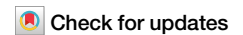


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Classical mean-field epidemic theory upper-bounds real disease spread



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Non-Markovian theory provides the most accurate stochastic description of an infection spreading on a contact graph with a general distribution of the infection time and curing time. However, the infection time is not directly measurable and instead the generation time is measured in real-world epidemics. Here we show that if the generation time follows approximately a Weibull distribution with shape parameter α , then the Weibull infection time of the spreading process possesses a similar shape parameter α . For a large list of published generation times in biological, mainly respiratory epidemics, the Weibull shape parameter α is computed, which reveals that nearly always $\alpha > 1$. The impact of this observation is that a complicated non-Markovian analysis is not needed, because the well-established Markovian case $\alpha = 1$ provides a lower bound for the epidemic threshold and an upper bound for the basic reproduction number and the nodal infection probabilities in the contact graph.

In most countries, academic groups together with public health agencies have been advising their governments during the the recent COVID-19 pandemic. In hindsight, the COVID-19 crisis has been remarkably well-managed in general, given that the computational methods were essentially based on the surprisingly simple SIR epidemic model. The main control parameter for the agencies was the basic reproduction number R_0 : if $R_0 < 1$, then the epidemic is “under control”, else actions are needed. While more accurate physical descriptions of epidemic spread in a time-variant contact graphs exist, our key message here is that the development of a difficult non-Markovian theory for epidemics on networks can be avoided for biological respiratory diseases. However, the comfortable and reassuring fact that a complicated non-Markovian theory is not needed, does not generally hold for non-biological spread in e.g. social contagion and information diffusion via online platforms.

The class of Susceptible-Infected-Susceptible (SIS) epidemics is the simplest compartmental model of a disease spread with re-infections in a population, in which individuals are either infectious (I) or healthy, but susceptible (S). The class of Susceptible-Infected-Removed (SIR) epidemics is the companion compartment model without re-infections, which adds the removed (R) compartment next to the I and S compartment. The SIR model [1, p. 713-716] arose first as a constant rate simplification of the more general Kermack-McKendrick theory of epidemics. Other compartmental models² can be described, both stochastically and deterministically (after a mean-field approximation), similarly to SIS/SIR epidemics. The SIR class is upper bounded in the nodal infection probability and in the basic reproduction number by its SIS companion^{3,4}, which justifies our confinement to SIS theory and the omission of an SIR analysis here. From a physical and mathematical point of view, the Markovian SIS process on a graph is the simplest and

still analytically tractable model in the class of “Local rule, Global emergence” (LrGe), that contains many physical processes such as Lotka-Volterra predator-prey, Kuramoto synchronization, opinion dynamics and others in e.g. [5, Sec. 1]. Most LrGe processes feature a phase transition.

The viral spreading process in SIS epidemics is described by the infection time T_{inf} while in real-world epidemics, the generation time T_{gen} is measured. We assume that the infection time T_{inf} and generation time T_{gen} can both be described by Weibull distributions. The Weibull distribution has a shape parameter $\alpha > 0$ and reduces to an exponential distribution for $\alpha = 1$. A large literature survey of published generation time measurements in biological epidemics reveals that the generation time shape parameter $\alpha_{\text{gen}} > 1$ in most cases. We show that a generation time shape parameter $\alpha_{\text{gen}} > 1$ implies an infection time shape parameter $\alpha_{\text{inf}} > 1$. Since the nodal infection probability decreases with α and the epidemic threshold which is inversely proportional⁶ to the basic reproduction number R_0 increases with α , we conclude that Markovian epidemics $\alpha = 1$ upper-bound the infection probability in real-world biological diseases, which supports our main message: current epidemic models, computed by most agencies, provide a safe-side overestimation of the real disease impact.

Methods

Markovian SIS epidemics on a graph

The contact graph G consists of a set of N individuals, called nodes in the graph, and L contacts as links in the graph. For simplicity, we assume here that the graph is fixed and does not change over time (a Markov theory of time-varying contact graphs exists, see e.g.^{7,8}). The graph G is described by the adjacency matrix A , where the element $a_{ij} = 1$ if there is a link between node i and node j , and $a_{ij} = 0$ otherwise.

At any time t in the SIS epidemic, each node is in one of two states: susceptible or infectious. There are two independent stochastic processes in the SIS model: the infection process is described by the infection time T_{inf} , and the curing process is described by the curing time T_{cure} .

The infection process determines the time at which an infectious node i spreads the disease to a susceptible node j , which can only occur if there is a link between node i and node j . In Markovian epidemics, an infectious node i infects a susceptible neighbor j at a constant rate β , which implies that the infection time T_{inf} is an exponentially distributed random variable with mean $\mathbb{E}[T_{\text{inf}}] = \frac{1}{\beta}$. Similarly, the curing process determines the time at which an infectious node i recovers from the disease. In Markovian epidemics, an infectious node i recovers from the disease at a constant rate δ , which implies that the curing time T_{cure} is an exponentially distributed random variable with mean $\mathbb{E}[T_{\text{cure}}] = \frac{1}{\delta}$.

Markovian SIS epidemics are fully parameterized by the infection rate β , the curing rate δ , and the contact graph G and can be described exactly by a Markov chain with 2^N states [9, Ch. 17.2.1]. The viral state of a node i at time t in a Markovian SIS process on a graph is specified by a Bernoulli random variable $X_i(t) \in \{0, 1\}$: the viral state $X_i(t) = 0$ for a susceptible node and $X_i(t) = 1$ for an infected node. The fact that the Markov state $X_i(t)$ in SIS epidemics is a Bernoulli random variable facilitates an elegant and physical differential equation for the infection probability of node i , first proposed in [10,

$$\frac{d\mathbb{E}[X_i]}{dt} = \mathbb{E}\left[-\delta X_i + \beta(1 - X_i) \sum_{k=1}^N a_{ki} X_k\right] \quad (1)$$

$$= -\delta \mathbb{E}[X_i] + \beta \sum_{k=1}^N a_{ki} \mathbb{E}[X_k] - \beta \sum_{k=1}^N a_{ki} \mathbb{E}[X_i X_k] \quad (2)$$

The time-derivative of the infection probability $\mathbb{E}[X_i] = \Pr[X_i = 1]$ of a node i consists of the expectation of two competing processes in (1), expressed in the Bernoulli random variable $X_i \in \{0, 1\}$: (i) if node i is infected $X_i = 1$, then the node i is cured at rate δ , else (ii) if node i is susceptible $X_i = 0$, then all infected neighbors $\sum_{k=1}^N a_{ki} X_k$ of node i try to infect the node i with rate β . The last term in (2), containing joint probabilities $\mathbb{E}[X_i X_k] = \Pr[X_i = 1, X_k = 1]$, is complicating and requires us to deduce the differential equation for $\mathbb{E}[X_i X_j]$, which is found in [9, Ch. 17.3]. The resulting differential equation for $\frac{d\mathbb{E}[X_i X_j]}{dt}$ contains the joint probabilities $\mathbb{E}[X_i X_j X_k]$ of the triples. Continuing in this manner as shown in [9, Ch. 17.3], we arrive at the 2^N linear differential equations.

Many interesting insights from (1) can be deduced. First, a powerful mean-field approximation, called N -Intertwined Mean Field Approximation (NIMFA) [11], explained in [12], follows from (1) by replacing the random variable X_i by its simplest approximation, its mean $v_i(t) = \mathbb{E}[X_i]$,

$$\frac{dv_i(t)}{dt} = -\delta v_i(t) + \beta(1 - v_i(t)) \sum_{j=1}^N a_{ij} v_j(t) \quad (3)$$

Since the NIMFA differential equation (3) is an approximation, $\mathbb{E}[\bar{X}_i]$ is written instead of $\mathbb{E}[X_i]$. The accuracy of NIMFA in (3) is assessed in [13]. Second, the NIMFA epidemic threshold $\tau_c^{(1)} = \frac{1}{\lambda_1}$ in [11, Lemma 6], where λ_1 is the largest eigenvalue or spectral radius [14] of the adjacency matrix A , is proved in [15] to be a lower bound for the Markovian epidemic threshold, i.e. $\tau_c > \tau_c^{(1)}$. Also [3], the NIMFA infection probability $v_i(t) = \Pr[\bar{X}_i(t) = 1]$ upper-bounds the Markovian probability of infection $\Pr[X_i(t) = 1]$. Further, NIMFA has been extended to a large variety of compartmental models in [2], exact solutions of the NIMFA differential equation (3) on the complete graph are derived in [16] and analytic solutions around the epidemic threshold in terms of a hyperbolic tangent function are deduced in [17].

Non-Markovian SIS epidemics on a graph

In non-Markovian SIS epidemics, the infection time T_{inf} and curing time T_{cure} can take any distribution. We limit ourselves here to a Weibull distribution with probability density function (pdf) [9, p. 56]

$$f_{\text{Weibull}}(x) = \frac{\alpha}{b} \left(\frac{x}{b}\right)^{\alpha-1} e^{-\left(\frac{x}{b}\right)^\alpha} \quad (4)$$

with shape parameter α and scale parameter b . The Weibull distribution is a good fit for many empirical distributions [18] and appears often in epidemic papers (e.g. [19–22]).

In general, the parameter α is the extreme value index [9, p. 398] of a probability distribution. Small changes in α lead to highly different behavior. If the parameter $\alpha \neq 1$, small x -values in the corresponding pdf tend to either 0 (for $\alpha > 1$) or infinity (for $\alpha < 1$). Only if $\alpha = 1$, the distribution is regular and possesses a Taylor series around $x = 0$. In particular, if $\alpha = 1$, then the Weibull distribution reduces to an exponential distribution, recovering the Markovian case. The parameter α also determines the fractional order of linear differential equations [23, Ch. 1], whose eigenfunctions are Mittag-Leffler functions $E_{\alpha,b}(z) = \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(\alpha k + b)}$, which are entire functions in the complex z -plane of order $\frac{1}{\alpha}$ and $E_{1,1}(z) = e^z$. Again, slightly different values of α lead to widely different asymptotic behavior of entire functions. The resulting α -process in continuous time, described as a linear combination of Mittag-Leffler functions with different b , features α -power law behavior for small and large times. The Mittag-Leffler distribution $1 - E_{\alpha,1}(-x^\alpha)$ behaves similarly [23, art. 101] as the Weibull distribution $1 - e^{-x^\alpha}$ for not too large $x > 0$ and the same $0 < \alpha \leq 1$. For $\alpha = 1$, the Mittag-Leffler distribution again reduces to the exponential distribution. In summary, the parameter α is a characteristic measure for non-Markovian behavior of a process.

For a random variable T , the hazard function $h_T(x) = \frac{f_T(x)}{S_T(x)}$ where $f_T(x)$ is the probability density function and $S_T(x) = \Pr[T > x] = 1 - F_T(x)$ is the survival function. The hazard function of a Weibull random variable is given by

$$h_{\text{Weibull}}(x) = \frac{\alpha}{b} \left(\frac{x}{b}\right)^{\alpha-1} \quad (5)$$

The hazard function $h(x)$ represents the instantaneous event rate at time x given that the event has not yet occurred by time x . Hence, the instantaneous rate at which an infectious node i recovers from the disease at time x is given by the hazard function $h_{T_{\text{cure}}}(x)$ of the curing time T_{cure} . Similarly, the instantaneous rate at which an infectious node i spreads the disease to a susceptible neighbor j is given by the hazard function $h_{T_{\text{inf}}}(x)$ of the infection time T_{inf} (under the so-called “rule 2” infection process defined by Boguñá et al. [24]). For shape parameter $\alpha = 1$, the Weibull distribution reduces to the exponential distribution and the hazard function (5) becomes constant, recovering the Markovian case.

Weibull infection time T_{inf} dramatically alters the SIS epidemic dynamics. The viral spreading process is very sensitive to the infection time shape parameter α_{inf} . Decreasing the infection time shape parameter α_{inf} leads to a significant increase in the prevalence as reported in [15] and [24]. The Weibull hazard function $h_{T_{\text{inf}}}(x)$ at small times x increases when the shape parameter α_{inf} decreases, which allows the virus to spread faster and leads to a higher prevalence. The epidemic threshold also decreases as shown in [15]. Therefore, the shape parameter α_{inf} characterizes the non-Markovian behavior. If $\alpha_{\text{inf}} \neq 1$, the process is non-Markovian, with smaller shape parameters corresponding to faster-spreading epidemics with higher prevalence. If the infection time shape parameter $\alpha_{\text{inf}} > 1$, then the Markovian case $\alpha_{\text{inf}} = 1$ provides a lower bound for epidemic threshold τ_c , which is in turn lower bounded by the NIMFA epidemic threshold $\tau_c^{(1)} = \frac{1}{\lambda_1}$. NIMFA therefore provide a safe-side overestimate of the epidemic process.

Results

Generation time in SIS epidemics

Direct measurement of the infection time T_{inf} is not possible, both in real epidemics as well as SIS epidemics. When an infectious node i is in contact with a susceptible node j , the infection time T_{inf} determines when node i will infect node j . However, the infection may possibly not occur, because either (i) another node k infects node j or (ii) node i cures before node i can infect node j . In reality, we can only observe infections that have actually occurred. Instead, the generation time T_{gen} is measured, defined as the time between the infection of the primary and secondary case²⁵. As illustrated in Fig. 1, node i becomes infected and is the primary case. Node i then infects node j , the secondary case. The time between the infection events is the generation time.

Since the time of infection is typically not known, the generation time is approximated by the serial interval, defined as the time between the onset of symptoms in the primary and secondary case. While generation times are always positive, serial intervals may be negative if people transmit the disease before symptom onset (Fig. 1). While the mean serial interval is a good estimator for the mean generation interval, the variance and other central moments differ²⁵, and these differences impact model predictions²⁶.

We deduce the distribution of generation time T_{gen} in SIS epidemics on networks and its relation to the infection time T_{inf} and curing time T_{cure} . The probability density function $f_{T_{\text{gen}}}(x)$ of the generation time T_{gen} is related to the basic reproduction number R_0 by

$$f_{T_{\text{gen}}}(x) = \frac{m(x)}{R_0} \quad (6)$$

where $m(x)$ is the so-called “infectivity profile”, defined as the average rate at which an infected individual infects new individuals at time x after his/her infection^{27,28}. Let $M(x) = \int_0^x m(x) dx$ denote the expected number of secondary infections caused by an infectious node i up to time x after node i becomes infected at time 0. Let $\Lambda(x)$ denote the expected number of secondary infections caused by an infectious node i up to time x after node i becomes infected, conditioned on the fact that node i has not cured by time x . If the curing time $T_{\text{cure}} > x$, which occurs with survival probability $S_{T_{\text{cure}}}(x) = 1 - F_{T_{\text{cure}}}(x) = \Pr[T_{\text{cure}} > x]$, the expected number of secondary infections before time x is $\Lambda(x)$. If the curing time $T_{\text{cure}} = \nu < x$, then the expected number number of secondary infections before time x is $\Lambda(\nu)$. Hence,

$$M(x) = S_{T_{\text{cure}}}(x)\Lambda(x) + \int_0^x f_{T_{\text{cure}}}(v)\Lambda(v) dv \quad (7)$$

The total expected number of secondary infections caused by an infectious node is thus the basic reproduction number $R_0 = \lim_{x \rightarrow \infty} M(x)$, which we assume is finite. The distribution function $F_{T_{\text{gen}}}(x) = \Pr[T_{\text{gen}} < x]$ of the generation time is the fraction of secondary infections which occur before time x ,

$$F_{T_{\text{gen}}}(x) = \frac{M(x)}{R_0} \quad (8)$$

The probability density function $f_{T_{\text{gen}}}(x)$ follows as the derivative

$$f_{T_{\text{gen}}}(x) = \frac{d}{dx} F_{T_{\text{gen}}}(x) = \frac{\lambda(x)S_{T_{\text{cure}}}(x)}{R_0} \quad (9)$$

where $\lambda(x) = \frac{d}{dx} \Lambda(x)$ is the instantaneous rate at which an infectious node i produces infections at time x , given that node i has not cured yet. The infectivity profile in the SIS model is thus $m(x) = \lambda(x)S_{T_{\text{cure}}}(x)$.

In order to obtain the probability density function $f_{T_{\text{gen}}}(x)$, it is sufficient to find any function $\hat{\lambda}(x)$ which is proportional to $\lambda(x)$, because the same pdf is obtained after normalizing the area to 1. As discussed in the previous section, the hazard function $h_{T_{\text{inf}}}(x) = \frac{f_{T_{\text{inf}}}(x)}{S_{T_{\text{inf}}}(x)}$ represents the

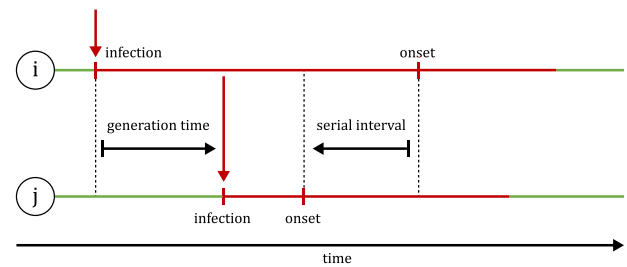


Fig. 1 | Timeline illustrating the generation time and serial interval. Node i and node j are both initially susceptible, indicated by green on the timeline. Node i then becomes infected, indicated by red on the timeline. The red arrow indicates the infection event. Node i then infects node j . The time difference between the infection events is the generation time, which is always positive. If people can transmit the disease before symptom onset, then the symptom onset of node i may possibly occur after that of node j , as illustrated in this example. The serial interval, defined as the time difference between symptom onset, may then be negative.

instantaneous rate at which an infectious node i infects a susceptible neighbor j at x time units after node i was infected (given that node i has not cured). Therefore, assuming that the number of susceptible neighbors is constant in time, the instantaneous rate at which an infectious node i produces secondary infections is proportional to the hazard function: $\hat{\lambda}(x) = h_{T_{\text{inf}}}(x)$. Under this assumption, the density function $f_{T_{\text{gen}}}(x)$ of the generation time in a Weibull SIS process follows as

$$f_{T_{\text{gen}}}(x) = \frac{1}{\Gamma\left(\frac{\alpha_{\text{inf}}}{\alpha_{\text{cure}}}\right)} \frac{\alpha_{\text{cure}}}{b_{\text{cure}}} \left(\frac{x}{b_{\text{cure}}}\right)^{\alpha_{\text{inf}}-1} e^{-\left(\frac{x}{b_{\text{cure}}}\right)^{\alpha_{\text{cure}}}} \quad (10)$$

where $\Gamma(x)$ denotes the gamma function [29, Ch. 6], α_{inf} is the infection time shape parameter, α_{cure} is the curing time shape parameter, and b_{cure} is the curing time scale parameter (see Supplementary Note 1 for the derivation).

In Supplementary Note 2, we assess the validity of (10) on different graphs (Supplementary Table S1) through computer simulations. On the complete graph, the measured generation times in simulations closely follows (10). On sparser graphs, (10) becomes less accurate because the assumption of a constant number of susceptible neighbors becomes worse due to the smaller number of neighbors (Supplementary Fig. S1). However, simulations show that on sparse graphs, (10) is accurate in the shape but not the scale, meaning that the error between (10) and the measurements can be almost completely removed by tuning the scale parameter b_{cure} (Supplementary Figs. S2–S4). Hence, (10) accurately describes the shape of the probability density of measured generation times, regardless of graph topology. Figure 2 shows a comparison of (10) with the measured density function of the generation time from computer simulations of a Weibull SIS process on the complete graph of $N = 100$ nodes for several infection time shape parameters. The solid lines show the theoretical density function (10) of the generation time and the points show the measured density from the simulation, which closely follows the theory.

In studies of biological epidemics, measured generation times are often fitted to a Weibull probability density function $\hat{f}_{T_{\text{gen}}}(x)$ with shape parameter α_{gen} . We therefore need to understand how the fitted generation time shape parameter α_{gen} is related to the underlying infection time shape parameter α_{inf} and curing time shape parameter α_{cure} . We consider infection time and curing time shape parameters in the range $\alpha_{\text{inf}}, \alpha_{\text{cure}} \in \{0.3, 0.4, \dots, 3.0\}$ and set the curing time scale parameter arbitrarily as

$$b_{\text{cure}} = \frac{\Gamma\left(\frac{\alpha_{\text{inf}}}{\alpha_{\text{cure}}}\right)}{\Gamma\left(\frac{\alpha_{\text{inf}}+1}{\alpha_{\text{cure}}}\right)}$$

such that the mean generation time $\mathbb{E}[T_{\text{gen}}] = 1$. We then fit a Weibull probability density function $\hat{f}_{T_{\text{gen}}}(x)$ to (10). Figure 3a shows the fitted generation time shape parameter α_{gen} and Fig. 3b shows the error ϵ defined as the total variation distance (TVD) between the probability densities

$$\epsilon = \frac{1}{2} \int_0^\infty |f_{T_{\text{gen}}}(x) - \hat{f}_{T_{\text{gen}}}(x)| dx$$

If the infection time shape parameter and curing time shape parameter are the same, $\alpha_{\text{inf}} = \alpha_{\text{cure}}$, then (10) is precisely a Weibull distribution. Therefore, on the diagonal of Fig. 3a, the generation time shape parameter

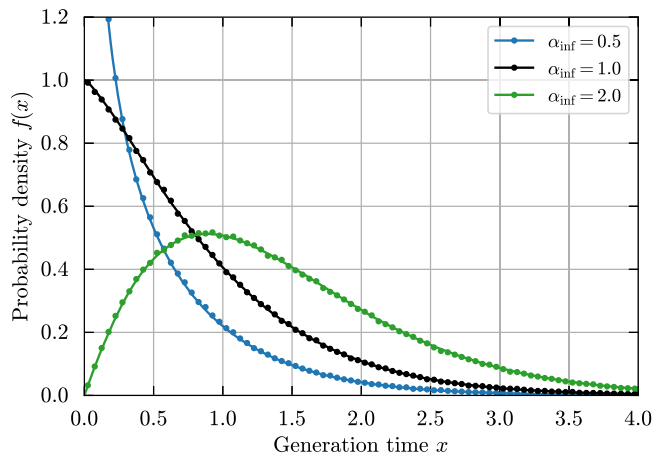


Fig. 2 | Comparison of the theoretical density function of the generation time with the measured density from computer simulations. The theoretical density function $f_{T_{\text{gen}}}(x)$ is indicated by the solid lines and the measured density is indicated by the points. Both the infection time T_{inf} and curing time T_{cure} are Weibull distributed. Three infection time shape parameters $\alpha_{\text{inf}} \in \{0.5, 1, 2\}$ are considered, while the curing time shape parameter is kept at $\alpha_{\text{cure}} = 1.3$. The scale parameters b_{inf} and b_{cure} are set such that the mean infection time $\mathbb{E}[T_{\text{inf}}] = 0.5$ and the mean curing time $\mathbb{E}[T_{\text{cure}}] = 1$ respectively. The simulations are performed on a complete graph of $N = 100$ nodes. The SIS process is modified to remove the all-healthy absorbing state by preventing the last infectious node from curing as in³². Each measured histogram is built from 10^6 measurements of the generation time.

$\alpha_{\text{gen}} = \alpha_{\text{inf}} = \alpha_{\text{cure}}$ and the total variation distance is 0 on the diagonal of Fig. 3b.

If the infection time shape parameter and curing time shape parameter are different, then (10) can be approximately described by a Weibull distribution with the generation time shape parameter α_{gen} somewhere between the infection time shape parameter α_{inf} and curing time shape parameter α_{cure} . When moving away from the diagonal of Fig. 3, the difference between the infection time shape parameter α_{inf} and curing time shape parameter α_{cure} increases and so does the error. In Supplementary Figs. S5–S6, we fit measured generation times from computer simulations with a Weibull distribution. The results are consistent with Fig. 3 and the fitted generation time shape parameter α_{gen} is not affected by graph topology.

The generation time shape parameter α_{gen} increases when the infection time shape parameter α_{inf} or curing time shape parameter α_{cure} increases. The green cells in Fig. 3a show when the fitted generation time shape parameter $\alpha_{\text{gen}} > 1$, which almost always occurs when the infection time shape parameter $\alpha_{\text{inf}} > 1$. This is intuitive because a Weibull distribution with $\alpha_{\text{gen}} > 1$ is bell-shaped and (10) is bell-shaped when $\alpha_{\text{inf}} > 1$. Hence, if the generation time T_{gen} can be approximately described by a Weibull distribution with shape parameter $\alpha_{\text{gen}} > 1$, then the underlying Weibull SIS process likely has infection time shape parameter $\alpha_{\text{inf}} > 1$.

If the infection time shape parameter α_{inf} is less than 1, then it is possible that the fitted generation time shape parameter $\alpha_{\text{gen}} > 1$ if the curing time shape parameter α_{cure} is sufficiently large. Realistically, a Weibull SIS process with an infection time $\alpha_{\text{inf}} < 1$ cannot yield a generation time shape parameter α_{gen} substantially larger than 1 because if $\alpha_{\text{cure}} \gg \alpha_{\text{inf}}$, then the generation time density (10) cannot be modeled with a Weibull distribution. This can be seen in Fig. 3a where the black outline shows when the TVD $\epsilon < 0.05$. If we consider the entries where the infection time shape parameter $\alpha_{\text{inf}} \leq 1$ and TVD $\epsilon < 0.05$, then the largest fitted generation time shape parameter $\alpha_{\text{gen}} = 1.3$. Therefore, if the generation time shape parameter α_{gen} is substantially larger than 1, then we can be almost certain that the underlying Weibull SIS process has infection time shape parameter $\alpha_{\text{inf}} > 1$.

Measurements of the generation time shape parameter α_{gen} for several diseases

The Weibull distribution (4) is a commonly accepted distribution to approximate generation times measured in biological epidemics. Our idea is to measure the generation time shape parameter α_{gen} as a characterizer of non-Markovianity in biological epidemics. In Supplementary Table S2, we

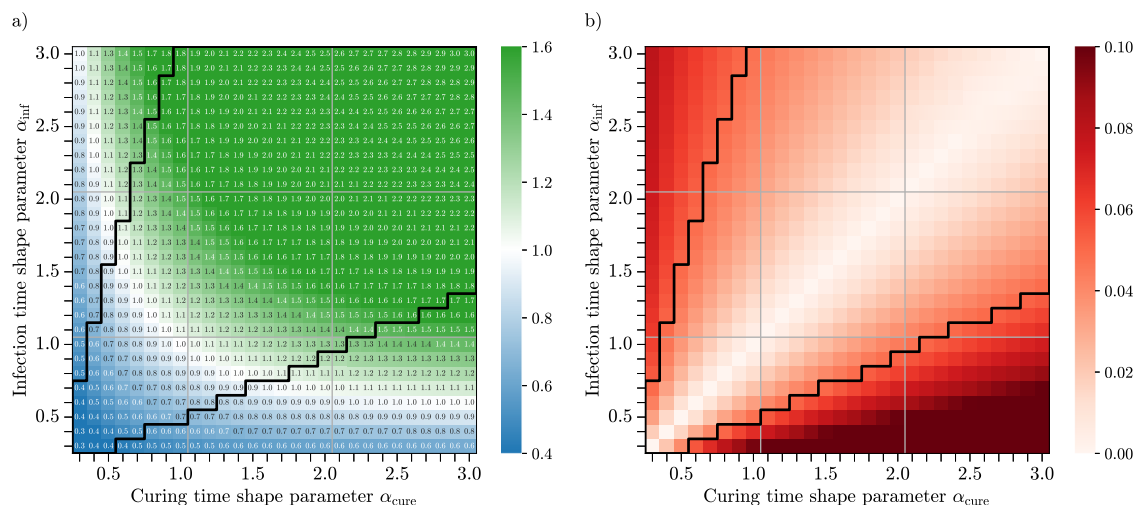


Fig. 3 | Shape parameter and error when fitting the theoretical density function of the generation time with a Weibull distribution. **a** Fitted generation time shape parameter α_{gen} . White indicates $\alpha_{\text{gen}} = 1$, green indicates $\alpha_{\text{gen}} > 1$, and blue indicates $\alpha_{\text{gen}} < 1$. **b** Corresponding error of the Weibull fit, defined as the total variation

distance $\epsilon = \frac{1}{2} \int_0^\infty |f_{T_{\text{gen}}}(x) - \hat{f}_{T_{\text{gen}}}(x)| dx$ between the theoretical density function $f_{T_{\text{gen}}}(x)$ and the fitted Weibull density function $\hat{f}_{T_{\text{gen}}}(x)$. Darker shades of red indicate higher error. The black outline in each panel shows where the total variation distance $\epsilon < 0.05$.

have extracted the shape parameter α_{gen} from a large number of published biological disease measurements (in total 126). Details on the data collection and method for extracting the shape parameter are presented in Supplementary Notes 3–4 and Supplementary Figs. 7–8. If the publication reports the raw data, then we fit a Weibull distribution to the reported data. Otherwise, we infer the Weibull shape parameter from the reported mean and standard deviation.

A histogram of the shape parameters is plotted in Fig. 4. Generation times or serial intervals measured in biological respiratory epidemics typically exhibit a bell shape, corresponding to a shape parameter of $\alpha_{\text{gen}} > 1$ when fitted with a Weibull distribution.

Available empirical data suggests that $\alpha_{\text{gen}} < 1$ is not biologically plausible for generation times of person-to-person infectious diseases. In a human challenge study of influenza, investigators experimentally infect people with live influenza virus. In a review of human challenge studies, Carrat et al.²⁰ showed that the day after infection, people begin shedding influenza virus from their respiratory tract, and they showed that the amount of shedding peaks two days after infection. These findings are consistent with a relatively low probability density of short generation intervals, such as the Weibull distribution with a shape parameter $\alpha_{\text{gen}} > 1$. In a case-ascertained household study, people seek care for an illness; and later, investigators visit the home to collect information from all members of the household. In a careful analysis of serial intervals of influenza virus infection from a case-ascertained household study, Cowling et al.¹⁹ found the best fit was a Weibull distribution with a mean of 3.6 days and a standard deviation of 1.6 days. This translates to a shape parameter of about $\alpha_{\text{gen}} = 2.4$. Chowell and Nishiura²⁰ fit a Weibull distribution to serial intervals for Ebola virus disease. They found a mean serial interval of 12 days with a shape parameter of $\alpha_{\text{gen}} = 2.6$, with a 95% confidence interval of 1.8–3.5. Vink, Bootsma and Wallinga³¹ present a systematic review of all serial interval data published as of 1 October, 2013 about influenza, respiratory syncytial virus, severe acute respiratory syndrome, measles, varicella, smallpox, mumps, rubella, pertussis, and tuberculosis. By matching the mean and variance of their results to a Weibull distribution, we again find that the shape parameter $\alpha_{\text{gen}} > 1$.

In the previous section, we demonstrated that a Weibull distributed generation time T_{gen} with shape parameter α_{gen} implies a comparable shape parameter α_{inf} for a Weibull distributed infection time T_{inf} . In other words, if $\alpha_{\text{gen}} > 1$, then it holds that $\alpha_{\text{inf}} > 1$, and vice versa, if $\alpha_{\text{gen}} < 1$, then it holds that $\alpha_{\text{inf}} < 1$. Hence, in biological and mainly respiratory epidemics, measurements in Fig. 4 reveal that the infection time T_{inf} typically has shape

parameter $\alpha_{\text{inf}} > 1$, corresponding to a slower spread than Markovian epidemics ($\alpha_{\text{inf}} = 1$).

Conclusions

After explaining Markovian and non-Markovian SIS epidemic theory, we conclude that a generation time shape parameter $\alpha_{\text{gen}} > 1$ implies an infection time shape parameter $\alpha_{\text{inf}} > 1$ when modeled with a Weibull distribution. The non-Markovian SIS infection probability with $\alpha > 1$ is upper bounded by the Markovian ($\alpha = 1$) nodal infection probability and the basic reproduction number with $\alpha > 1$ is upper bounded by the Markovian ($\alpha = 1$) basic reproduction number (while the corresponding epidemic threshold $\tau_c^{(1)} = \frac{1}{\lambda_1}$). A literature survey of published generation times and serial intervals in real-world person-to-person infections reveals that the shape parameter $\alpha > 1$ in most cases. Hence, Markovian models provide a safe upper bound.

The literature survey includes primarily respiratory pathogens, no sexually transmitted infections, and no blood-borne infections. Therefore, the development of a still missing $0 < \alpha < 1$ non-Markovian theory for biological respiratory epidemics does not seem needed anymore. For some infections, such as HIV, the idea of a bell-shaped distribution ($\alpha > 1$) for generation time might be more contentious. Hence, our claim only holds for pathogens spread by respiratory droplets, but not for all biological diseases. A practical limitation is that our theory is based on the generation time shape parameter α_{gen} while in practice, serial intervals are often measured. In cases where serial intervals are a poor proxy for generation times, one should not assume that the shape of the serial interval is comparable to that of the generation time; the distribution of the generation time should be recovered before drawing any conclusions about the underlying infection process. Finally, we have confined our treatment to SIS epidemics which upper-bound SIR epidemics in the nodal infection probability and in the basic reproduction number. For diseases where re-infections are rare, the SIS bounds may be excessively safe and therefore not of practical use when deciding on intervention measures in real-world epidemics.

Data availability

The data that support the findings of this study are available from the corresponding author upon request.

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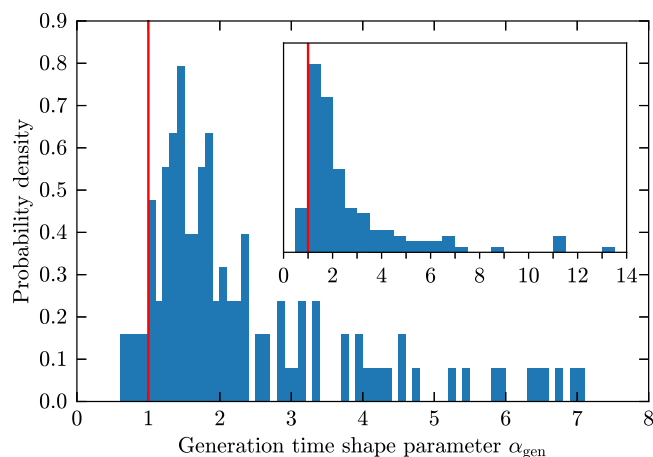


Fig. 4 | Histogram of generation time shape parameters collected from the epidemiological literature. The main figure uses a bin width of 0.1 and shows values up to $\alpha_{\text{gen}} = 8$. The inset uses a bin width of 0.5 and shows the full range of results. The red line in the main figure and inset indicates $\alpha_{\text{gen}} = 1$. Generation time shape parameter $\alpha_{\text{gen}} > 1$ implies an infection time shape parameter $\alpha_{\text{inf}} > 1$ in the underlying SIS process and thus a slower disease spread compared to the Markovian case ($\alpha = 1$).

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Author contributions

B.L.C. and P.V.M. contributed equally to this work. P.V.M. conceived the study. B.L.C. and P.V.M. designed research. B.L.C. performed simulations. B.L.C. and P.V.M. wrote the paper.

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Competing interests

The authors declare no competing interests.

Additional information

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Supplementary Information for Classical mean-field epidemic theory upper-bounds real disease spread

Brian L. Chang and Piet Van Mieghem

May 11, 2026

Supplementary Note 1: Derivation of the density function of generation time in the Weibull SIS process

In the main article, we deduced that the generation time T_{gen} in SIS epidemics has density function proportional to $h_{T_{\text{inf}}}(x)S_{T_{\text{cure}}}(x)$, where $h_{T_{\text{inf}}}(x)$ is the hazard function of the infection time T_{inf} and $S_{T_{\text{cure}}}(x)$ is the survival function of the curing time T_{cure} . For a Weibull distributed infection time T_{inf} with shape parameter α_{inf} and scale parameter b_{inf} , the hazard function is given by

$$h_{T_{\text{inf}}}(x) = \frac{\alpha_{\text{inf}}}{b_{\text{inf}}} \left(\frac{x}{b_{\text{inf}}} \right)^{\alpha_{\text{inf}}-1} \quad (\text{S1})$$

For a Weibull distributed curing time T_{cure} with shape parameter α_{cure} and scale parameter b_{cure} , the survival function is given by

$$S_{T_{\text{cure}}}(x) = e^{-\left(\frac{x}{b_{\text{cure}}}\right)^{\alpha_{\text{cure}}}} \quad (\text{S2})$$

Hence, the density function $f_{T_{\text{gen}}}(x)$ of the generation time T_{gen} in the Weibull SIS process can be written as

$$f_{T_{\text{gen}}}(x) = \xi^{-1} \frac{\alpha_{\text{inf}}}{b_{\text{inf}}} \left(\frac{x}{b_{\text{inf}}} \right)^{\alpha_{\text{inf}}-1} e^{-\left(\frac{x}{b_{\text{cure}}}\right)^{\alpha_{\text{cure}}}} \quad (\text{S3})$$

where ξ is a normalization constant given by

$$\xi = \int_0^\infty \frac{\alpha_{\text{inf}}}{b_{\text{inf}}} \left(\frac{x}{b_{\text{inf}}} \right)^{\alpha_{\text{inf}}-1} e^{-\left(\frac{x}{b_{\text{cure}}}\right)^{\alpha_{\text{cure}}}} dx \quad (\text{S4})$$

Solving (S4) using the substitution $v = \left(\frac{x}{b_{\text{cure}}}\right)^{\alpha_{\text{cure}}}$ yields

$$\xi = \frac{\alpha_{\text{inf}}}{\alpha_{\text{cure}}} \left(\frac{b_{\text{cure}}}{b_{\text{inf}}} \right)^{\alpha_{\text{inf}}} \int_0^\infty v^{\left(\frac{\alpha_{\text{inf}}}{\alpha_{\text{cure}}}-1\right)} e^{-v} dv = \frac{\alpha_{\text{inf}}}{\alpha_{\text{cure}}} \left(\frac{b_{\text{cure}}}{b_{\text{inf}}} \right)^{\alpha_{\text{inf}}} \Gamma\left(\frac{\alpha_{\text{inf}}}{\alpha_{\text{cure}}}\right) \quad (\text{S5})$$

where $\Gamma(x)$ denotes the gamma function [1, Ch. 6]. Substituting (S5) into (S3) yields the density function $f_{T_{\text{gen}}}(x)$ of the generation time in the Weibull SIS process

$$f_{T_{\text{gen}}}(x) = \frac{1}{\Gamma\left(\frac{\alpha_{\text{inf}}}{\alpha_{\text{cure}}}\right)} \frac{\alpha_{\text{cure}}}{b_{\text{cure}}} \left(\frac{x}{b_{\text{cure}}} \right)^{\alpha_{\text{inf}}-1} e^{-\left(\frac{x}{b_{\text{cure}}}\right)^{\alpha_{\text{cure}}}} \quad (\text{S6})$$

Note that the infection time scale parameter b_{inf} does not appear in (S6). The distribution function $F_{T_{\text{gen}}}(x)$ of the generation time follows by integrating (S6) using the substitution $v = (\frac{x}{b_{\text{cure}}})^{\alpha_{\text{cure}}}$ which yields

$$F_{T_{\text{gen}}}(x) = \int_0^x f_{T_{\text{gen}}}(x) dx = \frac{1}{\Gamma\left(\frac{\alpha_{\text{inf}}}{\alpha_{\text{cure}}}\right)} \int_0^{\left(\frac{x}{b_{\text{cure}}}\right)^{\alpha_{\text{cure}}}} v^{\left(\frac{\alpha_{\text{inf}}}{\alpha_{\text{cure}}}-1\right)} e^{-v} dv = 1 - \frac{\Gamma\left(\frac{\alpha_{\text{inf}}}{\alpha_{\text{cure}}}, \left(\frac{x}{b_{\text{cure}}}\right)^{\alpha_{\text{cure}}}\right)}{\Gamma\left(\frac{\alpha_{\text{inf}}}{\alpha_{\text{cure}}}\right)} \quad (\text{S7})$$

where $\Gamma(s, x)$ is the upper incomplete gamma function [2, eq. (8.2.2)]. The n -th moment of the generation time is found by integrating $x^n f_{T_{\text{gen}}}(x)$ using the substitution $v = (\frac{x}{b_{\text{cure}}})^{\alpha_{\text{cure}}}$ which yields

$$\mathbb{E}[T_{\text{gen}}^n] = \int_0^\infty x^n f_{T_{\text{gen}}}(x) dx = \frac{b_{\text{cure}}^n}{\Gamma\left(\frac{\alpha_{\text{inf}}}{\alpha_{\text{cure}}}\right)} \int_0^\infty v^{\left(\frac{\alpha_{\text{inf}}+n}{\alpha_{\text{cure}}}-1\right)} e^{-v} dv = b_{\text{cure}}^n \frac{\Gamma\left(\frac{\alpha_{\text{inf}}+n}{\alpha_{\text{cure}}}\right)}{\Gamma\left(\frac{\alpha_{\text{inf}}}{\alpha_{\text{cure}}}\right)} \quad (\text{S8})$$

Supplementary Note 2: Assessing the validity of the theoretical density function of generation time

The assumption made in deriving the theoretical density function (S6) of the generation time is that the expected number of susceptible neighbors of an infectious node is constant in time. To assess the validity of this assumption, we compare the theoretical density function (S6) with the measured generation time $\hat{T}_{\text{gen}}$ in SIS simulations.

In the main article, we claim that if the measured generation time $\hat{T}_{\text{gen}}$ is described by a Weibull distribution with shape parameter $\alpha_{\text{gen}} > 1$, then the infection time shape parameter α_{inf} of the underlying SIS process is also most likely larger than 1. Here, we also fit the measured generation time $\hat{T}_{\text{gen}}$ to a Weibull distribution and provide the basis for this claim.

Simulation method

The SIS simulations are performed using a standard discrete-event simulation method based on [3]. The starting states (compartments) of the nodes are first initialized. Then, according to the state of each node, infection events and curing events are generated which are sorted in a queue. In each simulation step, the earliest event is removed from the queue, the states of the nodes are updated, and new events are generated and sorted into the queue. The SIS process is modified to remove the all-healthy absorbing state by preventing the last infectious node from curing as in [4]. If the last infectious node i cures before infecting any other node, we instantly re-infect node i and the simulation continues.

Simulation parameters

We tested eight classes of graphs on $N = 100$ nodes: the complete graph, a random regular graph with degree 10, a Barabási-Albert graph (constructed starting from a star graph on 6 nodes and adding 5 links in each step), an Erdős-Rényi graph with 475 links, a 10×10 square lattice, a ring graph, a random tree, and a star graph. We chose these graphs in order to have a mix of sparse, dense, random, and structured graphs. The number of links in each graph is summarized in Table S1.

Table S1: Number of links in each tested graph.

Graph	Number of links	Link density
Complete graph	4950	1
Random regular	500	0.101
Barabási-Albert	475	0.096
Erdős-Rényi	475	0.096
Square lattice	180	0.036
Ring graph	100	0.020
Random tree	99	0.020
Star graph	99	0.020

On each graph, we consider the shape parameters $\alpha \in \{0.5, 0.9, 1, 1.1, 1.5\}$ for the infection time

T_{inf} and curing time T_{cure} . When varying the shape parameters, we keep the mean infection time constant at $\mathbb{E}[T_{\text{inf}}] = 0.5$ and the mean curing time constant at $\mathbb{E}[T_{\text{cure}}] = 1$ by adjusting the scale parameter of the Weibull distribution accordingly. Each simulation is stopped after 10^6 measurements of the generation time are obtained.

Comparison of theoretical density function with measured generation time

In each simulation, the measurements of the generation time $\hat{T}_{\text{gen}}$ are stored as a histogram with a bin width of $\Delta t = 0.05$. We compute the total variation distance (TVD) between the theoretical density function (S6) and the measured data as

$$\text{TVD} = \frac{1}{2} \sum_{k=1}^{\infty} |p_k - \hat{p}_k| \quad (\text{S9})$$

where

$$p_k = \int_{(k-1)\Delta t}^{k\Delta t} f_{T_{\text{gen}}}(x) dx \quad (\text{S10})$$

is the theoretical probability of observing a measurement in bin k and $\hat{p}_k$ is the fraction of actual measurements falling in bin k .

Figure S1 shows the TVD of the measured generation time in the 200 simulations. For each infection time shape parameter α_{inf} and curing time shape parameter α_{cure} , there are 8 rows which correspond to the different graphs sorted from highest to lowest link density. From top to bottom, the order is the complete graph, random regular graph, Barabási-Albert graph, Erdős-Rényi graph, square lattice, ring graph, random tree, and the star graph (the same order as in Table S1).

When the infection time is exponential $\alpha_{\text{inf}} = 1$, the measured generation time closely follows the theoretical density function (S6) across all of the graphs and curing time shape parameters α_{cure} . Across the 40 simulations with exponential infection time, the average TVD between the measured generation time and the theoretical density function (S6) is $5.0 \cdot 10^{-3}$. When the infection time shape parameter is close to 1, the TVD is slightly higher. For $\alpha_{\text{inf}} = 0.9$, the average TVD is $6.4 \cdot 10^{-3}$ and for $\alpha_{\text{inf}} = 1.1$, the average TVD is $8.9 \cdot 10^{-3}$.

On the complete graph (the first row in each block of Figure S1), the TVD is also very low. Across the 25 simulations on the complete graph, the average TVD between the measured distribution generation time and our theoretical density function (S6) is $5.3 \cdot 10^{-3}$. If we consider only the simulations with infection time shape parameter $\alpha_{\text{inf}} = 0.5$ and $\alpha_{\text{inf}} = 1.5$ in the complete graph (i.e. excluding the simulations where α_{inf} is close to 1), the average TVD is $6.9 \cdot 10^{-3}$. Hence, on the complete graph, our theoretical density function (S6) accurately describes the density of measured generation times. On other graphs, (S6) is still accurate if the infection time is exponential (or close to exponential).

Higher TVD is observed when the infection time is non-exponential on graphs other than the complete graph, and the error is worse when the graphs are sparse. This error is primarily due to a difference between the measured mean generation time $\mathbb{E}[\hat{T}_{\text{gen}}]$ in the simulations and the theoretical mean generation time $\mathbb{E}[T_{\text{gen}}]$ (derived in (S8)). In Figure S2, we plot the error $\epsilon = \frac{\mathbb{E}[T_{\text{gen}}] - \mathbb{E}[\hat{T}_{\text{gen}}]}{\mathbb{E}[\hat{T}_{\text{gen}}]}$ between the measured and theoretical mean generation time. Red cells show when the theoretical

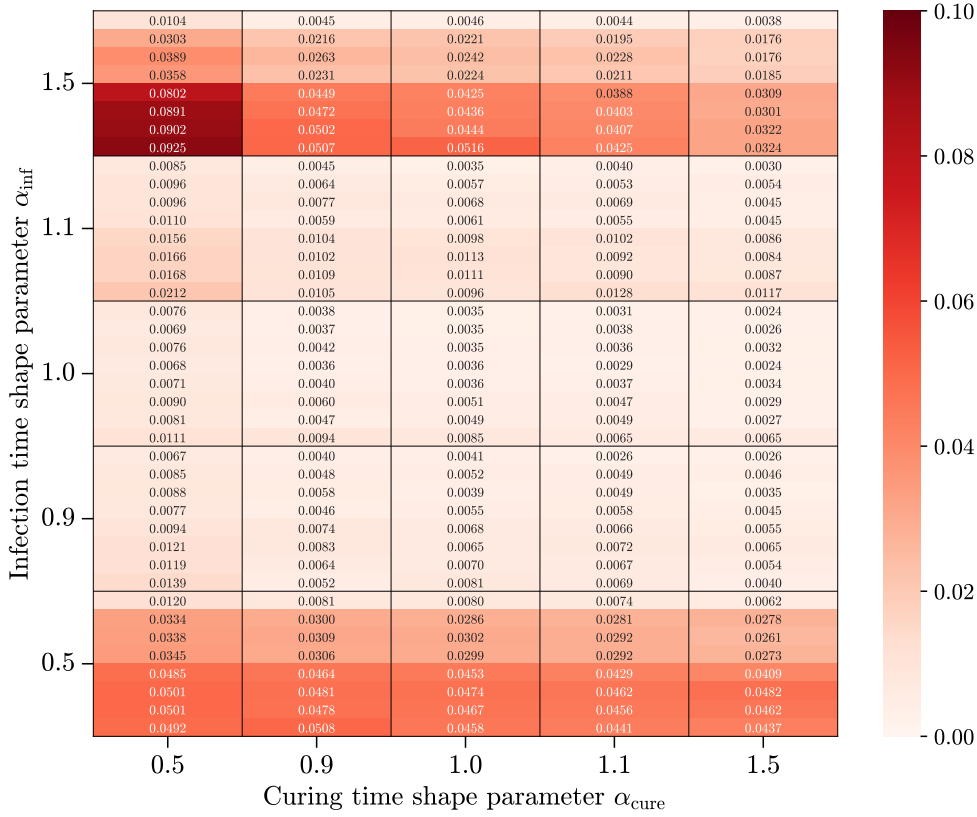


Figure S1: TVD between the theoretical density function (S6) of the generation time and the measured data. For each infection time shape parameter α_{inf} and curing time shape parameter α_{cure} , the rows (from top to bottom) correspond to the complete graph, random regular graph, Barabási-Albert graph, Erdős-Rényi graph, square lattice, ring graph, random tree, and the star graph.

mean is higher than the measurements and blue cells shows when the theoretical mean is lower than the measurements. When the infection time shape parameter $\alpha_{\text{inf}} > 1$, we overestimate the mean generation time on sparse graphs. When the infection time shape parameter $\alpha_{\text{inf}} < 1$, we underestimate the mean generation time on sparse graphs.

The difference in the mean is the main source of error which becomes apparent when we rescale our theoretical density function (S6) to have the same mean as the measured data. We replace the curing time shape parameter b_{cure} with an estimate $\hat{b}_{\text{cure}}$ computed from the measured data

$$\hat{b}_{\text{cure}} = \mathbb{E}[\hat{T}_{\text{gen}}] \frac{\Gamma(\frac{\alpha_{\text{inf}}}{\alpha_{\text{cure}}})}{\Gamma(\frac{\alpha_{\text{inf}}+1}{\alpha_{\text{cure}}})}$$

We then calculate the TVD between the rescaled theoretical density function and the measured generation time $\hat{T}_{\text{gen}}$ which is plotted in Figure S3. The TVD on sparse graphs decreases dramatically, confirming that the mismatch between the means is the primary source of error. Before rescaling, the worst error is observed on the star graph for $\alpha_{\text{inf}} = 1.5$ and $\alpha_{\text{cure}} = 0.5$ with a TVD of $9.2 \cdot 10^{-2}$ which is plotted in Figure S4. After rescaling, the TVD has decreased to $2.1 \cdot 10^{-2}$. This is a critical result because it shows that our theoretical density function (S6) still accurately captures the *shape* of the generation time density in sparse graphs; the error is mostly in the scale.

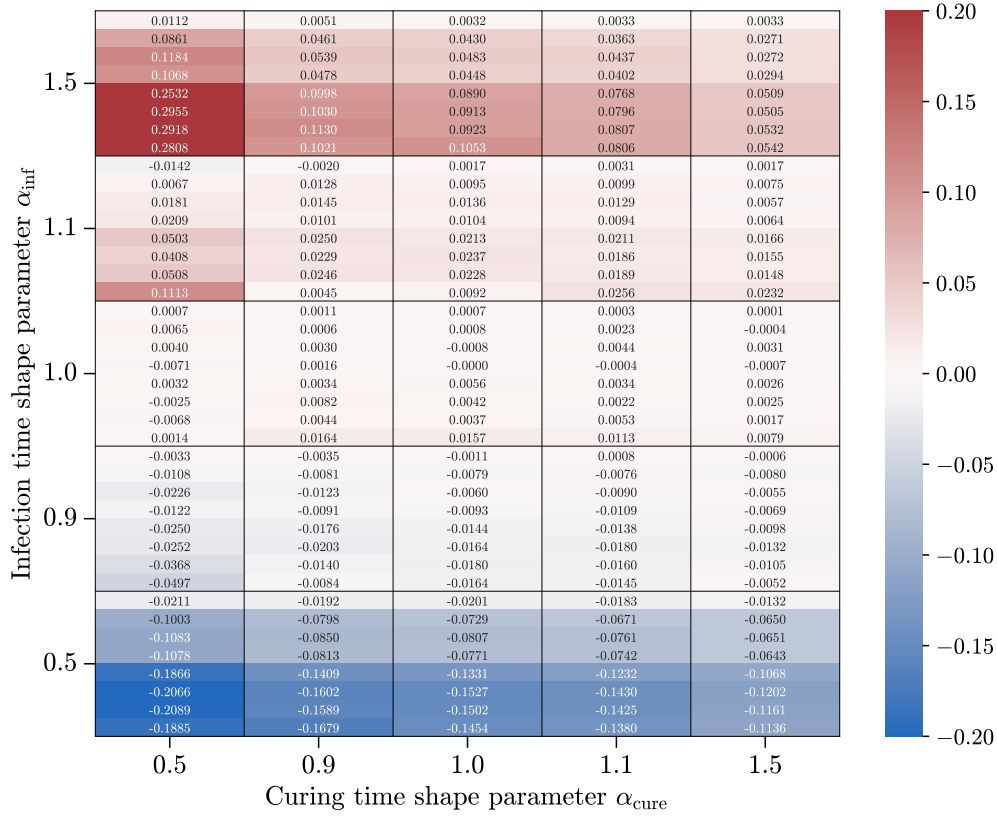


Figure S2: Error $\epsilon = \frac{\mathbb{E}[T_{\text{gen}}] - \mathbb{E}[\hat{T}_{\text{gen}}]}{\mathbb{E}[\hat{T}_{\text{gen}}]}$ between the theoretical mean generation time $\mathbb{E}[T_{\text{gen}}]$ and the measured mean generation time $\mathbb{E}[\hat{T}_{\text{gen}}]$. For each infection time shape parameter α_{inf} and curing time shape parameter α_{cure} , the rows (from top to bottom) correspond to the complete graph, random regular graph, Barabási-Albert graph, Erdős-Rényi graph, square lattice, ring graph, random tree, and the star graph.

Weibull fit of measured generation time

For each simulation, we fit the measured generation time $\hat{T}_{\text{gen}}$ with a Weibull distribution. Figure S5a shows the fitted shape parameter α_{gen} of the measured generation time and Figure S5b shows the TVD between the measurements and the Weibull fit. For each infection time shape parameter α_{inf} and curing time shape parameter α_{cure} , there are 8 rows which correspond to the different graphs sorted from highest to lowest link density (the same as in the previous section). From top to bottom, the order is the complete graph, random regular graph, Barabási-Albert graph, Erdős-Rényi graph, square lattice, ring graph, random tree, and the star graph.

We used the SciPy curve.fit method in Python to fit our measured data to a Weibull distribution using non-linear least squares regression. As seen in Figure S5a, for each infection time shape parameter α_{inf} and curing time shape parameter α_{cure} , the fitted Weibull shape parameter is almost the same on every graph. This is consistent with the results from the previous section where we observed that the link density and topology of graphs does not influence the shape of the generation time distribution.

Our theoretical density function (S6) for the generation time becomes a Weibull distribution when

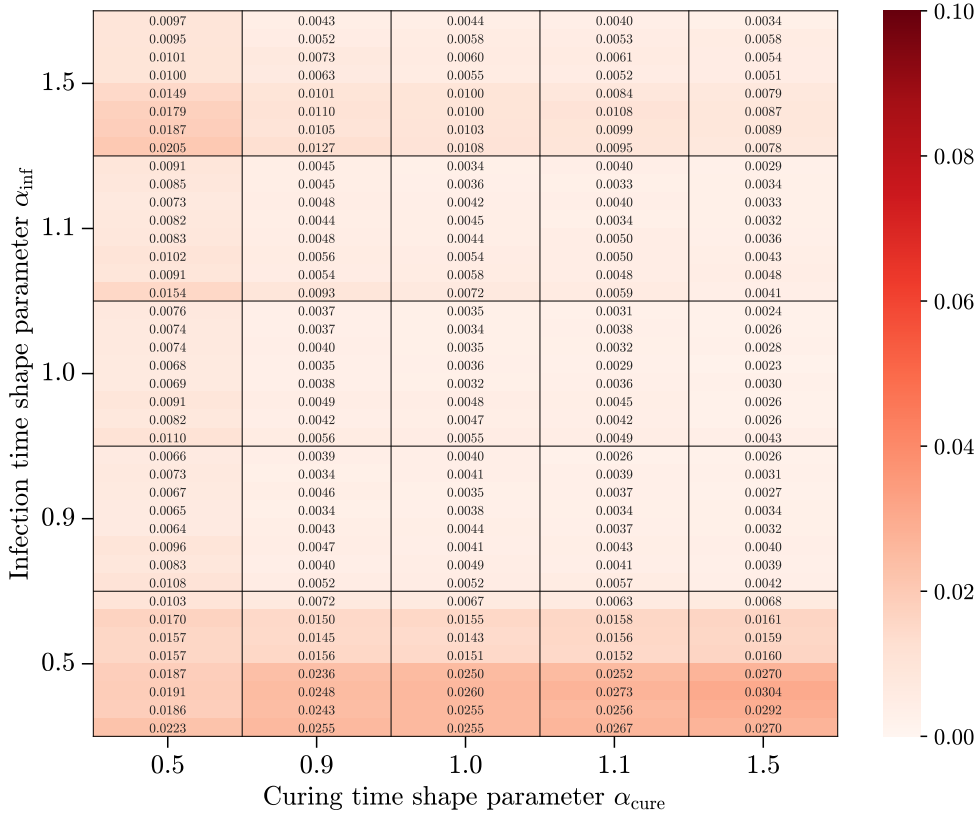


Figure S3: TVD between the theoretical density function (S6) of the generation time after rescaling and the measured data. For each infection time shape parameter α_{inf} and curing time shape parameter α_{cure} , the rows (from top to bottom) correspond to the complete graph, random regular graph, Barabási-Albert graph, Erdős-Rényi graph, square lattice, ring graph, random tree, and the star graph.

$\alpha_{\text{inf}} = \alpha_{\text{cure}}$, which is supported by the measurements. As seen in Figure S5b, the smallest TVD is observed when $\alpha_{\text{inf}} = \alpha_{\text{cure}}$ and the generation time shape parameter $\alpha_{\text{gen}} = \alpha_{\text{inf}} = \alpha_{\text{cure}}$. When $\alpha_{\text{inf}} \neq \alpha_{\text{cure}}$, we observe that the generation time shape parameter α_{gen} lies somewhere between the infection time shape parameter α_{inf} and curing time shape parameter α_{cure} .

In Figure S6, we plot the probability density of the measured generation time $\hat{T}_{\text{gen}}$ on the complete graph for infection time shape parameter $\alpha_{\text{inf}} \in \{0.5, 1, 1.5\}$ and curing time shape parameter $\alpha_{\text{cure}} \in \{0.5, 1, 1.5\}$ compared with the theoretical density function (S6) and a Weibull fit. The density of the measured generation time is bell-shaped when $\alpha_{\text{inf}} > 1$. Recall that (S6) is the normalized product of the infection time hazard function $h_{T_{\text{inf}}}(x)$ and the curing time survival function $S_{T_{\text{cure}}}(x)$. The infection time hazard function $h_{T_{\text{inf}}}(x)$ is monotonically decreasing in x for $\alpha_{\text{inf}} < 1$, constant for $\alpha_{\text{inf}} = 1$, and monotonically increasing in x for $\alpha_{\text{inf}} > 1$. The curing time survival function $S_{T_{\text{cure}}}(x)$ is always monotonically decreasing in x . Hence, (S6) is monotonically decreasing in x for $\alpha_{\text{inf}} \leq 1$ and only takes on a bell-shape if $\alpha_{\text{inf}} > 1$.

Summarizing, the generation time in a Weibull SIS process precisely follows a Weibull distribution when the curing time and infection time have the same shape parameter, in which case the generation time has shape parameter $\alpha_{\text{gen}} = \alpha_{\text{inf}} = \alpha_{\text{cure}}$. Hence, if the measured generation time $\hat{T}_{\text{gen}}$ is

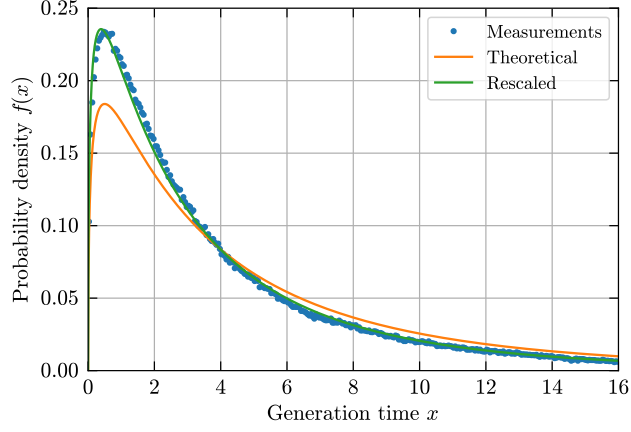
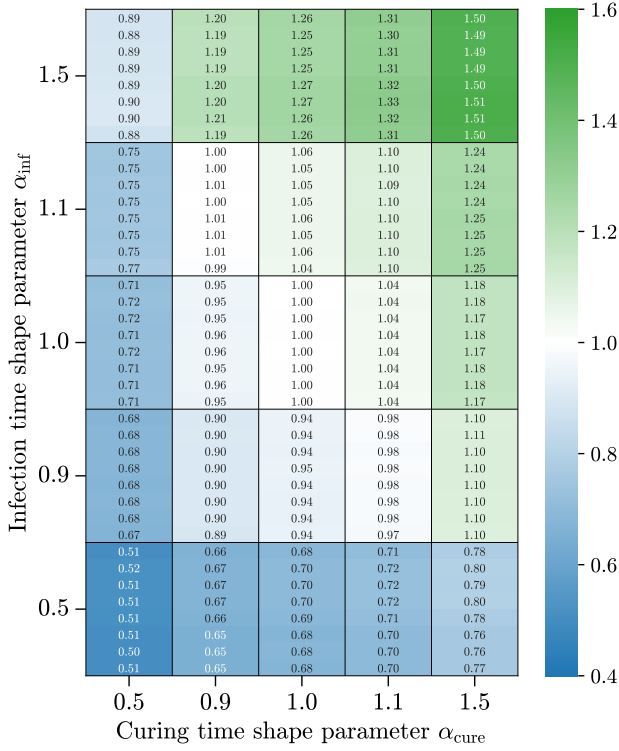
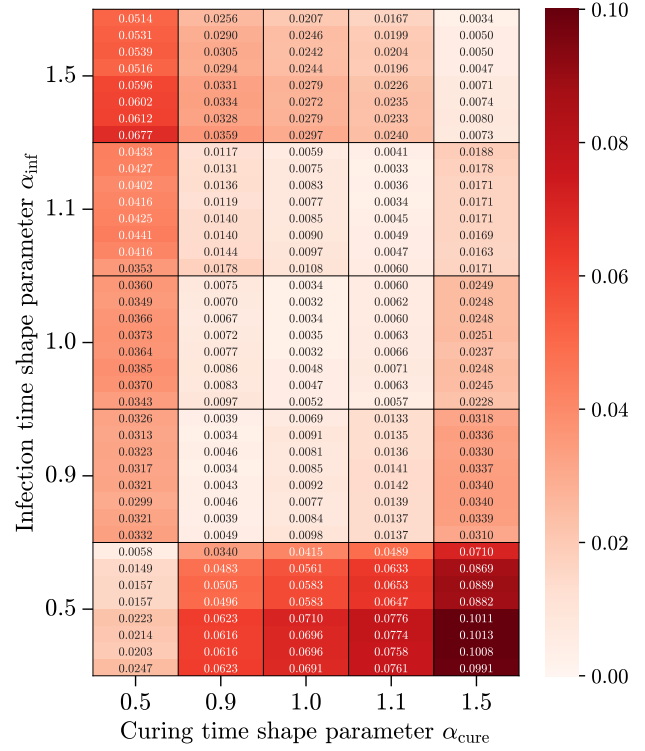


Figure S4: Probability density of the measured generation time $\hat{T}_{\text{gen}}$ on the star graph for infection time shape parameter $\alpha_{\text{inf}} = 1.5$ and curing time shape parameter $\alpha_{\text{cure}} = 0.5$ compared with the theoretical density function (S6) before and after rescaling.

(approximately) described by a Weibull distribution with shape parameter α_{gen} , then the infection time shape parameter α_{inf} and curing time shape parameter α_{cure} of the underlying SIS process should be somewhat close to the generation time shape parameter α_{gen} . In the previous section, we have demonstrated that the theoretical density function (S6) of the generation time accurately describes the shape of the measured generation time in a Weibull SIS process. If the measured density of the generation time is bell-shaped, i.e. described by a Weibull distribution with $\alpha_{\text{gen}} > 1$, then the underlying SIS process most likely has an infection time shape parameter $\alpha_{\text{inf}} > 1$ because (S6) is bell-shaped if and only if $\alpha_{\text{inf}} > 1$.



(a) Generation time shape parameter α_{gen} .



(b) TVD.

Figure S5: Fitted Weibull generation time shape parameter α_{gen} and the corresponding TVD between the Weibull fit and measured data. For each infection time shape parameter α_{inf} and curing time shape parameter α_{cure} , the rows (from top to bottom) correspond to the complete graph, random regular graph, Barabási-Albert graph, Erdős-Rényi graph, square lattice, ring graph, random tree, and the star graph.

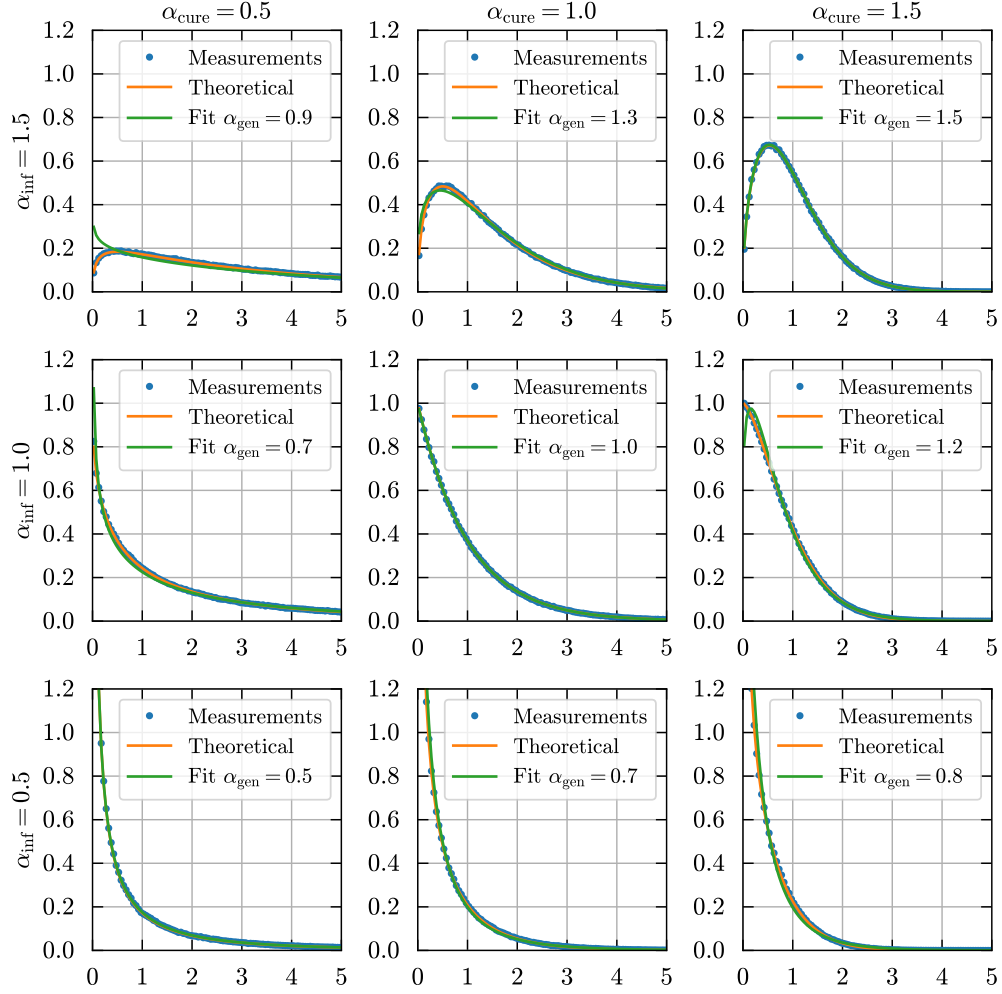


Figure S6: Probability density of the measured generation time $\hat{T}_{\text{gen}}$ on the complete graph for infection time shape parameter $\alpha_{\text{inf}} \in \{0.5, 1, 1.5\}$ and curing time shape parameter $\alpha_{\text{cure}} \in \{0.5, 1, 1.5\}$ compared with the theoretical density function (S6) and a Weibull fit.

Supplementary Note 3: Method for estimating the Weibull shape parameter α

Because published data on serial intervals or generation intervals are not always fit to a Weibull distribution, or because authors do not report the shape parameter, we infer the best shape and scale parameters of a Weibull distribution from the reported mean $\mathbb{E}[T]$ and variance $\text{Var}[T]$ of a measured time T . The mean and variance of a Weibull distributed time T are given by

$$\mathbb{E}[T] = b\Gamma\left(\frac{1}{\alpha} + 1\right)$$

and

$$\text{Var}[T] = b^2\left(\Gamma\left(\frac{2}{\alpha} + 1\right) - \Gamma^2\left(\frac{1}{\alpha} + 1\right)\right)$$

Hence, substituting $b = \frac{\mathbb{E}[T]}{\Gamma(\frac{1}{\alpha}+1)}$ yields

$$1 + \frac{\text{Var}[T]}{(\mathbb{E}[T])^2} = \frac{\Gamma(\frac{2}{\alpha} + 1)}{\Gamma^2(\frac{1}{\alpha} + 1)} \quad (\text{S11})$$

Given $w = 1 + \frac{\text{Var}[T]}{(\mathbb{E}[T])^2} \geq 1$, we aim to determine α from (S11). First, we show that there is a unique solution for α . Differentiation with respect to α yields

$$\frac{d}{d\alpha} \left(\frac{\Gamma(\frac{2}{\alpha} + 1)}{\Gamma^2(\frac{1}{\alpha} + 1)} \right) = \frac{2\Gamma(\frac{2}{\alpha} + 1)}{\alpha^2\Gamma^2(\frac{1}{\alpha} + 1)} \left(\psi\left(\frac{1}{\alpha} + 1\right) - \psi\left(\frac{2}{\alpha} + 1\right) \right) < 0$$

where the inequality follows from the strictly increasing nature [2, 5.11.13] of the digamma function $\psi(z)$ for real $z > 0$. Hence, $\frac{\Gamma(\frac{2}{\alpha}+1)}{\Gamma^2(\frac{1}{\alpha}+1)}$ is strictly monotonously decreasing with $\alpha > 0$ and a well-defined, unique solution for α in (S11) exists. A simple binary search algorithm can be used to calculate the shape parameter α numerically from (S11).

Supplementary Note 4: Remarks on the literature overview of published generation times

We searched PubMed publications for the terms “serial interval”, “generation interval”, or “generation time” with at least one of “influenza”, “RSV”, “SARS”, “measles”, “varicella”, “smallpox”, “mumps”, “rubella”, “pertussis”, “tuberculosis”, “SARS-CoV-2”, or “COVID-19”. We also searched the references of the results. Table S2 includes papers with sufficient information to estimate the shape α of generation times and serial intervals. If the publication reports the raw data, then we fit a Weibull distribution to the reported data. If only the mean and standard deviation is reported, then we determine the shape parameter α by solving (S11) using a binary search algorithm until a tolerance of 10^{-3} is reached. In total, there are 126 estimated shape parameters α in Table S2. We are aware of issues with misclassification of primary-secondary case pairs that actually are coprimary cases, primary-tertiary cases. A subset of the data sources has been analyzed with a method that accounts for this misclassification (e.g. as described in [5] and [6]) resulting in a mean² larger than variance, which suggests shape parameters $\alpha > 1$ if a Weibull distribution was used in the fit.

Figure S7 shows a plot of the mean and standard deviation, expressed in units of days, of the data in Table S2 where the lines indicate different shape parameters α . The black line corresponds to $\alpha = 1$ and any points below the black line correspond to shape parameter $\alpha > 1$. Almost all estimated shape parameters (118 out of 126) are $\alpha > 1$. Red points indicate data which has been fitted with a Weibull distribution and blue points indicate data which was fitted with a some other distribution from which we estimated the Weibull shape parameter α using (S11).

Figure S8 contains only the data from Table S2 which was fitted with a Weibull distribution (29 out of 126 entries). Error bars have been plotted if the publication reported confidence intervals for the mean and standard deviation. For the Weibull fitted data, all estimated shape parameters $\alpha > 1$.

Table S2: Estimates of mean serial intervals or generation times, and their shape parameter taken from the literature.

Reference		Disease	Interval	Fit	Mean	SD	Shape
Chowell 2014	[7, 8]	Ebola Virus	Serial	Weibull ^a	12	5.2	2.59
Chang 2004	[9]	Enterovirus 71	Serial	N/A	3.7	2.6	1.44
Cauchemez 2011	[10, 11]	Influenza	Serial	Gamma	2.6	1.5	1.79
Cowling 2009	[12]	Influenza	Serial	Weibull	3.6	1.6	2.40
White 2008	[10, 13]	Influenza	Serial	Gamma	2.6	1.5	1.79
Cauchemez 2009	[5, 14]	Influenza A(H1N1)pdm09	Serial	Normal	2.1	1.2	1.81
France 2010	[5, 15]	Influenza A(H1N1)pdm09	Serial	Normal	3.0	0.9	3.71
Hahné 2009	[5, 16]	Influenza A(H1N1)pdm09	Serial	Normal	1.7	1.2	1.44
Morgan 2010	[5, 17]	Influenza A(H1N1)pdm09	Serial	Normal	3.7	1.1	3.75
Papenburg 2011	[5, 18]	Influenza A(H1N1)pdm09	Serial	Normal	2.9	1.2	2.60
Savage 2011	[5, 19]	Influenza A(H1N1)pdm09	Serial	Normal	2.8	0.8	3.92
Viboud 2004	[5, 20]	Influenza A(H3N2)	Serial	Normal	2.2	0.8	3.00
Aaby 1990	[5, 21]	Measles	Serial	Normal	9.9	2.4	4.70
Bailey 1954	[5, 22]	Measles	Serial	Normal	10.9	1.9	6.73
Chapin 1925	[5, 23]	Measles	Serial	Normal	11.9	2.6	5.27
Fine 2003	[5, 24]	Measles	Serial	Normal	13.7	1.5	11.05
Fine 2003	[5, 24]	Measles	Serial	Normal	13.8	2.5	6.45
Groendyke 2012	[10, 25]	Measles	Serial	Gamma	14.9	3.9	4.32
Hope Simpson 1952	[5, 26]	Measles	Serial	Normal	10.9	2.0	6.37
Hope Simpson 1952	[5, 26]	Mumps	Serial	Normal	18.0	3.5	5.98
de Greef 2010	[5, 27]	Pertusis	Serial	Normal	22.8	6.5	3.93
Crowcroft 2008	[5, 28]	RSV	Serial	Normal	7.5	2.1	4.01
Aycock 1946	[5, 29]	Rubella	Serial	Normal	18.3	2.0	11.07
Cori 2009	[10, 30]	SARS-CoV-1	Serial	Gamma	8.4	3.8	2.35
Abudunaibi 2023	[31]	SARS-CoV-2 Delta	Serial	N/A	2.3	3.4	0.69
Abudunaibi 2023	[31]	SARS-CoV-2 Omicron	Serial	N/A	2.9	2.4	1.21
an der Heiden 2022	[32, 33]	SARS-CoV-2 Alpha	Serial	Gamma	4.50	3.21	1.42
an der Heiden 2022	[32, 33]	SARS-CoV-2 Delta	Serial	Gamma	4.19	2.91	1.47
an der Heiden 2022	[32, 33]	SARS-CoV-2 Omicron BA.1/BA.2	Serial	Gamma	3.61	2.48	1.48
an der Heiden 2022	[32, 33]	SARS-CoV-2 Wild Type	Serial	Gamma	4.80	3.38	1.44
Backer 2022	[32, 34]	SARS-CoV-2 Delta	Serial	Weibull ^b (shift 5)	3.69	2.84	3.37
Backer 2022	[32, 34]	SARS-CoV-2 Delta	Serial	Weibull ^b (shift 3)	3.32	2.55	2.67
Backer 2022	[32, 34]	SARS-CoV-2 Omicron BA.1	Serial	Weibull ^b (shift 4)	3.11	2.49	3.13
Backer 2022	[32, 34]	SARS-CoV-2 Omicron BA.1	Serial	Weibull ^b (shift 3)	3.00	2.21	2.96
Bendall 2023	[32, 35]	SARS-CoV-2 Alpha	Serial	Weibull ^b	2.60	1.70	1.57
Bendall 2023	[32, 35]	SARS-CoV-2 Delta	Serial	Log-normal	3.41	2.36	1.47
Bendall 2023	[32, 35]	SARS-CoV-2 Delta	Serial	Weibull ^b	4.23	2.90	1.49
Bendall 2023	[32, 35]	SARS-CoV-2 Omicron BA.1	Serial	Log-normal	2.92	2.55	1.15
Bendall 2023	[32, 35]	SARS-CoV-2 Omicron BA.1	Serial	Weibull ^b	3.96	3.04	1.32
Chen 2022	[32, 36]	SARS-CoV-2 Omicron BA.2	Serial	Gamma	3.2	1.7	1.96
Chen 2024	[37]	SARS-CoV-2 Delta	Generation	Log-normal	2.65	1.38	2.01
Chen 2024	[37]	SARS-CoV-2 Delta	Serial	Weibull	3.90	2.21	1.83
Del Águila-Mejía 2022	[32, 38]	SARS-CoV-2 Delta	Serial	N/A	5.4	3.1	1.80
Del Águila-Mejía 2022	[32, 38]	SARS-CoV-2 Omicron BA.1	Serial	N/A	4.8	3.0	1.64
Du 2020	[39, 40]	SARS-CoV-2	Serial	Normal	3.96	4.75	0.84
Ganyani 2020	[39, 41]	SARS-CoV-2	Generation	Gamma	5.20	1.72	3.33
Ganyani 2020	[39, 41]	SARS-CoV-2	Generation	Gamma	3.95	1.51	2.84
Ganyani 2020	[39, 41]	SARS-CoV-2	Serial	Other	5.21	4.32	1.21
Ganyani 2020	[39, 41]	SARS-CoV-2	Serial	Other	3.95	4.24	0.93
Guo 2022	[32, 42]	SARS-CoV-2 Delta	Serial	Gamma	5.8	3.9	1.52
Guo 2022	[32, 42]	SARS-CoV-2 Omicron BA.1/BA.2	Serial	Gamma	4.4	3.3	1.35
Guo 2023	[32, 43]	SARS-CoV-2 Omicron BA.2.12.1	Serial	Gamma	2.4	2.3	1.04
Guo 2023	[32, 43]	SARS-CoV-2 Omicron BA.4	Serial	Gamma	2.1	1.6	1.32

^aShape parameter taken from publication.

^bOur Weibull fit to the published raw data.

^cAlternative Weibull fit presented in publication.

Table S2: (Continued from previous page) – Estimates of mean serial intervals or generation times, and their shape parameter taken from the literature.

Reference		Disease	Interval	Fit	Mean	SD	Shape
Guo 2023	[32, 43]	SARS-CoV-2 Omicron BA.5	Serial	Gamma	2.4	2.0	1.21
Hammond 2022	[44]	SARS-CoV-2	Serial	Weibull ^b	4.44	2.53	1.82
Hart 2022	[32, 45]	SARS-CoV-2 Alpha	Generation	Other	5.5	4.0	1.39
Hart 2022	[32, 45]	SARS-CoV-2 Delta	Generation	Other	4.7	3.3	1.45
Kim 2022	[32, 46]	SARS-CoV-2 Omicron BA.1	Serial	Normal	3.78	3.33	1.14
Kim 2022	[32, 46]	SARS-CoV-2 Omicron BA.1	Serial	Weibull ^c	3.74	1.00	4.22
Kim 2023	[47]	SARS-CoV-2	Generation	Gamma	4.3	0.6	8.54
Kim 2023	[47]	SARS-CoV-2	Serial	Other	4.3	4.2	1.02
Kremer 2022	[32, 48]	SARS-CoV-2 Delta	Serial	Normal	3.00	2.48	1.22
Kremer 2022	[32, 48]	SARS-CoV-2 Omicron BA.1	Serial	Normal	2.75	2.53	1.09
Li 2020	[39, 49]	SARS-CoV-2	Serial	Gamma	7.5	3.4	2.34
Li 2023	[32, 50]	SARS-CoV-2 Omicron BA.2	Serial	Gamma	2.89	0.95	3.35
Li, D. 2022	[32, 51]	SARS-CoV-2 Delta	Serial	N/A	2.6	2.0	1.31
Li, L. 2022	[32, 52]	SARS-CoV-2 Delta	Serial	Normal	4.24	3.95	1.07
Linton 2022	[53]	SARS-CoV-2	Generation	Weibull	4.38	2.10	2.20
Lippiello 2023	[54]	SARS-CoV-2 Delta	Generation	Gamma	6.2	1.1	6.60
Lippiello 2023	[54]	SARS-CoV-2 Omicron	Generation	Gamma	3.2	0.8	4.54
Liu, C. 2023	[32, 55]	SARS-CoV-2 Delta	Serial	Gamma	4.15	2.0	2.19
Liu, C. 2023	[32, 55]	SARS-CoV-2 Delta	Serial	Weibull ^{a,c}	4.37	1.87	2.51
Liu, C. 2023	[32, 55]	SARS-CoV-2 Omicron BA.1	Serial	Gamma	3.84	2.3	1.72
Liu, C. 2023	[32, 55]	SARS-CoV-2 Omicron BA.1	Serial	Weibull ^{a,c}	3.85	2.15	1.86
Liu, C. 2023	[32, 55]	SARS-CoV-2 Omicron BA.2	Serial	Gamma	2.77	1.6	1.79
Liu, C. 2023	[32, 55]	SARS-CoV-2 Omicron BA.2	Serial	Weibull ^{a,c}	2.76	1.35	2.15
Liu, T. 2023	[32, 56]	SARS-CoV-2 Omicron BA.5	Serial	Gamma	2.13	1.69	1.27
Luo 2023	[32, 57]	SARS-CoV-2 Delta	Serial	Weibull ^a (shift 6)	4.3	3.6	3.12
Ma 2024	[58]	SARS-CoV-2 Delta	Serial	Weibull	4.79	3.47	1.40
Ma 2024	[58]	SARS-CoV-2 Omicron BA.2	Serial	Gamma	2.70	1.29	2.21
Ma 2024	[58]	SARS-CoV-2 Omicron BA.5.2	Serial	Log-normal	2.12	1.28	1.71
Ma 2024	[58]	SARS-CoV-2 Omicron BF.7	Serial	Gamma	2.43	0.93	2.83
Ma 2024	[58]	SARS-CoV-2 Wild Type	Serial	Gamma	5.69	2.91	2.05
Mefsin 2022	[32, 59]	SARS-CoV-2 Omicron BA.1	Generation	Weibull	2.36	0.59	4.54
Mefsin 2022	[32, 59]	SARS-CoV-2 Omicron BA.1	Serial	Weibull	3.30	1.96	1.74
Mefsin 2022	[32, 59]	SARS-CoV-2 Omicron BA.2	Serial	Weibull	2.23	1.26	1.83
Mefsin 2022	[32, 59]	SARS-CoV-2 Omicron BA.2	Serial	Gamma	2.72	1.51	1.87
Nishiura 2020	[39, 60]	SARS-CoV-2	Serial	Log-normal	4.7	2.9	1.67
Nishiura 2020	[39, 60]	SARS-CoV-2	Serial	Weibull	4.8	2.3	2.20
Ogata 2022	[32, 61]	SARS-CoV-2 Delta	Serial	Gamma (shift 3)	2.73	2.15	2.89
Park 2023	[32, 62]	SARS-CoV-2 Omicron BA.1	Serial	Gamma	2.6	1.9	1.39
Prete 2021	[32, 63]	SARS-CoV-2 Delta	Serial	Normal	2.97	3.29	0.90
Pung 2021	[32, 64]	SARS-CoV-2 Delta	Serial	Skewed normal	3.3	2.8	1.18
Pung 2021	[32, 64]	SARS-CoV-2 Delta	Serial	Weibull ^b (shift 3)	3.44	2.96	2.31
Pung 2021	[32, 64]	SARS-CoV-2 Wild Type	Serial	Weibull ^b (shift 3)	5.09	4.05	2.10
Ryu 2022	[32, 65]	SARS-CoV-2 Delta	Serial	Normal	3.6	4.9	0.75
Shim 2022	[32, 66]	SARS-CoV-2 Omicron BA.1	Serial	Normal	4.15	2.48	1.73
Somda 2022	[67]	SARS-CoV-2	Serial	Gamma	5.92	5.80	1.02
Song 2022	[32, 68]	SARS-CoV-2 Omicron BA.1	Serial	N/A	2.9	1.6	1.88
Song 2022	[32, 68]	SARS-CoV-2 Omicron BA.1	Serial	Weibull ^b	2.95	1.49	2.08
Sreedevi 2023	[69]	SARS-CoV-2	Generation	N/A	3.9	2.6	1.53
Sreedevi 2023	[69]	SARS-CoV-2	Serial	N/A	3.0	2.5	1.21
Wang 2022	[32, 70]	SARS-CoV-2 Delta	Serial	Weibull	4.79	3.47	1.40
Wang 2023	[32, 71]	SARS-CoV-2 Omicron BA.5	Generation	Gamma	2.8	3.7	0.77
Wei 2023	[32, 72]	SARS-CoV-2 Omicron BA.2	Serial	Weibull	3.9	3.7	1.05
Weil 2022	[32, 73]	SARS-CoV-2 Delta	Serial	Log-normal	4.91	2.48	2.08

^aShape parameter taken from publication.

^bOur Weibull fit to the published raw data.

^cAlternative Weibull fit presented in publication.

Table S2: (Continued from previous page) – Estimates of mean serial intervals or generation times, and their shape parameter taken from the literature.

Reference		Disease	Interval	Fit	Mean	SD	Shape
Weil 2022	[32, 73]	SARS-CoV-2 Delta	Serial	Weibull ^b	5.60	3.01	1.94
Weil 2022	[32, 73]	SARS-CoV-2 Omicron BA.1/BA.2	Serial	Log-normal	2.34	1.86	1.27
Weil 2022	[32, 73]	SARS-CoV-2 Omicron BA.1/BA.2	Serial	Weibull ^b	2.91	1.93	1.54
Yadav 2023	[74]	SARS-CoV-2	Serial	N/A	6.4	2.2	3.19
You 2020	[75]	SARS-CoV-2	Serial	N/A	4.60	5.55	0.83
Zeng 2023	[32, 76]	SARS-CoV-2 Delta	Serial	Gamma	4.31	2.70	1.64
Zeng 2023	[32, 76]	SARS-CoV-2 Omicron BA.1	Serial	Gamma	2.58	1.60	1.66
Zeng 2023	[32, 76]	SARS-CoV-2 Omicron BA.2	Serial	Gamma	2.64	1.19	2.36
Zhang 2020	[39, 77]	SARS-CoV-2	Serial	Gamma	5.1	2.68	1.99
Zhang 2021	[32, 78]	SARS-CoV-2 Delta B.1.617.2	Generation	Gamma	2.9	1.9	1.56
Zhang 2021	[32, 78]	SARS-CoV-2 Delta B.1.617.2	Serial	Gamma	2.3	3.4	0.69
Zhang 2022	[32, 79]	SARS-CoV-2 Delta	Serial	Gamma	4.27	2.65	1.65
Fenner 1988	[10, 80]	Smallpox	Serial	Gamma	22.4	6.1	4.13
Fine 2003	[5, 24]	Smallpox	Serial	Normal	16.7	3.3	5.87
Fine 2003	[5, 24]	Smallpox	Serial	Normal	17.3	1.9	11.01
MSA 1953	[5, 81]	Smallpox	Serial	Normal	18.9	4.0	5.45
Rastegar 2024	[82]	Tuberculosis	Serial	N/A	29.59	20.41	1.47
Hope Simpson 1952	[5, 26]	Varicella	Serial	Normal	14.1	2.4	6.90
Lai 2011	[5, 83]	Varicella	Serial	Normal	14.2	1.3	13.33
Vally 2007	[5, 84]	Varicella	Serial	Normal	13.1	2.2	7.00

^aShape parameter taken from publication.

^bOur Weibull fit to the published raw data.

^cAlternative Weibull fit presented in publication.

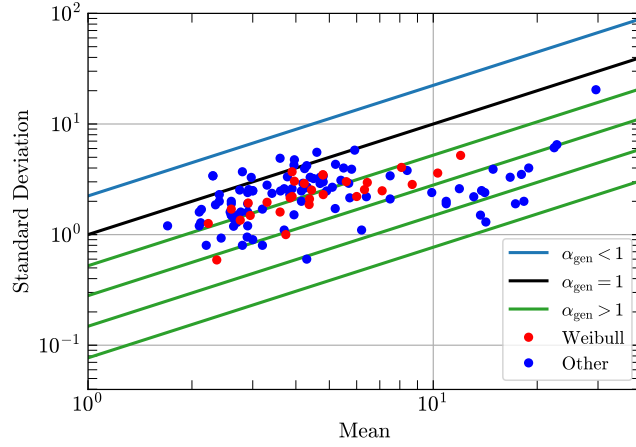


Figure S7: Plot of the mean and standard deviation of generation times and serial intervals from literature. The solid lines show $\alpha_{\text{gen}} \in \{0.5, 1, 2, 4, 8, 16\}$. The red points show publications which used the Weibull distribution as a fit while the blue points show other fits.

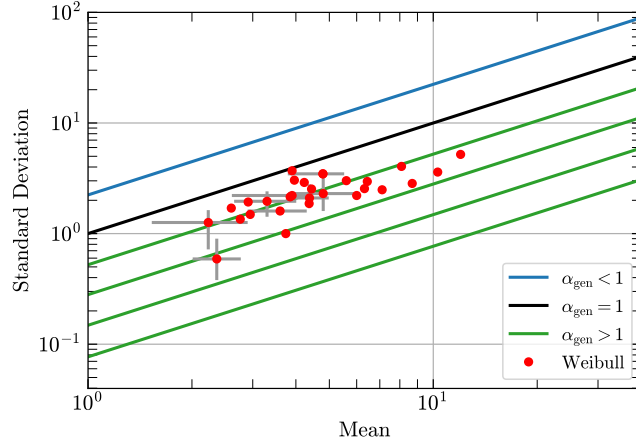


Figure S8: Plot of the mean and standard deviation of generation times and serial intervals from literature, only including publications which used the Weibull distribution as a fit. The solid lines show $\alpha_{\text{gen}} \in \{0.5, 1, 2, 4, 8, 16\}$. Confidence intervals for the mean and standard deviation are plotted if they were reported in the publication.

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