

Why do border controls fail to stabilise epidemics?

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Abstract—Can border controls and travel restrictions suppress epidemics? This is an important and recurring question for public health policymaking and pandemic preparedness. Although there is a consensus that infections imported from external locations are critical to sustaining local epidemic growth and driving the endemic equilibria of the disease, there remains ongoing debate on the effectiveness of interventions aimed at curbing importations. Most studies contributing to this debate rely on complex metapopulation models that preclude generalisable insights and formal control principles are rarely used. Here we demonstrate how classical control theory applied to an analytic but flexible and widely used transmission model provides compelling evidence that border and travel restrictions fail to stabilise spread. These restrictions always enter as precompensators and hence fail to shift the epidemic poles. Consequently, regions cannot respond to emergent outbreaks in isolation. We find that coordinating interventions across both the local and external regions converts precompensation into feedback control and derive new criteria for overall stability. We further develop formulae specifying how border controls shape performance, such as disease equilibria and total infections. During growing epidemic phases, cooperative control is crucial for stability. Once stability is assured, border control can be sufficient to meet performance targets.

I. INTRODUCTION

Although border controls, screening and travel restrictions are among the most widely implemented measures in response to outbreaks of directly transmitted infectious diseases, their effectiveness at controlling spread remains an active topic of debate [1]–[3]. During the growing and decaying phases of an epidemic, infections introduced from external regions can spur and sustain local transmission [4]. Repeated importations can drive epidemic waves in previously unaffected locations, precipitate pandemics and cause seasonal cycles among source and sink locations. While the use case for restricting mobility is clear, opinion is divided on the usefulness of these measures.

Some studies have found that even stringent border controls are ineffective against transmission and only delay epidemics by a few weeks [1], [3], [5]. Other works have linked timely travel restrictions to reduced spread [2], [6], [7], building a case for border control as a central part of epidemic response strategy. A third strand of research has questioned whether past data have been of sufficient fidelity to meaningfully infer and extricate the impact of border

control from local interventions, highlighting the value of enhanced import surveillance [3], [4]. However, there is a consensus that epidemiological modelling frameworks are a key tool for resolving this debate (even as data improve) and informing border control policy [5], [6].

The most common modelling approaches employ complex metapopulation or agent-based models [5], [8]. While valuable for detailed simulation of disease transmission, these models can be hard to validate and unsuitable for extracting generalisable insights. Here we aim to uncover the fundamental limits on what border and travel controls can and cannot achieve by applying classical control theory. Although control theory has found success in epidemiology, it has been underutilised in this field [9], [10]. By deriving new epidemic transfer functions and identifying stability and performance thresholds, we show how standard control analyses provide a definitive and testable answer about the effectiveness of border closures.

II. MODEL FORMULATION

A. Uncontrolled epidemic dynamics

We describe the transmission dynamics of a generic infectious disease using the renewal model in (1) [11]. This is a discrete-time, autoregressive process in which new infections at time t , $i(t)$, result from imported infections $m(t)$ and the convolution of past infections with a kernel $w(t)$.

$$i(t) = m(t) + R \sum_{s < t} i(s)w(t-s). \quad (1)$$

The kernel determines the distribution of times between primary and secondary infections i.e., $w(t-s)$ is the probability that it takes $t-s$ time units for an infection to be transmitted. The parameter R is the reproduction number of the disease and defines the level of branching in transmission. A single infection generates $R \sum_{s \leq d} w(s) = R$ new infections across the period d that it is infectious (after which recovery occurs and the individual is no longer infectious, $w(s > d) = 0$).

The renewal model assumes that infectious and susceptible individuals mix randomly and generalises many popular disease models (e.g., the susceptible-exposed-infected-recovered (SEIR) model) [12]. The dynamics of COVID-19, Ebola virus, SARS, measles and HIV, among others, have all been simulated by appropriately setting R and the generation time distribution $w(t)$ [13]. We focus on initial epidemic stages so that R is fixed until we apply control (i.e., the fraction of individuals infected is small relative to the total population size). We can use piecewise-constant switches in R to approximate later epidemic stages and this system is often used to determine asymptotic epidemic properties [14].

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We construct the epidemic transfer function $G(z)$ [15] by taking the Z transform of (1) with capitalised forms denoting the transformation e.g., $I(z) = \sum_{s=0}^{\infty} z^{-s} i(s)$. This yields (2), which has $W(1) = 1$ because it is the Z transform of a distribution. We also know that $RW(r) = 1$ from the Euler-Lotka relation [16] with r as the dominant pole of $G(z)$.

$$I(z) = \frac{1}{1 - RW(z)} M(z) = G(z)M(z). \quad (2)$$

This real pole is the multiplicative (asymptotic) growth rate r of the disease (this is the discrete-time form of the widely used growth rate [17]). Critical stability occurs when $z = r = 1$ i.e., the dominant pole sits on the unit circle. This corresponds to $R = 1$ because $RW(1) = 1$. The system is asymptotically stable at $r < 1$ or equivalently $R < 1$.

The relative stability of (2) is set by how close $|-RW(z)|$ is to -1 and this system has a phase crossover frequency of 0 [15] so $R^{-1}W(e^{0j})^{-1} = R^{-1}$ (the gain margin). This is the control effort required to force the epidemic to critical stability and is realisable by removing a proportion $1 - R^{-1}$ of new infections (e.g., via quarantine) over the infectious period d . This fraction relates to herd immunity and other epidemic control thresholds and leads to a controlled growth rate $r_c = 1$. The controlled reproduction number R_c is the inverse of how much we can scale infections to force critical stability and equals 1 [15]. The delay margin of (2) (when stable) is infinite. We generalise this model to include interventions next and derive stability and performance criteria.

B. Interventions on local and imported cases

The model above is idealistic because (i) we rarely observe all infections, (ii) we have not considered how interventions or control actions alter transmission (to obtain R_c) and (iii) we have ignored how infections imported into the location of interest can influence interventions. Here we incorporate (i)-(iii) to derive a more realistic controlled renewal model. A key novelty of this model is that we distinguish between control actions that limit the infections $m(t)$ imported from source regions and controls that suppress the net infections $e(t) = i(t) - m(t)$ generated in our local or sink region.

The most commonly recorded signal during an unfolding epidemic is the time series of symptomatic cases. Cases are under-reported and delayed indicators of infections [18], [19]. We let $h_e(x - s)$ be the cumulative probability that locally generated infections at time s are detected as cases by time x and define $c_e(x) = e(s)h_e(x - s)$. We describe a wide array of detection schemes as $h_e(x - s) = 1 - \sum_j \lambda_{ej}^{x-s}$, with the λ_{ej} as detection rates. This is the cumulative probability of detection under an Erlang distribution, the simplest of which is a geometric distribution with $h_e(x - s) = 1 - \lambda_e^{x-s}$.

We allow for differences in the detection of imported infections as cases because border screening or testing, for example, may be different in effectiveness from local case reporting or contact tracing. Hence, we also define $c_m(x) = m(s)h_m(x - s)$ with the geometric detection scheme $h_m(x - s) = 1 - \lambda_m^{x-s}$. As $(\lambda_e, \lambda_m) \rightarrow (0, 0)$, $(c_e(x), c_m(x)) \rightarrow (e(x), m(x))$ and detection is perfect. At

the other extreme, $(\lambda_e, \lambda_m) \rightarrow (1, 1)$ and no infections are detected. For ease of notation we write all subsequent results for the geometric distribution but note that extension to Erlang and more general discrete time phase-type distributions all follow directly and easily.

Detection is important because targeted interventions such as testing, tracing and isolation, pharmaceutical administration or quarantines only apply to observable infections. We model generic interventions as a removal of the proportions u_e and u_m of locally generated and imported cases, respectively. As a result, infections $i(s)$ are subject to control actions of effect $-u_e h_e(t - s)e(s) - u_m h_m(t - s)m(s)$. We therefore obtain the controlled renewal model of (3).

$$i(t) = m(t) + R \sum_{s < t} w(t - s)[1 - u_e h_e(t - s)]e(s) + R \sum_{s < t} w(t - s)[1 - u_m h_m(t - s)]m(s). \quad (3)$$

Maximum control effort applies $u_e = u_m = 1$ and minimum occurs when both are 0 (and recovers (1)). As far as we can tell, (3) is novel and expands on frameworks from [15], [20]. Unobserved local or imported infections transmit without any reduction as they are not identified as cases by targeted control schemes. Applying Z transforms to (3) and observing that the cumulative detection probabilities result in a time dilation of the generation time distribution we derive (4).

$$I(z) = \frac{1 + R(u_e O_e(z) - u_m O_m(z))}{1 - RW(z) + Ru_e O_e(z)} M(z). \quad (4)$$

We capture the case detection effects through $O_e(z) = W(z) - W(\lambda_e^{-1}z)$ and $O_m(z) = W(z) - W(\lambda_m^{-1}z)$. More general detection expressions require subtracting further time dilated terms involving additional detection rates (λ_{ej}) and demonstrate how detection probabilities and the generation time distribution are importantly coupled. The transfer function of (4), $G(z) = I(z)M(z)^{-1}$, is a central result defining what a region acting in isolation can achieve using targeted interventions. We analyse ramifications of (4) in the Results.

C. Interventions across source and sink locations

The framework developed above considers regions acting in isolation. Here we generalise (4) to factor in the infection dynamics within the source region. This allows us to model and assess interventions coordinated across source and sink regions. Previously, importations originated from the source. We use ϵ_{mi} , $\epsilon_{im} \in (0, 1)$ to denote the mobility of infected individuals between source and sink regions. At each time step, a proportion ϵ_{mi} of the newly infected individuals from the source migrate to the sink and a proportion ϵ_{im} of those newly infected from the sink migrate to the source. We note that $\epsilon_{ii} = 1 - \epsilon_{im}$ and $\epsilon_{mm} = 1 - \epsilon_{mi}$ are the proportions of new infections remaining in their original location.

The total number of newly infected individuals in the sink region at time t is $i(t) = \mu_{mi}(t) + e_i(t)$, with infections resulting from contagion in the sink region, $e_i(t)$, satisfying the controlled renewal equation (5). Our notation differs slightly from (3) as we use u_i to indicate the proportion of net local $e_i(t)$ infections removed and u_m for the equivalent

proportion of imported infections $\mu_{mi}(t) = \epsilon_{mi}e_m(t)$, which are removed. The cumulative detection probabilities are notated similarly and become $h_i(t-s) = 1 - \lambda_i^{t-s}$ and $h_m(t-s) = 1 - \lambda_m^{t-s}$ when detection is geometric. We use $R_i = \epsilon_{ii}R$ and $R_m = \epsilon_{mm}R$ to simplify formulae.

$$e_i(t) = R_i \sum_{s < t} w(t-s)[1 - u_i h_i(t-s)]e_i(s) + R_i \sum_{s < t} w(t-s)[1 - u_m h_m(t-s)]\mu_{mi}(s). \quad (5)$$

As we describe the dynamics of both source and sink regions, (5) is now accompanied by an additional controlled renewal model describing the net local infections generated at the source $e_m(t)$. This yields (6) with $\mu_{im}(t) = \epsilon_{im}e_i(t)$.

$$e_m(t) = R_m \sum_{s < t} w(t-s)[1 - u_m h_m(t-s)]e_m(s) + R_m \sum_{s < t} w(t-s)[1 - u_i h_i(t-s)]\mu_{im}(s) + b(t). \quad (6)$$

Because all source infections are subject to the same detection and control (i.e., imports in (5) are controlled in the source region), $\mu_{mi}(t)$ faces detection $h_m(t-s)$ and removal proportion u_m . Similarly, the exports from the sink $\mu_{im}(t)$ that enter the source region have been subject to $h_i(t-s)$ and u_i , with $b(t)$ as initial input infections to start the epidemic.

We take Z-transforms of our coupled renewal models to derive the matrix transfer function $\mathbf{G}(z)$ as in (7) where $I(z)$ and $M(z)$ are transforms of the total infections generated in the source and sink regions respectively.

$$\mathbf{I}(z) = \begin{bmatrix} I(z) \\ M(z) \end{bmatrix} = \begin{bmatrix} 1 & \epsilon_{mi} \\ \epsilon_{im} & 1 \end{bmatrix} \mathbf{G}(z) \begin{bmatrix} 0 \\ B(z) \end{bmatrix}. \quad (7)$$

Here $\mathbf{G}(z) = (\mathbb{I} - \mathbf{Q}(z))^{-1}$, with $\mathbf{Q}(z)$ in (8) and identity matrix $\mathbb{I}$. The diagonal terms of $\mathbb{I} - \mathbf{Q}(z)$ are similar to the denominator of our isolated region transfer function in (4).

$$\mathbf{Q} = \begin{bmatrix} R_i(W - u_i O_i) & \epsilon_{mi} R_i(W - u_m O_m) \\ \epsilon_{im} R_m(W - u_i O_i) & R_m(W - u_m O_m) \end{bmatrix}. \quad (8)$$

We examine the stability and performance of these equations in the Results to assess the coordinated interventions.

III. RESULTS

A. Border controls cannot stabilise epidemics

Equation (4) defines precisely how imports drive the new infections in our region of interest and reveals why border closures or travel controls cannot stop epidemic growth. No import control terms feature in the denominator of (4), despite the u_m and $h_m(t-s)$ factors directly reducing ongoing and future transmission as in (3). This effect is obvious from the block diagram of Fig. 1A with $E(z) = I(z) - M(z)$ and controllers $K_i(z)$ and $K_m(z)$ appropriately set to realise (4). Import control only alters the precompensator $K_m(z)$.

Further, as (4) is a linear system, it also possesses bounded input-bounded output (BIBO) stability provided its poles are inside the unit circle or equally the controlled reproduction number $R_c < 1$. As a result, if $m(t)$ decays in finite time i.e., is transient, then $i(t)$ decays regardless of the import control applied. Alternatively, if $m(t)$ is growing, then import control

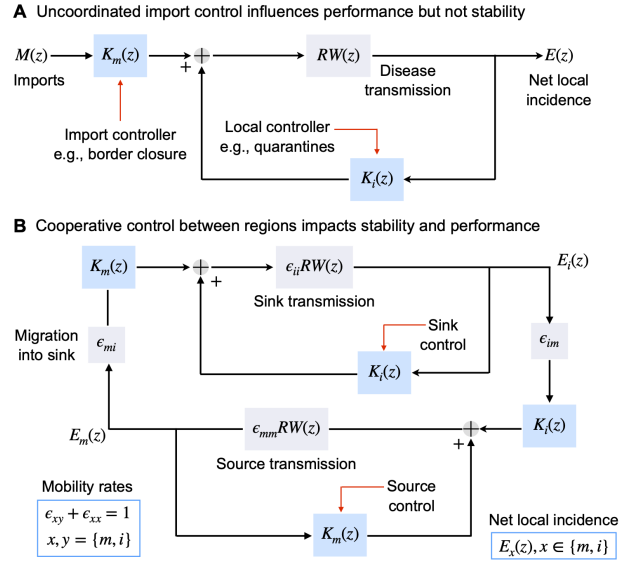


Fig. 1. Control system architectures for managing imported and local infections. A: from the viewpoint of the local region with net local (new) infections $E(z)$, imported infections $M(z)$ are disturbance inputs and border controls $K_m(z)$ always act as precompensators. This fundamentally limits the impact of $K_m(z)$ to shaping performance. Local measures $K_i(z)$ such as test-trace-isolate schemes always act in feedback with $E(z)$ and define stability. The controller forms are chosen to realise (4) with $I(z) = E(z) + M(z)$. B: under cooperative schemes $K_m(z)$ also becomes a feedback controller for the source region, which has total infections $M(z) = E_m(z) + \epsilon_{im}E_i(z)$. This also implicitly adds an import control term for the sink region. Symmetrical constructions apply to the sink region with total infections $I(z) = E_i(z) + \epsilon_{mi}E_m(z)$. The migration parameters mediate how infections are distributed. This structure with $(K_m(z), K_i(z))$ providing feedback control presents better (and likely more economical) solutions to suppressing the overall epidemic. We set the same R and $W(z)$ for both regions for clarity, but this is easily generalised without issue. Controllers are formulated to realise (7) (but we do not show $B(z)$).

cannot stop $i(t)$ from growing, even at $R_c \ll 1$. The only exceptions are unrealistic and trivial, requiring $R_c = 0$ or that all imports be removed upon arrival.

The poles of (4) satisfy $Ru_e O_e(z) = RW(z) - 1$. When $z = 1$ solves this expression (as the dominant pole), the system is critically stable. Substituting this condition, we find $R_c = R(1 - u_e O_e(1))$ and a stability criterion of (9), linking detection and control to a herd immunity-type threshold H .

$$u_e O_e(1) \geq 1 - \frac{1}{R} = H. \quad (9)$$

Hence, only the local controller $K_e(z)$ of Fig. 1A determines epidemic stability. The import controller has no influence on (9) but is important for shaping performance in the local region of interest as $K_m(z)$ controls the zeros of (4).

We define performance, given $R_c < 1$, as the steady-state infection count $i(\infty)$ if $m(t)$ is persistent, also known as the endemic equilibria, or the total new infections $\sum_t i(t)$ if $m(t)$ is transient (decaying), also termed the epidemic final size. Both quantities are of frequent public health importance. We compute the endemic state under a step input of size $m(\infty)$ by using the final value theorem. Setting a target ratio

$\phi = i(\infty)m(\infty)^{-1}$, we derive performance threshold (10).

$$u_e O_e(1) \geq H + \frac{1 - u_m O_m(1)}{\phi - 1}. \quad (10)$$

Equation (10) reveals that we must apply local control above that needed for stability from (9) by an amount based on our performance target (larger ϕ are easier to achieve) and our import control. We also find that (10) exactly describes final size performance with target $\phi = (\sum_t i(t))(\sum_t m(t))^{-1}$. $O_x(1) \leq 1$ and $u_x \leq 1$ are detection and control strengths.

Interestingly, epidemic models admit another endemic equilibria at the critical $R_c = 1$, when driven by transient importations. This system has subexponential growth if imports are persistent. Applying the final value theorem, we get $i(\infty)(\sum_t m(t))^{-1} = (1 - u_m O_m(1))(g + H O_e(1) \frac{dO_e(1)}{dz})^{-1}$ with $-\frac{dW}{dz}(1) = g$ as the mean generation time. While not directly comparable to (10) as $u_e O_e(1)$ is set at exactly H , we see that import control is crucial to reducing endemic infections via a factor $1 - u_m O_m(1)$ as in (10).

We explore and verify the above stability and performance thresholds for Covid-19, Ebola virus and Marburg in Fig. 2. Fig. 2A presents simulated epidemics with local control such that $R_c < 1$ and persistent step, sinusoidal and random import time series, with the same steady-state mean. Import control substantially shapes performance, not only reducing endemic equilibria, but also damping peaks and oscillations. Fig. 2B plots how detection delays (mean $o_e = \lambda_e(1 - \lambda_e)^{-1}$) can destabilise the control effort u_e required to achieve a specific R_c as in (9). Import control cannot shift these curves. Fig. 2C simulates epidemics at $R_c = 1$ with decaying step, sinusoidal and random imports, validating how import control also shapes this second type of endemic state.

In Fig. 2D we illustrate from (10) how import control can help reduce the local control effort needed to achieve a desired performance target ϕ . Perfect import control ($u_m O_m(1) \rightarrow 1$) merges the control and performance criteria to H and makes explicit that a region acting in isolation, once stable, can limit importations to improve public health outcomes. This is an important point (in simulations u_m can even halve final sizes or endemic loads), suggesting that border closures and travel controls can be valuable tools for tuning performance.

Last, we show that the failure of border controls to stabilise epidemics is robust to important model variations. Specifically, (3) assumes importations acquire the transmission characteristics of the region they enter. This is a sink assumption. We may instead let importations maintain the transmission characteristics of the source i.e., reproduction number R_m and generation time distribution $w_m(t)$. Under this source assumption, the numerator of (4) becomes $1 + (R_m W_m(z) - R_m u_m O_m(z)) - (R_m W_m(z) - R_e u_e O_e(z))$ but the denominator is unchanged. Consequently, even when the import region has $R_m \gg R$, we cannot stabilise the epidemic with border controls. These points also hold for more complex source architectures (e.g., imports from multiple sources in series or parallel) because import control is always a precompensator for a BIBO system.

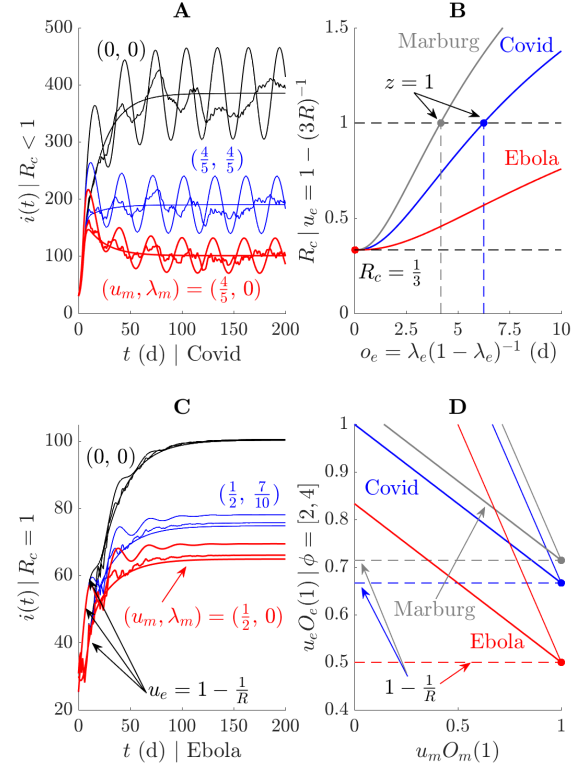


Fig. 2. Performance and stability thresholds for uncoordinated strategies. A: epidemic trajectories $i(t)$ simulated under Covid-19 parameters (from [21]) with stabilising local control ($R_c < 1$) and differing import control u_m and detection λ_m parameters. Persistent sinusoidal, step and random $m(t)$ inputs are applied and responses are coloured by various import control settings. B: stability thresholds at local control u_e achieving $R_c < 1$ showing how geometric detection delays with mean o_e cause instability for various diseases (Ebola and Marburg parameters from [22]). These thresholds must be satisfied before focussing on performance as in A and C. Note that if the source region is unstable then these thresholds are not sufficient because imported infections are unbounded. C: epidemic trajectories simulated under $R_c = 1$ with Ebola parameters, coloured by different import control choices. Transient (decaying) sinusoidal, step and random $m(t)$ inputs are applied and represent another way of attaining equilibria (as opposed to A). D: performance thresholds for achieving steady state and total infection ratios ϕ when $R_c < 1$ for various diseases. Import control i.e., u_m and $O_m(1)$ tuning, reduce how much local effort from u_e and $O_e(1)$ is needed above the stability threshold of B to achieve performance goals. Thinner lines are for the larger ϕ target, which requires smaller control effort. In all simulations of A and C, we run the uncontrolled model for a week before applying control.

B. Cooperative control provides stability and performance

As border and travel restrictions cannot stabilise infections, the only way to suppress epidemics is via cooperative source and sink control. The limitations of precompensation mean that if the source is unstable, there is no way for the sink region (acting alone) to stabilise infections. However, we can convert this precompensation effort into feedback control by considering sink and source regions jointly. This becomes apparent in Fig. 1B, which realises (5)-(6) and shows how net local infections in the sink $E_m(z)$ and source $E_i(z)$ are fed back to a suitable chosen $K_m(z)$ and $K_i(z)$.

The poles of the transfer matrix $\mathbf{G}(z)$ from (7)-(8) control

the overall stability of the system $(i(t), m(t))$ and solve $\det(\mathbb{I} - \mathbf{Q}(z)) = 0$. This yields the characteristic polynomial $\Delta(z)$ of (11), generalising the denominator of (4).

$$\Delta(z) = (1 - R_i(W - u_i O_i))(1 - R_m(W - u_m O_m)) - \epsilon_{mi} \epsilon_{im} R_i R_m (W - u_i O_i)(W - u_m O_m). \quad (11)$$

If either of the migration coefficients $\epsilon_{mi}, \epsilon_{im}$ equals 0, the poles are exactly the union of poles of the isolated systems, and we recover the stability condition $\Delta(1) = 0$ in (12).

$$(1 - R_i(1 - u_i O_i(1)))(1 - R_m(1 - u_m O_m(1))) = 0. \quad (12)$$

This produces the herd immunity type thresholds of $H_i = 1 - (\epsilon_{ii} R)^{-1}$ and $H_m = 1 - (\epsilon_{mm} R)^{-1}$, which are analogues of (9). If both migration terms equal 0, we recover $H_i = H_m = H$, where H is the threshold found in (9) in the absence of cooperative control. Importantly, when migration terms are non-zero, which is also necessary for μ_{mi} and μ_{im} to be realistic, coordinated interventions between regions can shift these thresholds and offer improved stability bounds.

We demonstrate this in Fig. 3A. When $u_i O_i(1) \geq H$ and $u_m O_m(1) \geq H$, the uncoordinated locations are stable ($\Delta(1) = \epsilon_{mi} \epsilon_{im} (1 - \epsilon_{mm} \epsilon_{ii}) > 0$). However, the coordinated domain of stability extends beyond these thresholds, being bounded by the hyperbola $\{\Delta(1) = 0\}$, which has the lines $\{u_i O_i(1) = H_i\}$ and $\{u_m O_m(1) = H_m\}$ as asymptotes. As a result, for given levels of detection λ_i and λ_m , cooperation between the source and sink region allows for smaller proportions of infections to be removed (and likely smaller intervention costs) while maintaining stability. In isolation, these control levels would result in unstable dynamics.

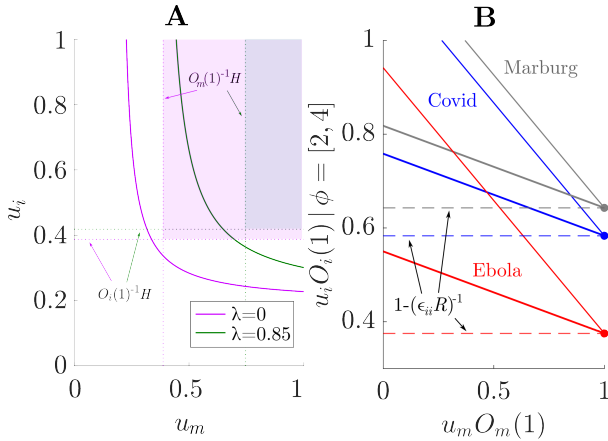


Fig. 3. Performance and stability thresholds for coordinated strategies. A: Stability threshold curves $\{\Delta(1) = 0\}$ for $R = 1.6$, $\epsilon_{mi} = \epsilon_{im} = 0.2$, with $\lambda_i = \lambda_m/2$ to illustrate a system with higher detection delays in the sink region. The asymptotes of the stability curves are the herd immunity thresholds H_i and H_m for the isolated systems when one of the $\epsilon_{mi}, \epsilon_{im}$ equals 0. Shaded regions represent the stability regions for the system without cooperative control given by (4). Gaps between a curve and its corresponding shaded region highlight the benefits of cooperative control. B: performance thresholds for achieving steady-state and total infection ratios ϕ for various diseases, as in Fig. 2. Stability regions (as in panel A) and threshold curves are much lower than in the equivalent uncooperative system, indicating (provided stability is achieved) that cooperative schemes can achieve desired performance with lower control effort.

This improvement becomes more appreciable (i.e., the asymptotes are more distant from the isolated thresholds) as detection delays become larger. Moreover, as $O_x(z) = W(z) - W(\lambda_x^{-1} z)$ for $x \in \{i, m\}$, is increasing in λ_x , (11) reveals that for a specific mobility structure $\epsilon_{im}, \epsilon_{mi}$ and fixed transmission parameter $R > 0$, outbreaks can also be stabilized by decreasing the delays in detection, even at fixed control levels u_i and u_m . The benefits of coordination on stability are consequently stark.

We can also assess performance, from the perspective of the sink region, by considering the steady-state infection count $i(\infty)$ under a step input $b(t)$. This can be computed using the final value theorem to derive (13).

$$\begin{aligned} \begin{bmatrix} i(\infty) \\ m(\infty) \end{bmatrix} &= \begin{bmatrix} 1 & \epsilon_{mi} \\ \epsilon_{im} & 1 \end{bmatrix} (1 - \mathbf{Q}(1))^{-1} \begin{bmatrix} 0 \\ 1 \end{bmatrix} = \\ &= \frac{1}{\Delta(1)} \begin{bmatrix} 1 & \epsilon_{mi} \\ \epsilon_{im} & 1 \end{bmatrix} \begin{bmatrix} \epsilon_{mi} R_i (1 - u_m O_m(1)) \\ 1 - R_i (1 - u_i O_i(1)) \end{bmatrix}. \end{aligned} \quad (13)$$

We set a target $\phi > 1$ for the ratio of infections in the sink region to the number of incoming infections from the source region, in line with (10). Considering only those infections that remain in the sink region and that only a fraction ϵ_{mi} of the total steady-state infections in the source region migrate to the sink, this means that we need to set controls such that $(\epsilon_{ii} i(\infty)) / (\epsilon_{mi} m(\infty)) = \phi$. Given the explicit solution from (13), we represent the necessary control levels in Fig. 3B for contagion levels associated to different diseases.

Again, in the limit of perfect control in the source region ($u_m O_m(1) \rightarrow 1$), for all target ratios ϕ , the necessary level of control in the sink region converges to the minimal level $H_i = 1 - (\epsilon_{ii} R)^{-1}$ that still guarantees stability. Remarkably, we find that the performance curves in Fig. 3B are uniformly lower than their equivalent in Fig. 2D (within the bounds of stability). This conclusively illustrates that cooperative control in interconnected regions not only improves global stability of the system, but also achieves similar levels of performance at lower control efforts. These benefits are important given the high financial and societal costs of epidemic control measures.

IV. DISCUSSION

We have demonstrated conclusively that border controls and travel restrictions cannot stabilise epidemics, simply because they do not alter the dominant pole of the epidemic. Regions acting in isolation can only meaningfully reduce spread by implementing local interventions that remove a proportion of infections above a herd immunity-type threshold H . However, even these measures fail if the importations originate from an unstable region (due to the limits of BIBO stability). Consequently, overall stability is best achieved by coordinating control strategies among interacting regions.

This supports more complex models from [8] suggesting that countries should share resources (e.g., vaccine stockpiles) and cooperatively respond to epidemics. We found that joint control actions reduce the overall control (or detection) effort required for stability and performance. Cooperative strategies notably expand the parameter space for stability

(Fig. 3A), meaning that lower levels of control effort in both source and sink regions can collectively stabilise the system, even under conditions where the isolated regions would remain unstable.

Although border closures fail to suppress outbreaks, they can be valuable, provided stability is achieved. The aim then becomes to minimise total and endemic infection loads. Achieving such performance usually requires local interventions of strength substantially above H . Border controls can make this problem considerably easier and even restore the lower bound of H (Fig. 2D). These benefits are realised both by isolated and cooperative strategies, but achieving performance targets demands less stringent measures in each location when acting jointly compared to isolated action (Fig. 3B). Similar phenomena have been discovered in models of networked agents [23], but had not yet been made explicit using control theory applied to generalised epidemic systems.

Several simplifying assumptions underlie these results. First, we neglected all stochasticity. During early epidemic stages, imported infections can randomly fade out (failing to trigger epidemics) or generate unexpectedly large outbreaks (superspreading) [24]. While these are key aspects of importation dynamics, they likely only set the timing of epidemic waves. Once an epidemic takes off we expect our analyses to be a fair approximation of dynamics. Second, we do not consider the non-linear effect of susceptible depletion. However, this is mostly an issue around the epidemic peak and model linearisation is common for initial and late epidemic stages. Last, we do not model the additional and hard-to-quantify feedback loops between control actions and population behaviours. This is a non-trivial effect (e.g., behaviour can alter import patterns [25]) that we aim to incorporate in future renewal models.

More broadly, the interaction between migration and heterogeneous control policies that are applied across different locations is a complex issue, which we have viewed through the simplifying yet insightful lens of two interacting locations. For larger systems with more complicated migration matrices, identifying the combinations of local interventions and border controls that achieve stability and performance goals becomes even more challenging [26], [27]. Devising optimal cooperative strategies in such settings remains an open question that we aim to explore further using the foundations laid by our controlled renewal approach, with the goal of providing guidance for pandemic management across interconnected regions.

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