k))^2
\]

with parameters $\lambda_1, \ldots, \lambda_4$ 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 Eqs. \(4\)–\(7\). Clearly, by adjusting the parameters we can put more weight on the quadratic difference terms or penalize large control efforts that will be discussed later in details. For example, $\lambda_1$ penalises small departures from the no-epidemic state, while $\lambda_2$ penalises large changes in rewiring rates. Now performing the above nonlinear optimization problem we obtain a sequence of optimal controls $u_1(k|k), u_1(k+1|k), \ldots, u_1(k+P-1|k)$ ($i = 1, 2$). This can be done by using a nonlinear optimisation routine, such as \texttt{lsqnonlin} in \texttt{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 and the same optimization procedure is implemented to calculate the next control.

It is intuitively clear that dynamical control is more effective compared to constant control. However, the number of parameters involved in setting up or specifying dynamic control makes it non-trivial 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 $\tau$. As expected, in Fig. 3 we can see that for very small values of $\tau$ (for about $\tau < 0.05$) the infection disappears from the system even without control. However, for higher values of $\tau$ the disease becomes more widespread and the prevalence level converges towards the full population size. When no control is applied the mean degree of the system remains unchanged in each step, so naturally the final value of the mean degree is the initial value $n(0) = 10$ for each $\tau$.

Now using the NMPC 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 $\tau$. 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 Fig. 3 the prevalence and the mean degree at time $T$ for different values of $\tau$ are plotted. The figure shows that for approximatively $\tau > 0.15$ the control is ineffective, both the value of $|I|(T)$ 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 amount of infected individuals. For even higher values of $\tau$, the control has little effect on $|I|$ or $n$, 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 values of $\tau$ which cannot be said in the uncontrolled case. However, the value of $n(10)$ becomes much lower for higher values of $\tau$ despite the control.

This behaviour 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 $\tau$. For high infection rates even $SS$ edges are cut, but again with a limited strength and making little difference. In fact, this only results 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 $\tau$, a detailed numerical exploration is carried out. To carry out this exploration, we fix the damping parameters as follows $\lambda_1 = 10^4$ and $\lambda_2 = \lambda_3 = \lambda_4 = 1$. This choice penalises even a small departure from the ultimate target of disease eradication. While, we fix these, the damping parameters themselves will impact on 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 $\tau$ 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 Fig. 3. If we wish to keep $M_2 = 0.001$, we should increase the value of $M_1$. Figure 3 shows that $M_1 = 18$ makes the system controllable.

Extensive numerical simulations suggest that for a fixed value of $\tau$ and $M_2$, there is a
critical value $M^c_1$, such that if $M_1$ is lower than $M^c_1$, 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 $M^c_1$ as the bound for $M_1$. This critical value is the strictest bound admissible. In Fig. 5 (left panel) the critical value $M^c_1$ for three different values of $M_2$ is plotted as $\tau$ 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 figures 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 same applies as previously: 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.

### 3.3 Effects of $\Delta t$ and the damping parameters on controllability

In this section we analyze how the value of the step size $\Delta t$ and the damping parameters (i.e. $\lambda_i$ ($i=1,2,3,4$)) in the cost functional affect system controllability. Let us first deal with the step size. A greater value for this means a slower reaction, 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 $\Delta t \leq 5$ are effective - which means $U = 0.2$ (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 period $D$, and $U$ needs to take values less than one 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 $M^c_1 = 7.8$. In Fig. 6 the effect of control is shown for four different values of $\Delta t$. 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 $\Delta u_1$ and $\Delta u_2$ are $\lambda_2$ and $\lambda_4$, respectively. When both are large compared to $\lambda_1$ and $\lambda_3$, achieving the control target will be difficult due to small increments in the rewiring rates. As shown in Fig. 7 (continuous line), infection seems to eradicated after a large excursion into high infection levels, but network connectivity is far from the target. This is exacerbated by a magnitude difference in size of the $\lambda_1$ and $\lambda_3$, with control focused more on achieving eradication of the disease. However, when the control functional depends solely on controlling the spread, then this target is quickly achieved, but this happens at the price of the network being completely disconnected, see Fig. 7 (dashed line). When, the control adjustment is not penalised and with a stronger focus on achieving the target connectivity, the system proves to be uncontrollable since the disease cannot be eradicated at the end of the control period, see Fig. 7 (dotted line).
Table 2: 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    |

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 $\tau$, 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 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 Fig. 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 Fig. 8 it is clearly illustrated that the $n^* = 10$ cannot be achieved, see top row. However, modifying the target to $n^* = 7.5$, the system becomes controllable, see bottom row. Let us fix the parameters $M_2$ and $\tau$ above and analyse the highest possible achievable $n^*$ for different values of $M_1$. Table 2 below shows the results of some of our simulations.

4 Discussion

The control in this paper does not appear in the form of what could be termed as classic control. More precisely, classic control problems in epidemiology involve the minimisation of an integral or cost function. Here, we focus on the end target and we select the piecewise constant control signal that allows us to be as close as possible to the final target. Obviously, within the control parameters that we assume, we ignore costs and a cumulative measure of the amount of intervention and costs due to infection. This can obviously be built into further models.

While setting up the problem in this way has been a first step to bridge the gap between classic compartmental control and modern epidemiological models, it is straightforward to
apply the same methodology to more complicated settings involving costs and competing
effects, such as the trade of in cost between vaccination and the number of infectious
individuals, namely more vaccination increase the cost, but results in less infectious cases,
which in turn reduces cost. In our case this trade of was realised by aiming to control
disease spread while maintaining social cohesion. Obviously, if the network cohesion is
not required, control will lead to the trivial case of cutting the network to the point where
transmission is no longer possible. In real life this is not the case, as for 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 breaking the network of contacts
completely.

The next step for this method is to extend it to individual-based network simulations,
and work out to what extent the control predicted by the pairwise model would also trans-
late to good/optimal control in the stochastic network model. Such endeavours already
exist and the first signs are positive in that control from mean-field type models seem to
translate, at least for some cases, to the simulation counterpart \[2\].

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**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

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

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

\[
[I] = 0, \\
[SI] = 0, \\
[II] = 0, \\
[SS] = N(N - 1).
\]
Denoting the disease-free steady state as $E_d$, the Jacobian at state $E_d$ is

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

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 $2 \times 2$ submatrix:

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

The determinant of this submatrix is $2\gamma(\gamma - \tau(N-2) + u_1)$, 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, so we can exclude that. Thus our only criterion for the disease-free steady state to be stable is:

$$u_1 > \tau(N-2) - \gamma \quad (11)$$  

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

To calculate the endemic steady state(s), we first express the variable $[SI]$ from equation (10a) to get

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

Then we express $[SS]$ from equations (10b)-(10d):

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

We substitute these expressions of $[SI]$ and $[SS]$ in Eq. (10c). We obtain a quadratic equation for $[II]$, from which $[II]$ can also be expressed in terms of $[I]$:

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

where

$$A = \frac{[SI] + N-[I]}{N[SI]},$$

$$B = \gamma \cdot \frac{N-[I]}{\tau N[SI]},$$

$$C = 2[SI] + [SS],$$

$$D = (A - B \cdot C)^2 - 4B(1-A \cdot C).$$  

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 $[II]$ to equation (10b), 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 $(\ast)$. By solving equation $(\ast)$ we get the endemic steady states. Our numerical experiments show,
that there is always only one all-positive solution, 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 [15] 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 $b_3 = \text{Tr } J(E_e)$, $b_0 = \det J(E_e)$ 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 $4 \times 4$ matrices the necessary and sufficient condition for the existence of pure imaginary eigenvalues is

$$
b_0b_3^2 = b_1(b_2b_3 - b_1) \text{ and } \text{sign} b_1 = \text{sign} b_3,
$$

(12)

Thus the Hopf-bifurcation set can be defined as

$$
H = \{(u_1, u_2) \in \mathbb{R}_+^2 : \exists [I] \in [0, N] \text{ such that (\ast)}, (12) \text{ hold}\}
$$

(13)

This is a simple curve in the $(u_1, u_2)$-parameter plane. $E_e$ is stable above the curve and is unstable below. There is notable oscillation in the value of $[I]$ according to time in the unstable region.
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Figure 1: Typical system behaviours (top row) and bifurcation diagram (bottom panel) for $N = 1000$, $n(0) = 10$, $\tau = 0.1$, $\gamma = 1$ and $I(0) = 10$.

Figure 2: Time evolution of prevalence and network connectivity for $N = 1000$, $n(0) = 10$, $\tau = 0.1$, $\gamma = 1$ and $I(0) = 10$. 
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 $\tau$ 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 $\lambda_2 = \lambda_3 = \lambda_4 = 1$.

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 $\lambda_2 = \lambda_3 = \lambda_4 = 1$. 
Figure 5: Threshold plots illustrating the relation between system ($\tau$) 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 $\lambda_2 = \lambda_3 = \lambda_4 = 1$.

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 $\lambda_2 = \lambda_3 = \lambda_4 = 1$. 

19
Figure 7: Dependence of system’s controllability on the damping parameters in terms of the time evolution of prevalence and network connectivity for $\tau = 1$, $\gamma = 1$, $N = 1000$, $I(0) = 10$, $I^* = 0$, $n(0) = n^* = 10$, $M_1 = 7.8$, $M_2 = 0.5$, $\Delta t = 0.1$ and three sets of damping parameters.

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 $\lambda_2 = \lambda_3 = \lambda_4 = 1$. 