Transition from growth to decay of an epidemic due to lockdown

Hamid Khataee,^{1,*} Jack Kibble,¹ Istvan Scheuring,^{2,3} Andras Czirok,^{4,5} and Zoltan Neufeld¹

¹School of Mathematics and Physics, The University of Queensland, St. Lucia, Brisbane, Queensland, Australia; ²Evolutionary Systems Research Group, Centre for Ecological Research, Budapest, Hungary; ³MTA-ELTE Theoretical Biology and Evolutionary Ecology Research Group and ⁴Department of Biological Physics, Eotvos University, Budapest, Hungary; and ⁵Department of Anatomy and Cell Biology, University of Kansas Medical Center, Kansas City, Kansas

ABSTRACT We study the transition of an epidemic from growth phase to decay of the active infections in a population when lockdown health measures are introduced to reduce the probability of disease transmission. Although in the case of uniform lockdown, a simple compartmental model would indicate instantaneous transition to decay of the epidemic, this is not the case when partially isolated active clusters remain with the potential to create a series of small outbreaks. We model this using the Gillespie stochastic simulation algorithm based on a connected set of stochastic susceptible-infected-removed/recovered networks representing the locked-down majority population (in which the reproduction number is less than 1) weakly coupled to a large set of small clusters in which the infection may propagate. We find that the presence of such active clusters can lead to slower than expected decay of the epidemic and significantly delayed onset of the decay phase. We study the relative contributions of these changes, caused by the active clusters within the population, to the additional total infected population. We also demonstrate that limiting the size of the inevitable active clusters can be efficient in reducing their impact on the overall size of the epidemic outbreak. The deceleration of the decay phase becomes apparent when the active clusters form at least 5% of the population.

SIGNIFICANCE Restricting movement and interaction of individuals has been widely used in trying to limit the spread of coronavirus disease 2019 (COVID-19); however, there is limited understanding of the efficiency of these measures, as it is difficult to predict how and when they lead to the decay of an epidemic. In this article, we develop a mathematical framework to investigate the transition to the decay phase of the epidemic, taking into account that after lockdown, a large number of active groups remained with the potential to produce localized outbreaks, affecting the overall decay of infections in the population. Better understanding of the mechanism of transition to the decay of the epidemic can contribute to improving the implementation of public health control strategies.

INTRODUCTION

Quantitative epidemiological models are being used as a tool to inform public health decisions about infectious disease outbreaks, like recently during the coronavirus disease 2019 (COVID-19) pandemic. Simpler mathematical models were useful to introduce concepts such as the basic reproduction number R_0 (1), whereas more complex agent-based models were used to estimate hospital occupancy or the effect of planned social distancing measures (2–4). Arguably the simplest and most influential epidemic model operates with the susceptible, infected, and removed/recovered

subpopulations and hence referred to as the SIR model (5). The corresponding system of nonlinear ordinary differential equations (ODEs) exhibits an initial exponential spread of an epidemic, followed by saturation as the susceptible proportion of the population decreases. Eventually, as the immune subpopulation reaches a critical threshold, often referred to as herd-immunity threshold, the infected subpopulation decays to 0.

The simplest SIR model assumes that the epidemic parameters (e.g., the probability of transmission from an infected to a susceptible individual) remain constant during the outbreak, and the whole population is perfectly mixed: any two individuals have the same chance for disease transmission. However, in the COVID-19 pandemic, a range of public health measures were introduced with the goal of reducing transmission efficiency. Accordingly, the initial

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*Correspondence: h.khataee@uq.edu.au

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exponential growth phase of the infectious fraction of the population was followed by an exponential decay after the introduction of social distancing measures, primarily reflecting the reduced transmission rate well before a significant fraction of immune population would have accumulated (6).

Detailed analysis of epidemic and mobility data from various European countries during the first wave of the COVID-19 pandemic has revealed that after the introduction of social distancing measures the transition from growth to decay phase may involve a fairly long, and country-specific delay (6). The 4- to 5-day-long incubation period of the disease (7,8), as well as the average survival time of patients with fatal COVID-19 illness are expected to introduce a certain delay between the alteration of social behavior and its observable effect in epidemic data, such as the daily new infections and death rate. However, in most countries the delay between the introduction of control measures to the peak of the epidemic was significantly longer, usually lasting for several weeks. The same study also revealed that the delay was longer in countries where the control measures were less strict (6).

Within the context of an SIR model, a substantial sudden reduction of the infection transmission rate due to social distancing efforts should flip the exponential growth into exponential decay instantaneously. In this article, we demonstrate that the delay τ between the introduction of social distancing measures and the onset of the decaying phase of an epidemic can arise when control measures affect various groups of society highly selectively. For example, under official lockdown orders, transmission can be reduced for the majority of the population, although the disease remains able to spread among various distinct clusters—like workers in warehouses, meat-processing plants, or among prison inmates. To study this effect, we propose a generalization of the SIR model to take into account the heterogeneity of the population with respect to epidemic control measures. We consider a dual population structure in which most of the population has a strongly reduced rate of transmission, although the transmission rate remains high within distinct clusters of a certain size. By stochastic simulations, we explore how the delay τ , the decay rate, and the total number of infected people are influenced by the size of the nonrestricted subpopulation as well as the typical size of the clusters within the nonrestricted population.

METHODS

In this section, we develop a mathematical model that uses the Gillespie stochastic simulation algorithm (9–11) to study the transition of an epidemic from growth to decay in a heterogeneous population, in which the public health control measures (lockdown strategies) were implemented. The heterogeneity of the population is represented as two subpopulations: the locked-down population (in which the spread of infection decays because

of effective health measures) and the rest of the population as a large set of small clusters (in which the infection may spread). In each subpopulation, the spread of the epidemic is modeled using the standard SIR epidemic model. Thus, the total population is modeled as a connected set of SIR networks. Finally, the Gillespie algorithm uses the probabilities of transmission of infections between the subpopulations to simulate the time evolution of the infection in the population. A detailed description of these steps is provided below.

To model the spread of the epidemic in a population, we use the standard SIR epidemic model, defined by the following system of ODEs (5,12):

$$\frac{dI}{dt} = kSI - \gamma I, \quad \frac{dS}{dt} = -kSI, \quad \frac{dR}{dt} = \gamma I, \quad (1)$$

where I , S , and R denote the infected, susceptible, and recovered fractions of the population, respectively; i.e., $S + I + R = 1$. Here, k is the rate of transmission from I to S per infected individual, whereas γ represents the rate of recovery from I to R , either through acquired immunity or death. The behavior of the model is determined by a single nondimensional parameter, the reproduction number $R_0 = k/\gamma$, which gives the number of new infections caused by a single infected person within a fully susceptible population. The condition for an epidemic outbreak is $R_0 > 1$, whereas for $R_0 < 1$, the epidemic dies out.

In the initial phase of an epidemic (i.e., before the lockdown), most of the population is susceptible and the number of infected individuals grows exponentially. We are interested in the epidemic dynamics within a population under social distancing measures that have been implemented sufficiently early (i.e., $I, R \ll 1$, and $S \approx 1$) and are efficient enough to reduce the reproduction number below 1. In this regime, Eq. 1 simplifies to $dI/dt = kI - \gamma I$ and yields:

$$I \approx e^{(R_0-1)\gamma t}, \quad (2)$$

where parameters $R_{01} > 1$ and $R_{02} < 1$ characterize the initial growth and postintervention decay phases of I , respectively. Eq. 2 will be used for the comparison of the results with the case where there are no active clusters in the population.

To model the heterogeneity of the population with respect to epidemic control measures, we divide the total population of size T into subpopulations: 1) the locked-down population fraction of size $0 < L < 1$, in which the social distancing measures are effective and the infection transmission rate reduces ($R_0 < 1$), and 2) the rest of the population (e.g., nursing home inhabitants, prison inmates, essential service providers) are sorted into partially isolated clusters, and within the clusters, the infection transmission rate remains unaffected ($R_0 > 1$). For simplicity, we assume that each cluster has the same size, M , a model parameter that reflects both the organization of social institutions as well as potential efforts to control the epidemic. The number of such clusters is N , and thus, the size of the non-socially-distancing subpopulation is $NM = (1 - L)T$.

The locked-down subpopulation and each cluster has its own SIR dynamics, which are also coupled; see Fig. 1 A. Specifically, we define the transmission rates as follows. Within the locked-down subpopulation (L), we define $k_{L \rightarrow L} = R_{02}\gamma$. Thus, in this subpopulation, in the absence of external interactions, the number of infections would decay exponentially. In contrast, for each cluster $j = 1, \dots, N$, we assume that the infection transmission rate within the cluster remains unchanged, i.e., $k_{j \rightarrow j} = R_{01}\gamma$. Transmission rates are also reduced between clusters and the locked-down subpopulation: $k_{L \rightarrow j} = k_{j \rightarrow L} = R_{02}\gamma$. Finally, we also assume that there is no direct transmission of infection between different clusters.

We can now establish the dynamics for each of the SIR subsystems. For each cluster j , new infections occur at the rate given by:

$$Q_j = MI_j\beta_j k_{j \rightarrow j} S_j + LTI_L \frac{M}{T} k_{L \rightarrow j} S_j, \quad (3)$$

FIGURE 1 SIR model. (A) SIR networks in the locked-down (green) and not-socially-distancing (red) subpopulations. The latter is composed of N clusters, each has its own SIR dynamics. S, I , and R are the proportions of susceptible, infected, and recovered individuals, respectively. Dashed curves represent infections transmitted by infected individuals to the susceptible individuals. (B) The total number of infected individuals versus time calculated using the Gillespie stochastic simulation algorithm. Solid curves represent five example runs of the algorithm (in different colors) for $N = 100$ clusters of size $M = 10$. Black dashed line represents $I_{tot}(t)$, average of 10 runs of the algorithm. Other simulation parameters are summarized in Table 1. To see this figure in color, go online.

where I_j and S_j are the proportions of infected and susceptible in the cluster, the first term represents new infections transmitted within the cluster, and the second term denotes new infections introduced from the locked-down population. The parameter $0 < \beta_j < 1$ represents the probability that an individual in a cluster j interacts with others within her own cluster (we use $\beta_j = 0.9$). In the second term, the factor M/T captures the probability that an individual of the locked-down subpopulation interacts with a member of a cluster j .

Likewise, in the locked-down subpopulation, the total rate of new infections is given by:

$$Q_L = LTI_L Lk_{L \rightarrow L} S_L + \sum_{j=1}^N MI_j \beta_L k_{j \rightarrow L} S_L \quad (4)$$

where $\beta_L = 1 - \beta_j = 0.1$ represents the probability of interaction between individuals of a cluster j and the locked-down subpopulation, and I_L and S_L are the proportions of infected and susceptible individuals in the locked-down subpopulation, respectively. The first term describes the transmission within the locked-down subpopulation, and the second term denotes the infections originated from the interaction of individuals in the clusters with the locked-down subpopulation.

Now, the dynamics of the proportion of infected individuals in the locked-down subpopulation (I_L) and in the active clusters (I_j) are determined by the normalized rates of new infections combined with the rate of recovery, $Q_L/LT - \gamma I_L$ and $Q_j/M - \gamma I_j$, respectively. Note, although the SIR dynamics is the same for each cluster j , the temporal dynamics differs as the first in-cluster infection transmission could be initiated at various time points.

In the case of the standard SIR model, it is natural to use a deterministic description because in a large population the fluctuation of individual discrete events can be neglected, and the overall dynamics is well approximated by continuous variables following mass action type kinetics. However, in our model, although the total population is still assumed to be large (for concreteness, we consider a city with total population $T = 10^6$), after implementing lockdown strategies, the size of partially isolated clusters (in which the infection is still able to propagate) are relatively small; we will assume typically values in the range $M = 20$ – 150 . Thus, a single newly infected person in a cluster can increase I_j by a much larger extent than a similar infection in the larger population increases I_L . Therefore, the

discreteness of the population and the stochastic nature of the epidemic dynamics is important, especially at the initiation of epidemic outbreaks within the clusters, triggered by a single random infection event.

We model the time evolution of the interacting system of subpopulations as a series of discrete events in continuous time using the Gillespie stochastic simulation algorithm (9–11). The steps taken by this discrete event simulation algorithm are as follows:

- Step 1: Set the initial number of the infected individuals to 667 and the simulation time to 0; see Table 1.
- Step 2: Calculate the current probabilities of the transmissions, given by the normalized Q_j and Q_L ; see Eqs. 3 and 4.
- Step 3: Using the probabilities from Step 2, calculate the time to the next transmission event and update the simulation time. Note, the larger the transmission probabilities, the shorter the time to the next event (9).
- Step 4: Update the total number of infected individuals by adding the number of infected in the locked-down and active subpopulations (i.e., I_L and I_j).
- Step 5: If the simulation time is less than the end time, return to Step 2.

The parameter values used in the simulations are summarized in Table 1. We used $R_{01} = 3$ and $R_{02} = 0.6$ because the estimations of R_{01} and R_{02} are in the ranges 2–3.5 (13–15) and 0.36–0.82 (6), respectively. Although the exact value of γ for COVID-19 is uncertain (16,17), we used $\gamma = 0.1$ (day^{-1}) as a reasonable estimate for this parameter (18). Yet, our analysis of the epidemic dynamics is rather general and does not rely on the values of the model parameters. Stochastic trajectories of the total number of infected individuals are shown in Fig. 1 B. The typical time course of the epidemic (I_{tot}) was obtained as an average over 10 runs of the algorithm for each set of parameters N and M ; see Fig. 1 B.

RESULTS

We first explore how the time course of the total number of infected individuals in the whole population, $I_{tot}(t)$, is affected by the number and size of the clusters which remain active after the lockdown. We find that raising the number N and size M of these clusters can lead to a dramatic increase in the number of infections in comparison to the reference case when the whole population is locked down; (dashed green line in Fig. 2, A–I). This difference is caused by a series of small outbreaks happening randomly when the infection is transmitted into some of the active clusters, whereas the overall level of infection decays in the rest of the population. The aggregate effect of the isolated outbreaks can be a delayed transition to the decaying phase of the epidemic relative to the time when the lockdown is implemented, in addition to a slower decay compared with what would be predicted based on the reproductive number R_{02} of the locked-down population. Results in Fig. 2, A, D, and G also show that increasing the number of the active clusters has a negligible effect when the size of the clusters is small. This suggests that dividing the active subpopulation into groups with smaller sizes, independent of the number of groups, can reduce the total number of infected individuals in the population. These effects of the active clusters on the decay phase become significant when the active clusters form at least 5% of the population, with $N \approx 500$ clusters of size $M \approx 100$.

The significant differences observed in Fig. 2, A–I in the number of infected individuals with varying the numbers

TABLE 1 Model parameters

Parameter	Value
Total population size, T	1,000,000
Initial reproductive number, R_{0i}	3 (13–15)
Reproductive number after lockdown, R_{0z}	0.6 (6)
Recovery rate, γ	0.1 (day ⁻¹) (18)
Initial ratio of infected individuals, I_{0i}	667×10^{-6}
Time of lockdown, t_{LD}	10 (days)

and sizes of the active clusters in the population persuade us to further examine the transition to the decay of the epidemic. We characterize the transition by three properties: the decay rate of the number of infected individuals (α), the delay in the decay phase caused by the presence of the active clusters (τ), and the additional number of infected individuals (I_{extra}) due to the slower and delayed decay of the epidemic.

The decay rate is calculated by fitting $I_{\text{tot}}(t) \approx I_{0z} e^{-\alpha t}$ to the simulation data (averaged over 10 repeated realizations) within a selected range in which there is a well-defined exponential decay (see Figs. 2, A–I and 3 A, orange circles and red curves). Then, the fit parameter α represents the decay rate (slope on the log-linear plot) of the number of infected individuals in the population.

Next, the delay τ of the onset of the exponential decay phase, caused by the not locked-down clusters, is calculated as the time-shift relative to decay with the same rate α passing through $I_{LD} = I_{\text{tot}}(t_{LD})$; see Fig. 3 A. This decay follows $I_{0z} e^{-\alpha(t+\tau)}$ (illustrated using dotted brown line in Fig. 3 A). Therefore, $I_{LD} = I_{0z} e^{-\alpha(t_{LD}+\tau)}$ results in the following equation:

$$\tau = -\frac{1}{\alpha} \ln\left(\frac{I_{LD}}{I_{0z}}\right) - t_{LD} \quad (5)$$

Finally, the number of new additional infected individuals, caused by the presence of not locked-down clusters, is calculated as,

$$I_{\text{extra}} = \gamma \int_{t_{LD}}^{\infty} I_{\text{tot}}(t) dt - \gamma \int_{t_{LD}}^{\infty} I_{LD} e^{\gamma(R_{0z}-1)(t-t_{LD})} dt, \quad (6)$$

which corresponds to the area of the yellow shaded region in Fig. 3 A, multiplied by γ . Note that the second integral, representing the area under the dashed green line in Fig. 3 A, gives the number of individuals infected during the lockdown ($t > t_{LD}$) if a complete lockdown was implemented with no active clusters ($N = 0$). This can be calculated explicitly and has the value $I_{LD}/(1 - R_{0z})$, which we use as a reference value for the extra infections. Thus, the effect of the clusters in the incomplete lockdown $N > 0$ can be characterized by the relative increase of total infections:

$$\mu = I_{\text{extra}}(1 - R_{0z})/I_{LD} \quad (7)$$

Results in Fig. 3 B show that as the number and size of the active clusters increases, the number of infected individuals decays slower than the value expected for a uniformly locked-down SIR model: $\alpha_0 = \gamma(1 - R_{0z}) = 0.04(\text{day}^{-1})$. For the range of values considered in the simulations, we find that when the locked-down population fraction decreases to around $L = 0.8$, the characteristic decay time of the epidemic, α^{-1} , increases from the reference value of 25 days (for $L = 1$) to around 50 days, i.e., $\alpha \approx 0.02(\text{day}^{-1})$. This implies that a 20% decrease in the fraction of the locked-down population results in doubling the decay time of the epidemic.

Fig. 3 C shows that the delay of the decay phase, τ , can increase substantially in the range of tens to 100 days when the locked-down fraction is reduced to 80–90%. Also we find, again, that for a certain locked-down population fraction, the delay can be much longer when the active population is composed of larger clusters. The relative increase of the total infections during the lockdown due to the active clusters (μ) shows a similar behavior (see Fig. 3 D) indicating up to more than a 10-fold increase when the active proportion is increased to around 10% of the population.

To further illustrate the differential impact of the number and size of the active clusters on the extra infections during the decay phase of the epidemic, we generate a heat-map representing $I_{\text{extra}}(M, N)$; see Fig. 4, A and B. Using a logarithmic scale for both variables N and M , the linear isocontours in Fig. 4 B suggest that to approximate the dependence on these variables by a power law of the form:

$$I_{\text{extra}} \approx N^a M^b. \quad (8)$$

We find that this functional form leads to a good fit for the scaling of the data from the stochastic simulations (Fig. 4, C and D) resulting in $a \approx 1$ and $b \approx 1.8$. This confirms our earlier prediction that increasing the size of the clusters (M) has a stronger impact on the total infections than increasing the number of clusters (N) by the same proportion.

We finally examine how the rate α and delay τ of the decay phase contribute to the extra infections in the population. This relationship can be quantitatively expressed as:

$$I_{\text{extra}} \approx I_{LD} \left(\frac{1}{\alpha} - \frac{1}{\alpha_0} \right) + I_{LD} \tau = \hat{I}_{\text{extra}}, \quad (9)$$

where the two terms represent the contributions due to slower and later decay, respectively. This approximation is based on replacing the transition region (dotted region in Fig. S1 A) by a constant value $I(t) = I_{LD}$. Although this formula slightly underestimates the total infections in some cases, it provides a very simple representation of the two components.

FIGURE 2 Total number of infected individuals ($I_{\text{tot}}(t)$) versus time for different numbers (N) and sizes (M) of active clusters. Circles represent $I_{\text{tot}}(t)$ calculated using the Gillespie algorithm after averaging over 10 repeated simulations. The solid red line is obtained by fitting $I_0 e^{-\alpha t}$ to the simulation data points highlighted by orange using the Mathematica routine NonlinearModelFit. To perform the fitting with minimal effects of the stochasticity, in most cases, the fitting range is set to start at a time after lockdown time t_{LD} where $100 < I_{\text{tot}}(t) < 3000$ (see Table S1 for the fit parameters I_0 and α). Dashed green line represents $I_{\text{LD}} e^{\gamma(R_0-1)(t-t_{\text{LD}})}$ that passes through $I_{\text{LD}} = I_{\text{tot}}(t_{\text{LD}})$, where t_{LD} is the time of lockdown. Blue line represents $I_0 e^{\gamma(R_0-1)t}$. Black dashed line represents t_{LD} . Other simulation parameters are in Table 1. To see this figure in color, go online.

We find a good agreement between the numerical and analytical calculations of the extra number of infected individuals, calculated using Eqs. 6 and 9, respectively; see Fig. S1 B. The increase of infected individuals appears to be mainly due to the delay (τ) in the decay phase, rather than the rate (α) of the decay (compare Fig. S1, C and D). These results provide an explanation for our earlier prediction, that dividing the active subpopulation into smaller clusters reduces the total number of infected individuals, mainly because of faster transition from growth phase to decay phase when the size of the outbreaks in infected clusters is smaller.

DISCUSSION

We studied the effect of a large number of small active clusters within a locked-down majority population on

the transition to the decay phase of an epidemic. We have shown that the active clusters can lead to delayed onset of the decay of the number of infected individuals in the population and a slower decay rate of the epidemic. In our representative example simulations, considering a COVID-19-like epidemic in a city of 1 million inhabitants, we find that the delay can be in the range of several weeks to a few months, whereas the characteristic time of the exponential decay, assumed to be around 3 weeks for the locked-down population, may double. This could lead to up to a 10-fold increase in the total number of infections occurring from the start of the lockdown relative to the case of a complete uniform lockdown. We found that the size of the clusters has a much stronger impact on the total extra infections in the incomplete lockdown than the number of these clusters, and the extra infections can be approximated with a double power law expression

FIGURE 3 Analysis of the decay phase of an epidemic. (A) A sketch illustrating three properties of the decay phase (for example, with $N = 2000$ and $M = 70$). The decay rate α corresponds to the slope of the fit $I_0 e^{-\alpha t}$ (red solid line). The delay τ in the epidemic decay, because of the presence of the active clusters, is the time-shift relative to an exponential decay with the same slope (α) that passes through the lockdown point (t_{LD}, I_{LD}) (brown dotted line); see Eq. 5. Finally, the number of extra infected individuals I_{extra} , calculated using Eq. 6, is represented by the area of the yellow shaded region. Green dashed line: $I_{LD} \exp(\gamma(R_0 - 1)(t - t_{LD}))$. (B–D) Decay rate α , delay τ , and relative increase of total infections μ versus the active population ratio not locked-down, $1 - L$. Dashed line in (B) represents $\alpha_0 = \gamma(1 - R_0)$. Other simulation parameters are in Table 1. To see this figure in color, go online.

of the form: $I_{extra} \approx N \times M^{1.8}$. We also identified that the dominant factor leading to the extra infections is the delayed transition to the decay phase which has an approximately fivefold larger contribution than the slower than expected decay.

The above results are based on a series of simplifying assumptions that allow us to keep a fairly simple general mathematical framework and efficient computer simulations while capturing the important impact of small minority groups in disease transmission, which is completely missed in standard ODE based compartmental epidemic models. This is particularly relevant in the context of the current COVID-19 pandemic, in which unexpected strong overrepresentation of certain affected subgroups in the population become evident (e.g., very high proportion of nursing home deaths, or frequent outbreaks in meat factories, livestock plants, and prisons) (19–22). Our model of course gives a very crude representation of these subgroups, considering a single average size of the clusters and assuming that the reproductive ratio remains the same as in the initial uncontrolled epidemic. Also, for simplicity, we assumed that the outbreaks occurring within the clusters are only stopped by the development of natural herd immunity within the cluster. In reality these outbreaks may be detected before they reach herd immunity and can be controlled through quarantine measures. This could be implemented quite easily in the model and would likely be equivalent to considering a smaller effective cluster size in the current model.

We also note that the disproportionate impact of small subpopulations in epidemic models have also been observed in various other contexts. For example, in social-contact-network-based epidemic models, it was shown that a very small fraction of highly connected individuals can have a large effect on the spread of infections in the population (23). Likewise, other models have shown that the rate at which an epidemic progresses is related to the strictness of control measures (24), different community mixing rates (25), and overlapping subpopulations (26) (reviewed in (27)). In the case of COVID-19, it was suggested that certain super-spreading events may play an essential role in the epidemic dynamics (28). These examples also demonstrate potential limitations of mean field type models restricted to representing the average properties and behavior of the interacting components.

SUPPORTING MATERIAL

Supporting material can be found online at <https://doi.org/10.1016/j.bpj.2021.04.004>.

AUTHOR CONTRIBUTIONS

Z.N. and A.C. conceived the idea. H.K. and J.K. performed the mathematical and numerical analyses. I.S. implemented the computational simulations. H.K., Z.N., A.C., and I.S. wrote the article.

FIGURE 4 Analysis of the extra number of infected individuals, I_{extra} calculated using Eq. 6. (A and B) I_{extra} vs N and M in linear (A) and log (B) scales. (C and D) Dashed lines represent fits $rN^a M^b$ to $I_{\text{extra}}(N; M)$ simulation data points, where $a = 1.08 \pm 0.02$, $b = 1.86 \pm 0.02$, and $r = 0.01 \pm 0.001$. Parameter values correspond to mean $\pm$ SE. Dotted lines represent the reference value of total infected from the start of lockdown in a population without active clusters ($N = 0$), i.e., the area under the dashed green curve in Fig. 3 A. To see this figure in color, go online.

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