The impact of the infectious period on epidemics

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Abstract

The duration of the infectious period is crucial in determining the ability of an infectious disease to spread. We consider an epidemic model that is network-based and non-Markovian, containing classic Kermack-McKendrick, pairwise, message passing and spatial models as special cases. For this model, we prove a monotonic relationship between the variability of the infectious period (with fixed mean) and the probability that the infection will reach an arbitrary subset of the population by time t. The striking importance of this relationship, even under standard assumptions, is demonstrated.

In a homogeneously mixing large population, under standard assumptions, the central epidemiological quantity R_0 (this being the expected number of secondary cases per typical primary case in an otherwise susceptible population) only depends on the infectious period through its mean [1]. However, other important quantifiers such as the probability of a major outbreak and the initial growth rate can depend on the variability of the infectious period; with higher variability tending to decrease these quantities [1]. When incorporating a much greater degree of realism such that individuals can only make contacts to their neighbours in a contact network [2], R_0 typically depends on the variability of the infectious period and, even when R_0 is held fixed, the probability that any given individual will eventually get infected is still dependent on the variability of the infectious period [3]. In this letter, we extend these results to a much more general epidemic model and consider the effect of the infectious period distribution on the fundamental probability $P(\mathcal{A}, t)$ that the disease will spread to an arbitrary subset $\mathcal{A}$ of the population by an arbitrary time t. This probability is relevant to the likelihood of an epidemic, and the speed and extent of its propagation.

It is commonplace to assume that the infectious period is exponentially distributed because this leads to greater mathematical tractability. In choosing the parameter for this distribution, the modeller may try to replicate the estimated average infectious period or the estimated value for R_0. In any case, data shows that the exponential distribution is not very realistic for this variable. For example, it has been suggested that gamma, Weibull and degenerate

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(non-random) distributions may be more realistic for diseases such as smallpox, ebola and measles [4, 5, 6, 7]. Thus, investigating the effect of the infectious period distribution is important for obtaining a qualitative understanding of the ability of different diseases to propagate, and the effects of intervention strategies which may modify this distribution. It is also important for informing parameter choices in epidemic models.

The Susceptible-Exposed-Infected-Recovered (SEIR) compartmental model for the spread of infectious diseases may be considered in a general stochastic and network-based form (see, for example, [8] and [9]). Here we consider a similar stochastic epidemic model which we construct as a non-Markovian stochastic process taking place on an arbitrary static contact network (or graph). We allow arbitrarily distributed exposed and infectious periods, heterogeneous contact processes between individuals, and heterogeneity in susceptibility and infectiousness. Many previously studied models such as Kermack-McKendrick [10], pairwise [11, 12], message passing [9] and spatial models [13, 3] are special cases [14].

The convex order [15], which provides a type of variability ordering for random variables with the same mean, is central to the work that we present here. Our main result shows that, under mild assumptions, by changing the infectious period distributions such that they decrease in convex order, we can only increase $P(\mathcal{A}, t)$. We discuss some important corollaries of this and then present examples and a numerical illustration.

The most relevant previous work [3] compares two Susceptible-Infected-Recovered (SIR) network-based epidemic models, where the infectious period is random in one and non-random in the other, and where the 'transmission probability' that an individual, given that it gets infected, will contact a given neighbour before recovering is the same in both models. It was shown that, under stronger assumptions than here, the long term probabilities $\lim_{t \to \infty} P(\mathcal{A}, t)$ are greater (or the same) in the model with the non-random infectious period. To relate more directly to this result, we define (following [16]) the 'transmissibility' to be the posterior probability that an infected individual, with a given infectious period, will make a contact to a given neighbour before recovering. Thus, the transmissibility is a random variable since it is a function of the infectious period, and its expected value is the transmission probability. We show that by changing the infectious period such that the transmissibility is decreased in convex order, which we shall see keeps R_0 constant, we can only increase $P(\mathcal{A}, t)$. We discuss some important corollaries of this and then present an example and numerical illustration.

The SEIR epidemic model under consideration is defined as follows: Let $G = (\mathcal{V}, \mathcal{E})$ be an arbitrary simple undirected graph, where $\mathcal{V}$ is a finite or countably infinite set of vertices (individuals) and $\mathcal{E}$ is a set of undirected edges between the vertices. For $i \in \mathcal{V}$, let $\mathcal{N}_i = \{j \in \mathcal{V} : (i, j) \in \mathcal{E}\}$ be the set of neighbours of i and let $|\mathcal{N}_i| < \infty$ for all $i \in \mathcal{V}$ (the graph is thus described as 'locally finite'). We assume that two individuals are neighbours if and only if at least one can make direct contacts to the other. Let $\nu_i \in [0, \infty]$ denote i's exposed (infected but not infectious) period if i is ever infected; $\mu_i \in [0, \infty]$

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is i's infectious period if i is ever infected; $\omega_{ji} \in [0, \infty]$ is the time elapsing between i first becoming *infectious* and it making a sufficient (for transmission) contact to j, if i is ever infected (note that the sufficient contact cannot cause infection if it occurs after i's infectious period has terminated); W_{out}^i is some variable on which all of the sufficient contact times $\omega_{ji}(j \in \mathcal{N}_i)$ may depend, e.g. a quantifier of infectiousness arising from sources other than the length of the infectious period; W_{in}^i is some variable on which all of the sufficient contact times $\omega_{ij}(j \in \mathcal{N}_i)$ may depend, e.g. a quantifier of susceptibility. For $t \in [0, \infty)$, i makes an *infectious contact* to j at time t if and only if (i) i enters the infected (as opposed to exposed) state at some time $s \le t$, (ii) $\omega_{ji} = t - s$, and (iii) $\omega_{ji} \le \mu_i$. Susceptible individuals enter the exposed state as soon as they receive an infectious contact, exposed individuals immediately enter the infected state when their exposed period terminates, and infected individuals immediately enter the recovered state when their infectious period terminates. Individuals may be in any state at $t = 0$ and may also be vaccinated.

Letting $\mathcal{X} = \cup_{i \in \mathcal{V}}\{\nu_i, \mu_i, W_{\text{in}}^i, W_{\text{out}}^i, \omega_{ji}(j \in \mathcal{N}_i)\}$, the situation which we wish to consider is where $\mathcal{X}$ and the initial conditions are random. We will assume that μ_i is independent from $\mathcal{X} \setminus \{\mu_i\}$ for all $i \in \mathcal{V}$; ω_{ji} and $\mathcal{X} \setminus \{\omega_{ji}\}$ are independent given W_{in}^j and W_{out}^i for all $i \in \mathcal{V}, j \in \mathcal{N}_i$; and the initial state of the population is independent from $\mathcal{X}$.

To understand the impact of the infectious periods on the likelihood, speed and extent of epidemic spread, we will first focus on a single individual $i \in \mathcal{V}$ and label a subset $\mathcal{B}$ of its neighbours using a bijection to $\{1, 2, \ldots, |\mathcal{B}|\}$. Assume that i gets infected and consider its behaviour after it leaves the exposed state and immediately enters the infectious state, and also assume that all of the variables except μ_i and $\omega_{ji}(j \in \mathcal{N}_i)$ have already been drawn from their joint distribution. Let $i \nrightarrow$ denote the event that i does not make an infectious contact to neighbour 1 within time period $x_1 \ge 0$, neighbour 2 within time period $x_2 \ge 0$, ..., and neighbour $|\mathcal{B}|$ within time period $x_{|\mathcal{B}|} \ge 0$, where the x_j are arbitrary non-negative numbers. We may now write

$$
\begin{aligned}
P^*(i \nrightarrow) &= P^*(\omega_{1i} > \min\{x_1, \mu_i\}, \omega_{2i} > \min\{x_2, \mu_i\}, \\
&\qquad \ldots, \omega_{|\mathcal{B}|i} > \min\{x_{|\mathcal{B}|}, \mu_i\}) \\
&= E[\phi(\mu_i)],
\end{aligned}
\tag{1}
$$

where

$$
\phi(\tau) = \prod_{j=1}^{|\mathcal{B}|} \phi_j(\tau) \qquad (\tau \in [0, \infty)),
$$

and,

$$
\phi_j(\tau) = P^*(\omega_{ji} > \min\{\tau, x_j\}).
$$

We use P^* to indicate that we are conditioning on the values already drawn for W_{out}^i and $W_{\text{in}}^j(j \in \mathcal{N}_i)$, since the $\omega_{ji}(j \in \mathcal{N}_i)$ may depend on these. The form of (1) may be understood by observing that if the infectious period takes the

value τ and $\tau < x_j$, then for no infectious contact to j within time period x_j we only need the sufficient contact time ω_{ji} to be greater than τ (since then the infectious contact never takes place).

Let us now consider the conditions under which $\phi(\tau)$ is convex since this will be necessary for our analytical results. It is straightforward that it is convex if $\phi_j(\tau)$ is convex for all $j \in \mathcal{N}_i$, since the $\phi_j(\tau)$ are non-negative and non-increasing. It is also straightforward that $\phi_j(\tau)$ is convex if the survival function for ω_{ji}, after conditioning on any possible values for W_{out}^i and W_{in}^j, is always convex; and a non-increasing PDF (probability density function) is sufficient for a convex survival function. If contact processes are independent Poisson processes, which is a common assumption, then the ω variables are exponential and thus have convex survival functions. If the ω variables are independent and gamma distributed with shape parameters less than or equal to 1 then their survival functions will be convex. We also note that the survival function $f(x) = (1 + x/\lambda)^{-\alpha}$, where $\lambda, \alpha > 0$, for the heavy-tailed Lomax distribution is convex on $[0, \infty)$. Moreover, since any function of the form $f(x) = x^{-\alpha}$, where $\alpha > 0$, is convex on $[a, \infty)$, then ω variables which have other heavy-tailed distributions may have convex survival functions. It has been shown how processes which depend on human decision-making may develop inter-event times which have heavy-tailed distributions, and data for some such processes do indeed indicate heavy tails [17].

An important example where $\phi(\tau)$ is certainly convex is the case where $W_{\mathrm{in}}^i, W_{\mathrm{out}}^i (i \in \mathcal{V})$ take values in $(0, 1]$ and, for all $i \in \mathcal{V}, j \in \mathcal{N}_i$, we have $\omega_{ji} \sim \exp(\beta_{ji} W_{\mathrm{out}}^i W_{\mathrm{in}}^j)$ where $\beta_{ji} > 0$. This corresponds to a scenario where i, while infectious, makes contacts to j according to a Poisson process of rate β_{ji} and a contact is sufficient with probability $W_{\mathrm{out}}^i W_{\mathrm{in}}^j$ (a time-inhomogeneous Poisson process could be used instead, but the rate would need to be non-increasing).

Having discussed scenarios where $\phi(\tau)$ is convex and put forth arguments for the realism of this, we will for the remainder of the letter assume it to be the case. We next use its convexity to prove our analytical results concerning the effect of the infectious period distribution on the ability of the disease to spread.

Let X_1 and X_2 be two random variables which take values in some interval of the real line. If $E[\psi(X_1)] \geq E[\psi(X_2)]$ for all convex functions ψ then we say that X_1 is greater than X_2 in convex order and write $X_1 \geq_{\mathrm{cx}} X_2$. An important result for the convex order is that

$$X_1 \geq_{\mathrm{cx}} X_2 \quad \text{implies} \quad E[X_1] = E[X_2], \mathrm{Var}(X_1) \geq \mathrm{Var}(X_2).$$
(2)

Another useful result is that if $E[X_1] = E[X_2]$, and F_{X_1} and F_{X_2} cross exactly once (where these are the cumulative distribution functions for X_1 and X_2), and the sign sequence of $F_{X_2} - F_{X_1}$ is $-, +$, then this implies that $X_1 \geq_{\mathrm{cx}} X_2$ [15]. We will refer to this as the graphical sufficient condition for the order.

Thus, since $\phi(\tau)$ is convex, then decreasing μ_i in convex order can only

decrease $P^*(i \nrightarrow)$ because the expectation in (1) can only decrease. Since the x_j were arbitrarily chosen non-negative numbers and $\mathcal{B}$ was an arbitrary subset of i's neighbours, the transmission probability that i will make an infectious contact to $j \in \mathcal{N}_i$, given that i gets infected, can only increase. We will assume, naturally, that R_0 is monotonic with respect to these transmission probabilities. Therefore, R_0 can only increase.

More importantly, for all subsets $\mathcal{A} \subset \mathcal{V}$ and all $t \geq 0$, the fundamental probability $P(\mathcal{A}, t)$, that a member of $\mathcal{A}$ will become infected before time t, can only increase. To understand this, note that since we have already drawn all of the variables except μ_i and $\omega_{ji}(j \in \mathcal{N}_i)$, then either it is already known whether or not the infection reaches subset $\mathcal{A}$ by time t (for instance, if all of $\mathcal{A}$ are initially susceptible and $\omega_{kl} > t$ for all $k \in \mathcal{A}, l \in \mathcal{N}_k$), or there exists some choice of $\mathcal{B}$ and the x_j such that this occurs if and only if $i \nrightarrow$ does not occur; and, as we have shown, the probability of $i \nrightarrow$ can only decrease.

Since i is an arbitrary member of $\mathcal{V}$ and all of the infectious period distributions were arbitrary, we can repeatedly apply this argument to conclude that $P(\mathcal{A}, t)$ can only increase if any subset of the infectious periods are decreased in convex order. We note here that an entirely analogous argument, which does not require $\phi(\tau)$ to be convex, shows that $P(\mathcal{A}, t)$ can only increase if infectious periods are increased in the usual stochastic order; and it is necessary that the the means are not decreased for this order to hold.

Let us now consider what this suggests more generally about the importance of the shape of the infectious period distributions. Firstly it is straightforward that, for given means, the infectious periods which maximise $P(\mathcal{A}, t)$ are non-random, i.e. have zero variance. This follows from the graphical sufficient condition for the convex order which shows that any other infectious periods with the same means are necessarily greater in convex order. Secondly, for given means and given maximum values, i.e. bounded infectious periods, the infectious periods which minimise $P(\mathcal{A}, t)$ are such that they are either equal to zero or to their maximum values (their variance is maximal). Again, this follows similarly from the graphical sufficient condition for the convex order. Thus, the tendency of decreasing the variances of the infectious periods to increase the probability that the infection will spread to a given part of the network by a given time is made clear. This tendency is also highlighted by (2).

Gamma and Weibull distributions are realistic for the infectious periods; they allow concentration about their mean values unlike the exponential distribution. For two gamma distributions with the same mean, it is straightforward, using the graphical sufficient condition, that the one with greater variance is necessarily greater in convex order; the same applies for two Weibull distributions with the same mean. So if we restrict our distributions to one of theses two families, and keep the means fixed, then decreasing the variances of the infectious periods can only increase $P(\mathcal{A}, t)$. An illustration of the extent of this increase, for the case of the gamma distribution, is shown in Fig. 1. The effect is remarkable when one considers that the mean is fixed and we have just interpolated between the exponential distribution and the degenerate distribution, both of which are commonly assumed for the infectious period. It also reveals
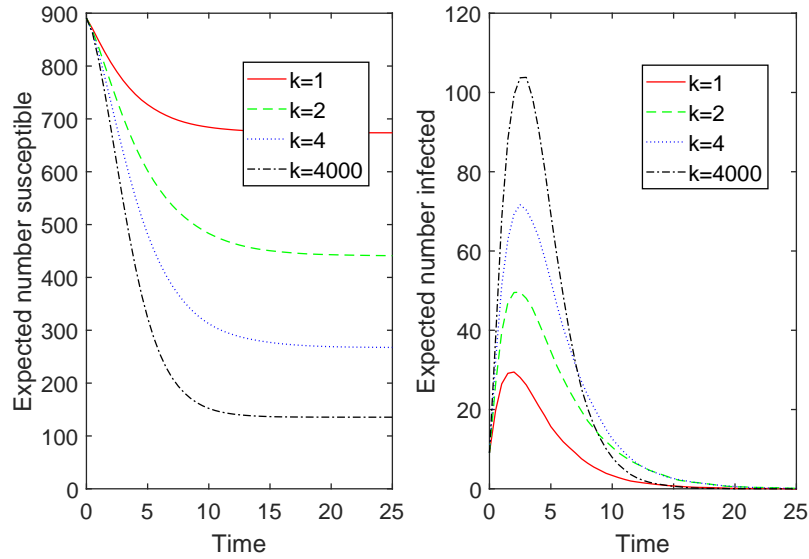
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Figure 1: We consider a special case of the stochastic model where the graph is a square lattice of 900 individuals and $\mathcal{X}$ is mutually independent; $\omega_{ji} \sim \mathrm{Exp}(1)$ for all $i \in \mathcal{V}, j \in \mathcal{N}_i$; $\nu_i = 0$ for all $i \in \mathcal{V}$; $\mu_i \sim \Gamma(k, 3/4k)$ for all $i \in \mathcal{V}$; every individual is independently initially infected with probability 0.01 and initially susceptible otherwise. On the left we have approximated the expected number susceptible against time for $k = 1, 2, 4, 4000$, corresponding to variances of approximately 0.56, 0.28, 0.14, 0.00014, while on the right we have approximated the expected number infected against time for $k = 1, 2, 4, 4000$. Each approximation was computed as the average of 1000 stochastic simulations. Here, the mean infectious period is the same for all individuals and kept constant at 3/4.

the large amount of error that could be introduced, at all points in time, when approximating the epidemic as a Markov process and using the reciprocal of the estimated average infectious period as the recovery rate in the model.

We have shown how R_0 is decreasing with respect to the variability (in the sense of the convex order) of the infectious period. Since it may be sensible to choose an infectious period distribution for our model such that the estimated value of R_0 for the disease is replicated, as opposed to the estimated mean of the infectious period, then it is pertinent to consider the sensitivity of $P(\mathcal{A}, t)$ to the infectious period distribution when R_0 is fixed. We will assume, naturally, that R_0 may be fixed by keeping the transmission probabilities between all ordered pairs of neighbours constant.

To proceed, let us now assume that the ω variables are mutually independent and so we discard the $W_{\text{in}}^i, W_{\text{out}}^i (i \in \mathcal{V})$ variables; and assume that, for each $i \in \mathcal{V}$, the $\omega_{ji}(j \in \mathcal{N}_i)$ are independent and identically distributed (i.i.d.). However, one gain here is that we do not make any assumptions about the survival functions of the ω variables. Again, we assume that all of the variables except μ_i and $\omega_{ji}(j \in \mathcal{N}_i)$ have already been drawn from their joint distribution. Given our motivation, let $F_{\omega_{.i}}(\tau)$ denote $P(\omega_{ji} < \tau)$ and let Z_i denote the random 'transmissibility' variable $F_{\omega_{.i}}(\mu_i)$. It is the transmission probability $E[Z_i]$ which we desire to be kept constant. Now note that $F_{\omega_{.i}}^{-1}(Z_i) = \mu_i$ where $F_{\omega_{.i}}^{-1}(\tau) = \sup\{\tau' : F_{\omega_{.i}}(\tau') = \tau\}$, assuming for the moment that $F_{\omega_{.i}}(\tau)$ is continuous. We can write

$$P(i \nrightarrow) = E[\theta(Z_i)],$$

where

$$\theta(\tau) = \prod_{j=1}^{|\mathcal{B}|} \theta_j(\tau) \qquad (\tau \in [0, 1]),$$

and,

$$\theta_j(\tau) = \begin{cases} 1 - \tau & \text{if } 1 - \tau \geq P(\omega_{ji} > x_j) \\ P(\omega_{ji} > x_j) & \text{otherwise.} \end{cases}$$

This holds because $P(\omega_{ji} \geq F_{\omega_{.i}}^{*-1}(\tau)) = 1 - \tau$ for all $\tau \in [0, 1]$ and all $j \in \mathcal{N}_i$. If we allow $F_{\omega_{.i}}(\tau)$ to be discontinuous then $F_{\omega_{.i}}^{-1}(\tau)$ is undefined for all $\tau \in A$, for some $A \subset [0, 1]$, but then the probability that Z_i belongs to A is zero. It is straightforward that $\theta(\tau)$ is convex on $[0, 1]$.

Thus, altering the infectious period such that Z_i is decreased in convex order can only cause $P^*(i \nrightarrow)$ to decrease and $P(\mathcal{A}, t)$ to increase by the same arguments as before. Using the graphical sufficient condition we must then have that, keeping R_0 constant, $P(\mathcal{A}, t)$ is maximised when the Z_i are non-random. This is the case when the infectious periods are non-random. So, whether the infectious periods are altered such that the means are held constant, or such that R_0 is held constant (with the slightly different sets of assumptions), $P(\mathcal{A}, t)$ is maximised when the infectious periods are non-random. On the other hand,
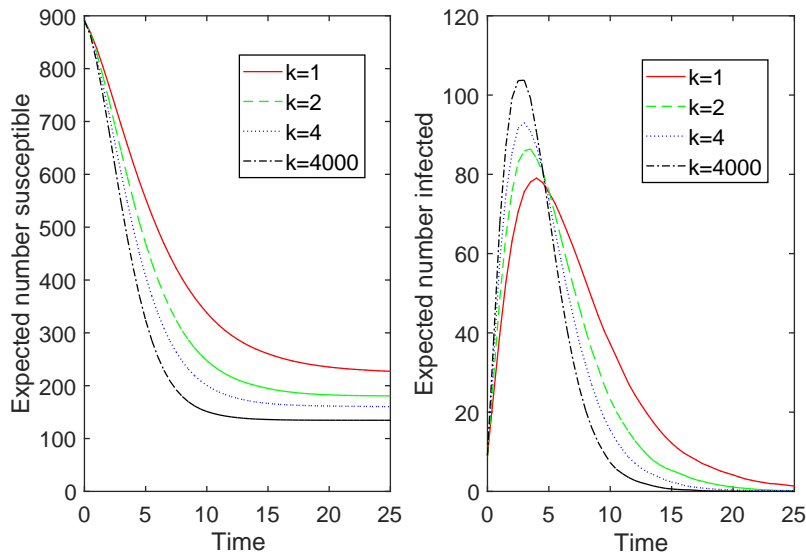
Figure 2: We consider the same scenario as for Fig. 1 except with $\mu_i \sim \Gamma(k, e^{3/4k} - 1)$ for all $i \in \mathcal{V}$. Here, the transmission probability is the same for all ordered pairs of neighbours and kept constant at $1 - e^{-3/4} \approx 0.53$, giving $R_0 \approx 3 \times 0.53 = 1.59$. For $k = 1, 2, 4, 4000$, the mean of the infectious period is approximately 1.1, 0.91, 0.82, 0.75, with variance 1.2, 0.41, 0.17, 0.00014, respectively.

$P(\mathcal{A}, t)$ is minimised when the Z_i can only be equal to either 0 or 1. This is the case when the infectious periods can only be zero or infinite. Thus, like with the infectious periods themselves, there is a clear tendency for decreasing the variances of the transmissibility variables to increase $P(\mathcal{A}, t)$.

Fig. 2 demonstrates the extent to which the infectious period distribution can affect $P(\mathcal{A}, t)$ when R_0 is held constant; it is here clearly less important than when the means of the infectious periods are held fixed. This suggests that we should base our choice for the infectious period distribution more on the estimated value of R_0 than on the estimated average infectious period - at least when computing the timecourse of the number susceptible (equivalently, the timecourse of the total number of cases). For a given epidemic model, this also suggests the strategy of computing the transmission probability, or R_0, first and then using this to inform a new choice for the infectious period distribution which will ease numerical solution or mathematical analysis. For example, for Poisson contact processes, the deterministic message passing and pairwise models [9, 12] may be solved as ordinary differential equations or delay differential equations in the case of a zero-or-infinite infectious period or non-random infectious period respectively. In fact, where these deterministic models are consistent with the stochastic model [14], we would then get rigorous lower

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and upper bounds on the epidemic timecourses.

The results of this paper may also be applied to the classic deterministic SIR model proposed by Kermack and McKendrick [10]. The model is defined as follows:

$$\dot{S}(t) = S(t)\left[\int_0^t h(\tau)\bar{F}_\mu(\tau)\dot{S}(t-\tau)\mathrm{d}\tau - I(0)h(t)\bar{F}_\mu(t)\right], \qquad (3)$$

$$I(t) = 1 - S(t) - R(t), \qquad (4)$$

$$R(t) = R(0) + \int_0^t f_\mu(\tau)[1 - R(0) - S(t-\tau)]\mathrm{d}\tau, \qquad (5)$$

where the variables on the left hand sides represent the fraction susceptible, infected and recovered respectively at time t; $h(\tau)$ is the rate at which an individual, that has been infected for time period τ, makes contacts to others; and μ is the random infectious period with density function f_μ and survival function $\bar{F}_\mu$. Let $Z^* = \int_0^\mu h(\tau)\mathrm{d}\tau$ such that $E[Z^*]$ is the expected number of infectious contacts that an infected individual will make before recovering. Thus Z^* plays a similar role to the previously defined transmissibility random variable. Equations 12-15 of Kermack and McKendrick [10] may be obtained from (3)-(5) after multiplying through by the total population size N and after appropriately renaming the variables and functions.

Let $S_1(t)$ be given by system (3)-(5) but with μ replaced by μ_1. Let $S_2(t)$ be given by (3)-(5) but with μ replaced by μ_2. Let $h(\tau)$ be continuously differentiable. Assume at least one of the following conditions:

(i) $\mu_1 \leq_{\mathrm{st}} \mu_2$

(ii) $\mu_1 \geq_{\mathrm{cx}} \mu_2$ and $h(\tau)$ is non-increasing on $[0, \infty)$

(iii) $Z_1^* \leq_{\mathrm{st}} Z_2^*$ (defined using μ_1 and μ_2 respectively)

(iv) $Z_1^* \geq_{\mathrm{cx}} Z_2^*$

Then for all $t \geq 0$, we have $S_1(t) \geq S_2(t)$. The proof for this is in the Appendix. Note that if individuals are assumed to make contacts according to a homogeneous Poisson process then $h(\tau)$ is constant and therefore non-increasing and continuously differentiable. It is also worth noting that if $h(\tau)$ is non-increasing, then by replacing the infectious period in the Kermack-McKendrick model by one which is non-random, but with the same mean, a lower bound on $S(t)$ is achieved for all $t \geq 0$.

In conclusion, for an extremely general epidemic model, we have proved a monotonic relationship between the expected number to get infected by time t and the variability of the infectious period (with constant mean). Our numerical results illustrate the high sensitivity to the infectious period distribution when the mean is fixed compared to when R_0 is fixed. However, R_0 is much more difficult to compute empirically. Thus, this adds to recent research which has sought to articulate the impact of non-Markovian dynamics in epidemic models [18, 19, 20, 1, 21]. Notably, our results do not depend on the assumption of

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exponential contact times, the validity of which has recently been questioned since heavy-tailed distributions have been inferred from observation [18, 17, 22].

It is unclear whether similar results can be found in compartmental structures, such as Susceptible-Infectious-Susceptible (SIS) dynamics, where individuals may be infected multiple times. Indeed, it has recently been shown by Ball et al. [23] that for a particular stochastic SIS model, in which contact processes are Poisson, the expected total time that the system spends in any given state only depends on the infectious period distribution through its mean.

Appendix

Let us consider the special case of the stochastic model where the graph G is an infinite n-regular tree (also known as a Bethe lattice); $\nu_i = 0$ for all $i \in \mathcal{V}$; $\mu_i \overset{d}{=} \mu_j$ for all $i, j \in \mathcal{V}$ (let μ denote the random infectious period with this distribution); the W variables are independent from the ω variables and so may be discarded; $\omega_{ij} \overset{d}{=} \omega_{kl}$ for all $j, l \in \mathcal{V}, i \in \mathcal{N}_j, k \in \mathcal{N}_l$ (let ω denote the random contact time with this distribution); and the initial states of individuals are independent and identically distributed (i.i.d.) random variables. Let $Z = F_\omega^*(\mu)$, where $F_\omega^*(\tau) = P(\omega < \tau)$, be the transmissibility.

Consider a sequence of stochastic models indexed by $n = 2, 3, \ldots$, as defined above, but where the density function f_ω depends on n as follows

$$ f_{\omega(n)}(\tau) = \frac{h(\tau)}{n} \exp\Big(-\frac{1}{n} \int_0^\tau h(\tau') \mathrm{d}\tau' \Big) \qquad (\tau \geq 0), $$

and $h(\tau)$ is taken from the Kermack-McKendrick model. Note that if $h(\tau)$ is non-increasing then the density function $f_{\omega(n)}(\tau)$ is non-increasing and the survival function $\bar{F}_{\omega(n)}(\tau)$ is convex, for all n.

Note that Z must now depend on n, and we have $Z(n) = 1 - \mathrm{e}^{-Z^*/n}$ which is a concave function of Z^* for all n, where Z^* is defined in the Kermack-McKendrick model (see the main text). It is straightforward to show that a non-increasing convex function, applied to a concave function, is a convex function. Therefore, if the third or fourth condition holds, then $E[\theta(Z(n))]$ is greater (or the same) if $\mu \overset{d}{=} \mu_1$ than if $\mu \overset{d}{=} \mu_2$, for all n.

Thus, the probability that an arbitrary individual is susceptible at time $t \geq 0$ is greater (or the same) if $\mu \overset{d}{=} \mu_1$ than if $\mu \overset{d}{=} \mu_2$, for every model in the sequence. Now, using Theorem 6 in [14], which tells us that as $n \to \infty$ the probability that an arbitrary individual is susceptible at time t converges to $S(t)$ (since $n f_{\omega(n)}(\tau) \to h(\tau)$), we have $S_1(t) \geq S_2(t)$ for all $t \geq 0$.

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