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ORIGINAL ARTICLE

The Effect of Time Distribution Shape on a Complex Epidemic Model

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Abstract In elaborating a model of the progress of an epidemic, it is necessary to make assumptions about the distributions of latency times and infectious times. In many models, the often implicit assumption is that these times are independent and exponentially distributed. We explore the effects of altering the distribution of latency and infectious times in a complex epidemic model with regional divisions connected by a travel intensity matrix. We show a delay in spread with more realistic latency times. More realistic infectiousness times lead to faster epidemics. The effects are similar but accentuated when compared to a purely homogeneous mixing model.

Keywords Prevention and control · Stochastic process · Epidemiology · Infectious time · Latency time · Epidemic model

1. Introduction

In elaborating a model of the progress of an epidemic, assumptions must be made about the distributions of latency times and infectious times. In many models, the often implicit assumption is that these times are independent and exponentially distributed. This distribution is in most cases far from what is known or observed.

There are many reasons for the widespread use of exponential distributions. Regardless of whether a particular model is analyzed with computer simulations, differential equations, or stochastic methods, it often has the advantage of being easy to use. The exponential distribution is inherently *memory-less*. This means that predictions of the future state of the epidemic in terms of number of latent and infectious individuals, etc., is based solely on the current state and not on any prior history. Thus, a model with expo-

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nential times makes it possible to base the analysis of the model on obvious Markovian properties, which will greatly simplify both simulations and analytic derivations.

An equivalent way of stating the assumption of exponential sojourn times is that the hazard of when the latency or infectious period ends is independent of how long it has been since they started, clearly underscoring the unreasonableness of the assumption. Depending on the problem at hand, a model using exponential sojourn times may still provide reasonable descriptions of important aspects of an epidemic. For instance, when analyzing the final size[1] of an epidemic or when comparing mass vaccination policies, the speed at which the infection spreads is of little importance (Svensson, 2007). For such problems, it is sufficient to have reasonable values for the total amount of infectivity spread by an individual, e.g., R_0.

Recently, however, research interest has been directed at questions involving the time aspect of an epidemic outbreak. Models in this area have been prompted by the call for pandemic preparedness. In the event of an outbreak of an emerging infectious disease, the initial, highly stochastic phase of the epidemic is of crucial importance, determining the future course of the epidemic (Anderson and Watson, 1980; Asikainen, 2006; Daley, 1990; Svensson, 2007). Here, differences in assumptions about latency and exponential times play a pivotal role, determining whether or not timely countermeasures will have an effect. After the initial phase, the progress of the epidemic will almost deterministically depend on mass action.

There is another reason why modelers should concentrate their effort on the initial phase of the epidemic. Any countermeasures are likely to be put in effect after the initial phase when the epidemic has reached its full potential in strength. Modelers must assume that their initial assumptions about contact rates and infectiousness are no longer valid after the initial phase.

Thus, it is essential to investigate the effect that different assumptions about latency and infectious times have on how fast an epidemic spreads initially. Even though it has been observed that the exponential time assumption is risky when studying time-related properties of epidemic spread, many recently published authors disregard the consequences of this assumption. One reason for using this assumption may be that it is traditional in the field and, as mentioned above, makes the analysis of the model comparatively simple. We are concerned that the use of exponential sojourn times have persisted into an era when models are becoming increasingly complex.

In this paper, we demonstrate that the choice of time distribution is important when analyzing the speed of the outbreak. This has been shown before by Asikainen (2006), Daley (1990) among others. We opted for using the gamma distribution, which enabled us to retain the Markov approach and gave us a certain freedom to adapt the distribution to fit real data (Malice and Kryscio, 1989). Again, this is nothing new. Gamma distributions are often used in modeling the progress of chronic diseases (e.g., cancer and HIV/AIDS) through different stages. Lloyd demonstrated that realistic sojourn times introduce instabilities in oscillating endemic models (Lloyd, 2007).

To our knowledge, however, studies of the effects of time distributions have only been conducted on simple epidemic models. Here, we investigate the impact using a model with higher complexity where the outcome is not predictable either by common sense

[1] Interestingly, size distribution is dependent on the infectiousness time distribution (Anderson and Watson, 1980).

or any method of analytic derivation that we are aware of. Our results show that time distribution is a matter of careful consideration.

Other time distributions have also been used before, such as uniform, Log-normal, and Weibull (Medley et al., 1987; Lui et al., 1986); the latter two primarily differing in their tails from the gamma distribution. The choice is rather arbitrary, but a discussion is certainly necessary to justify the choice. Times with single point distributions are sometimes considered a reasonable approximation (Carrat et al., 2006; Kaplan et al., 2003), but offer no real advantages in terms of simplification as far as we can see.

2. Travel

Much work has been done showing the effect of travel and migration on the evolution of epidemics (Rvachev and Longini, 1985; Hufnagel et al., 2004; Colizza et al., 2006a, 2006b; Cooper et al., 2006). For today's global outbreaks, notably the SARS outbreak of 2001, the need to incorporate what information we have on travel networks in our simulations has become increasingly apparent. Models that take the Markov approach seem well suited for this purpose as was demonstrated by Hufnagel et al. The population is divided into a number of local regions, which can be countries, municipalities, or other geographic or even social groupings. They are interconnected by an infectiousness intensity matrix that describes how infection is transferred between regions. This matrix can, for example, be estimated from travel data.

Hufnagel et al. used the catchment area around each international airport and within these used a SLIR model, where every individual can be in one of the following states: susceptible (S), latent (L), infectious (I), and recovered (R). These local processes were linked together by the international aviation network, which enabled the disease to be transmitted along flight paths.

Camitz and Liljeros (2006) adapted Hufnagel et al. model to a SARS-like outbreak in Sweden. In this model, municipal borders were used to partition the country. Using detailed travel data between municipalities, not just air traffic, a travel intensity matrix was estimated and the geographic spread could be studied as well as the effect of travel restrictions.

The model we use in this paper is the same as in Camitz and Liljeros (2006), adapted for use with gamma distributions.

In more detail, the SLIR-model works as follows. The population of each municipality is assigned to one of four states defined above. A susceptible may become latent with a probability that depends on the number of infectious persons in his/her own municipality as well as in connected municipalities, depending on the intensity of travel between connected municipalities. After being infected, the latent individual moves through stages L and I with times corresponding to known latency and infectious times. The actual time for any given individual will vary randomly about the mean, which is fixed. The crucial point is how these times vary. In Hufnagel et al. (2004) and in Camitz and Liljeros (2006), the times are picked from exponential distributions.

2.1. The exponential and gamma distribution

The dark blue curve in Fig. 1 shows a plot of the probability density function of the exponential distribution as a special case of the gamma distribution; the circumstances of

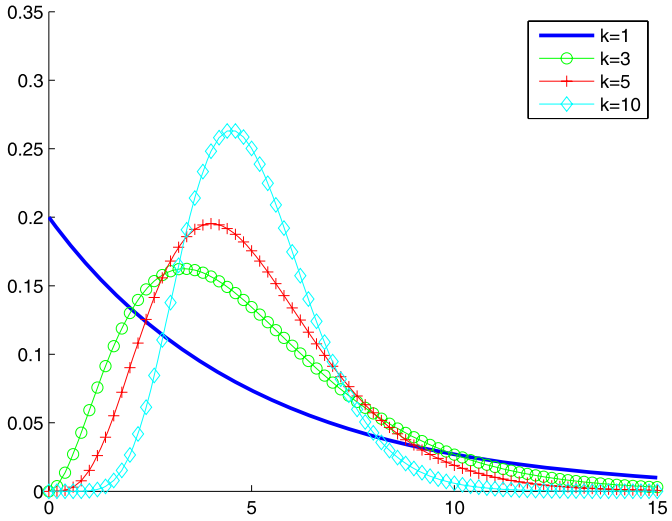

Fig. 1 Probability distribution of the gamma distribution for varying k, all with expectation value 5. The special case of $k = 1$ produces an exponential distribution.

this relationship to be explained later. The exponential distribution has a single parameter equal to the expectation value. It is highly skewed, with high densities for short times and a long tail. Empirical latency times and infectious times are not exponentially distributed, but rather have a more symmetric density about their mean. The exponential distribution has a high variance, equal to its expectation value squared, whereas empirical times tend to deviate little from the mean. In summary, exponentially distributed times is not a reasonable assumption in any case where the variance cannot be disregarded.

Since the median is lower than the expectation value, most times sampled from an exponential distribution will be shorter than the mean. With an expectation value of the latency time of 5 days, 18% of the sampled times will be shorter than 1 day. Such short latency times can safely be considered unrealistic.

In stochastic epidemic simulations, the outcome is highly dependent on the very early initial stages of the outbreak. Individuals with short latency times will predominantly make up the initially infected and will inevitably speed up the outbreak. The skewness of the distribution is important in the early stages of the simulation whereas the expectation value is not apparent until the stochasticity has averaged out.

A few authors have proposed that the gamma distribution be used instead (Anderson and Watson, 1980; Bailey and Estreicher, 1987; Longini et al., 1989). Using this or another less skewed distribution will prevent the predominance of short times of the exponential distribution. The effect will be fewer people in the infectious and latent stages in the initial phase of the epidemic, in turn affecting results where the initial phase is of consequence.

The gamma distribution, denoted $\Gamma(\kappa, \theta)$ has two parameters, a shape parameter κ and a scale parameter θ. For an integer κ, the probability density function takes on a

particularly simple form:

$$f(t;\kappa,\theta) = t^{\kappa-1}\frac{e^{-t/\theta}}{\theta^{\kappa}(\kappa-1)!}. \quad (1)$$

The mean is $\kappa\theta$ and variance $\kappa\theta^2$. For $\kappa = 1$, the gamma distribution is, in fact, identical to the exponential distribution. Keeping the expectation value constant, with increasing κ, the gamma distribution becomes increasingly symmetric. The skewness of the density function is, in fact, $2/\sqrt{\kappa}$. For a suitable choice of κ, the density function can be made to resemble latency/infectious time distributions of empirical studies.

The gamma distribution can actually be realized with an uncomplicated extension of a Markov model, as the sum of several exponentially distributed times is in fact gamma-distributed. This is expressed as follows. Let $X_1, \ldots, X_n$ be independent stochastic variables from an exponential distribution $\mathrm{Exp}(\xi)$. Then $Y = \sum_{i=1}^{n} X_i$ belongs to $\Gamma(n, \xi)$. This is what we will do here, extending the model of Camitz and Liljeros (2006).

In practice, instead of having only a single latency stage and a single infectious stage, we add stages, forcing each individual to go through several stages of latency before becoming infectious, and in the same manner, several stages of infectiousness before recovering. In doing so, we alter κ, which is equivalent to the number of stages, adjusting the second parameter θ so as to keep constant the expected time $\kappa\theta$. The added stages have no epidemiological significance, but serve only to change the appearance of the time distribution. We can achieve an arbitrarily symmetric time distribution with a minimal alteration to our SLIR-model.

The results show that ignoring the shape of the time distribution devalues the results by comparing the results for different κ for both latency times and infectious times. The difference in absolute terms is significant.

3. Data and methods

The intermunicipal infectiousness network is the same as in Camitz and Liljeros (2006). It is based on an interview survey conducted in Sweden between 1999 and 2001 containing some 35,000 trips. This resulted in approximately 12,000 matrix elements (or links) γ_{ij} governing infections caused by traveling (Hufnagel et al., 2004). See the definition in the supplement.

The disease is a fictive, moderately infectious disease with an R_0 of 2.5 within each homogeneous subpopulation. For more information about the specifics of the model, please consult (Camitz and Liljeros, 2006). Details of the extension of the model are straightforward and have been placed in the supplement A for instructive purposes.

To describe the state of the epidemic, we introduce the vector S to keep track of the number of susceptibles in each municipality. Additionally, two sets of vectors $L_1, \ldots, L_\kappa$ and $I_1, \ldots, I_\lambda$ are defined to keep track of latent and infectious persons. The indexes κ and λ are the form parameters of the gamma distribution for latency and infectious times, respectively. The second follows from keeping the mean fixed.

As the initial state, the elements S_i of S are set equal to the population size N_i of each municipality. For each municipality, we now have $1+\kappa+\lambda$ possible state transitions, each one incrementing an element corresponding to the municipality in one vector and decrementing the preceding. A system of epidemic growth equations has been set up to govern

the transitions between the states and are presented in detail in the supplement A. Latents pass through the κ stages of latency at a rate $\upsilon\kappa$, $1/\upsilon$ being the average latency period. The corresponding infectiousness stage rate is $\beta\lambda$, where β is the average infectiousness period. Together with the contact rate α, the expected number of secondary infected per infectious at the start of the simulation can be formulated as α/β. α appears in the most important equation governing the rate of infection, that is, the number of people per time unit entering the first stage of latency. The notation is also described in the supplement 7.

$$Q_i^{\mathcal{L}_1} = \left[\alpha \sum_{k=1}^{\kappa} I_{ki} + \sum_{\substack{j=1 \\ j\neq i}}^{M} \gamma_{ij} \sum_{k=1}^{\lambda} I_{kj}\right] \frac{S_i}{N_i}. \tag{2}$$

4. Results

We carried out two sets of four simulations, each consisting of 1,000 realizations of an outbreak initiated with one infected individual in Stockholm. We placed a time limit of 60 days on each realization. In the first set, we confined the population to a single artificial municipality with the population set to that of the whole country, 9 million. The growth equations then reduce to a normal homogeneous mixing model and the results can be compared to similar models (Anderson and Watson, 1980).

As the travel term in these simulations disappeared, we needed to compensate by increasing the contact rate in the ordinary mixing term. One way to do this is simply to match the cumulative incidence after 60 days. This is crude but produces values which can be compared to the full scale simulation. We matched the case when both latency and infectious times are exponentially distributed, and the contact rate was kept constant for all κ and λ.

In the second set, we used the full travel network for a full scale simulation. In each set, we ran a reference simulation with both the latency and infectious times distributed according to an exponential distribution with the mean being 5 days. Except for different parameters, this setup corresponds exactly to the one used in Camitz and Liljeros (2006). The other three had gamma-distributed latency times, infectious times or both. In the case of gamma distributions, $\kappa = 3$ was used. θ was adjusted to obtain an expected time of, again, 5 days.

We chose to cease simulation after 60 days. By this time, only an insignificant minority of simulated scenarios will not have either developed into epidemics or become extinct. The object of interest in not the final size but the delay in time of the outbreaks. It is also important to note that none of the runs are in the declining phase at this point, either in individual municipalities or counting the whole country. Different latency time distributions do not necessarily affect the height of the incidence peak, only the time it occurs.

The prevalence and cumulative incidence along with some additional results from the first set of four simulations confined to a single municipality is presented in Table 1. It is clear that the shape of the time-distribution determines the outcome of the simulated epidemic. The prevalence after 60 days follows the anticipated pattern, decreasing with more realistic latency times and increasing with more realistic infectious times.

The results of the second set of simulations is presented in Fig. 2 and Table 2. Figure 2 is a geographic plot overlaid on Sweden, with each municipality represented by a colored dot. The prevalence is represented by color on a logarithmic scale. With these plots

Table 1 Results from 200 × 4 runs of an epidemic without municipal borders. In this case, the dispersion equations (2) reduce to a simple homogeneous mixing model. The simulations were aborted after 60 days. For these runs, we increased the contact rate in order to match the cumulative incidence at 60 days of the base case of the full simulation. Extinction runs are defined as runs in which the epidemic dies out before the 60-day limit is reached. With rare exceptions, this occurs within a few days. All means exclude extinction runs. The delay is calculated by recording the time of the 500th infected person. The value given is the mean time relative to the reference run, lower left. All nonextinction runs reach a cumulative incidence of 500, which is why this value was chosen. However, the results are quite stable for any choice of limit. The prevalence is defined as the current number of infectious, that is, latents are excluded from this count. The figures should follow the predicted behavior for homogeneous mixing models, and indeed the number of extinction runs is compatible with predictions made in Anderson and Watson (1980)

	λ (latency)	κ (infectiousness)	
		1	3
Cumulative incidence	3	671,930	119,590
	1	207,290	38,240
Prevalence	3	187,000	27,790
	1	49,320	7,860
Delay (days)	3	−3.3	2.1
	1	0	7.3
Mean time for extinction (days)	3	5.8	3.6
	1	3.7	3.4
Extinction runs (%)	3	12	23.5
	1	26.5	28

Table 2 The results for the full simulations over all municipalities, after a 60-day simulation. The infections attributed to traveling were estimated by adding up the probability for each new infected person being infected from outside another municipality using (2). Please see the legend of Table 1 for a description of the values. The behavior exhibited in the single municipality simulations is even more apparent here, which means that retransmission from connected municipalities amplifies the distribution effects. We added the cumulative incidence from these runs in Stockholm only for comparison

	λ (latency)	κ (infectiousness)	
		1	3
Cumulative incidence	3	718,830	140,530
	1	184,240	44,810
Prevalence	3	212,600	35,260
	1	46,340	9,828
Delay (days)	3	−5.1	1.6
	1	0	6.4
Cumulative incidence in Stockholm	3	160,310	35,770
	1	44,030	11,760
Mean number of afflicted municipalities	3	279.3	250.7
	1	249.0	190.6
Extinction runs (%)	3	9.5	9.9
	1	24.1	29.5
Mean time for extinction (days)	3	4.4	3.9
	1	3.5	3.3
Infections attributed to traveling (%)	3	71	72
	1	33	33

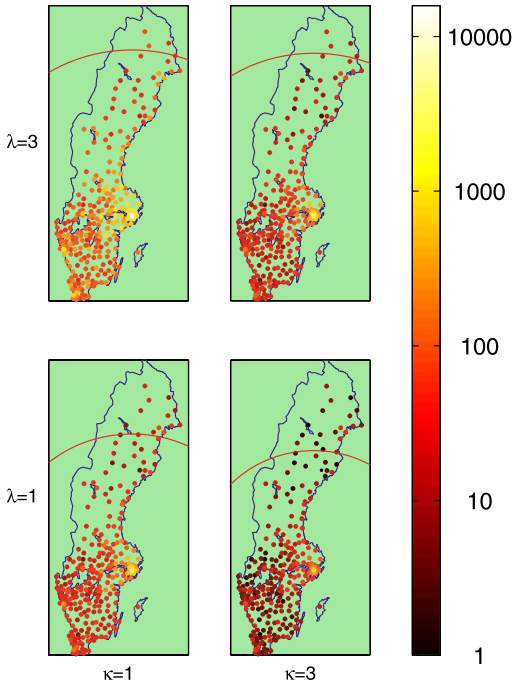

Fig. 2 Image visualizing the epidemic state after a 60-day simulation, averaged over 1,000 runs. The form parameter for latency times increases from the left to right column and for infectious times from the bottom to top row. The prevalence after 60 days of simulation in each municipality is color-coded on a logarithmic scale. The arcs represent the attained spreading distance after 60 days of the epidemic as measured from Stockholm, averaged over all runs. Clearly, a more realistic latency time distribution delays the epidemic significantly.

complemented by the information in the Table 2 it is possible to compare the different scenarios. Again, the prevalence, cumulative incidence, and geographic spread are highly dependent on the shape of the time-distribution. As with the first set, the order of severity after 60 days is as anticipated but the effects are even more apparent. We observe from the plots that no unexpected effects from the travel model appear to complicate the results. However, retransmission from connected municipalities appears to amplify the effect of the distribution shape from the perspective of the whole country. The same pattern is also seen when looking at just the cumulative incidence from these runs in Stockholm. We also added a figure (Fig. 3 for additional support with κ simultaneously varied from 1 to 20).

5. Discussion

Considering first the simple case of a large single municipality, the extremely short latency times generated by the exponential distribution are expected to accelerate the epidemic. More individuals become infectious early in the simulation, in turn, infecting others earlier. It can be shown, however, that the final size of the epidemic is unchanged by the shorter mean latency time (Anderson and Watson, 1980).

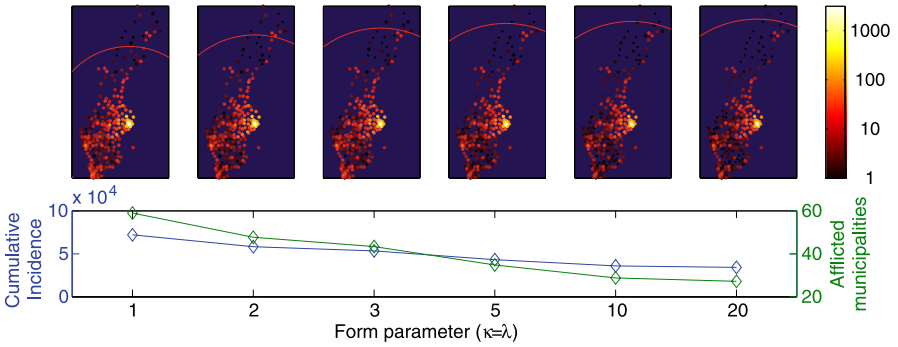

Fig. 3 Here, the form parameter for both latent and infectious time distributions are set equal. Cumulative incidence, that is, the total number of infected after 60 days is plotted below for each setting, as is the number of municipalities touched by the epidemic at any time.

Applying a skewed distribution to the infectious time reverses the effect. The outbreak will instead be delayed, due to the abundance of very short infectious times. Each infected person will contact and, therefore, infect a fewer number of people before recovering. It is harder for the disease to become epidemic, and the probability of the disease dying out completely is higher. As long as the epidemic is in the growth phase, there will be fewer people in the infectious phase with a highly skewed distribution than with one less skewed.

Since we are dealing with stochastic simulations, the events are random. The crucial period is the initial phase of the simulated epidemic, which is decisive for the future evolution of the epidemic, both in terms of speed and proportions. As there are very few infectious persons, the initial phase of the outbreak proceeds in a highly random fashion. After the initial phase, the process smoothes out and becomes more predictable (Colizza et al., 2006b), determined by the mass action principle. When considering the effects of altering the time distributions, it is important to consider effects which occur during the initial phase but are evened out as more people become infected.

In a complex meta-population model, the dynamics are less predictable and logical deduction may not offer enlightenment. Intuition tells us that the combined effect of two contributions is more than the sum. We may therefore expect a high incidence when the infectious period is prolonged due to the combined contribution of a larger number of infectious persons and the amount of traveling they do during their infectious period. As it turns out, the results of our simulations support those assumptions. The effects of distributions apparent in the simple one-municipality test are accentuated in the multimunicipal test.

We should mention that the gamma distribution is not the only choice for modelers. Many alternatives have been proposed and used in models, such as the log-normal distribution and Weibull distributions (Medley et al., 1987; Lui et al., 1986). All three have visually similar probability density plots, but differ in key points, particularly in regard to their tails. As we have illustrated, the tails of the assumed distribution are important for the outcome of the simulations. These differences have to our knowledge not been explored in epidemic models. Our particular choice of the gamma distribution over other possibilities is as much a consequence of design as deliberate choice, as may be the case

for the exponential distribution in other models. Possible benefits of alternative choices of distributions will be for future experiments to show.

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Appendix A: Model details

To describe the state of the epidemic, we introduce the vector S to keep track of the number of susceptibles in each municipality. Additionally, two sets of vectors $L_1, \ldots, L_\kappa$ and $I_1, \ldots, I_\lambda$ are defined to keep track of latent and infectious persons. The indexes κ and λ are the form parameters of the gamma distribution for latency and infectious times, respectively. We will use a general formalism for the time being, but later we set the parameters to either 1 or 3. In the first case, corresponding to an exponential distribution, there will only be one vector in the set. If κ is greater than 1, then this will be the number of stages of latency or infectiousness that each individual needs to pass through. The size of each vector is equal to the number of municipalities. Let P be this number. The dimensionality of the entire state space is equal to $D = P \cdot (1 + \kappa + \lambda)$. The vectors are indexed as $I_{k,i}$ where i is the municipality and k is the stage of disease. Summing over all k and i yields in this case the total number of infected. Note that recording the number of recovered individuals is redundant since it is simply the sum of the number in the three states already covered, subtracted from the population.

At the start of the run the element S_i of S is equal to the population size N_i of each municipality. This is the initial state in each run. For each municipality, we now have $1 + \kappa + \lambda$ possible state transitions, each incrementing an element corresponding to the municipality in one vector and decrementing the "preceding". This is true for all transitions except from the last stage of infectiousness, which only involves a decrement.

We are now ready to set up the equations that will define the transition matrix of our Markov process. The quantities $Q_i^{\mathcal{X}}$ below, are for each municipality i the *intensity* of individuals passing on to the next stage of illness and are connected to the probabilities of the corresponding state transitions. $\mathcal{X} \in \{\mathcal{L}_1, \ldots, \mathcal{L}_\kappa, \mathcal{I}_1, \ldots, \mathcal{I}_\lambda, \mathcal{R}\}$ is a *label* signifying transitions to one of the latency states, one of the infectious states or the recovered state. It is written in a calligraphic font to avoid confusion with L_k, I_k, and R which are vectors.

$$
\begin{aligned}
Q_i^{\mathcal{L}_2} &= \upsilon\kappa L_{1i}, \\
&\vdots \\
Q_i^{\mathcal{L}_\kappa} &= \upsilon\kappa L_{\kappa-1,i}, \\
Q_i^{\mathcal{I}_1} &= \upsilon\kappa L_{\kappa i},
\end{aligned}
$$

$$
\begin{aligned}
Q_i^{\mathcal{I}_2} &= \beta\lambda I_{1i}, \\
&\vdots \\
Q_i^{\mathcal{I}_\lambda} &= \beta\lambda I_{\lambda-1,i}, \\
Q_i^{\mathcal{R}} &= \beta\lambda I_{\lambda i}.
\end{aligned}
\tag{A.1}
$$

Finally, people are infected (become latent) with an intensity that depends on the number of infected in all the municipalities and the travel intensity between each of them:

$$
Q_i^{\mathcal{L}_1} = \left[\alpha \sum_{k=1}^{\kappa} I_{ki} + \sum_{\substack{j=1 \\ j\neq i}}^{M} \gamma_{ij} \sum_{k=1}^{\lambda} I_{kj}\right] \frac{S_i}{N_i}. \tag{A.2}
$$

In the equations above, α/β is the expected number of secondary infected per infectious person at the start of the simulation. υ is the inverse latency period. The first row for instance reads: The number of people per unit time leaving the first latency stage is the number of people in that stage times the number of stages times the scalar rate υ. The last row is similar, as is the first term of the first row, but summed over all infectious stages and also includes a factor to account for a decreasing number of susceptibles. The second term is the contribution from other municipalities via the infectiousness network. It includes a sum of infectious individuals over all stages and all municipalities but the current one.

The component γ_{ij} is estimated with

$$
\gamma_{ij} = \gamma M_{ij} / \sum_j M_{ij}, \tag{A.3}
$$

where M_{ij} is the number of journeys per day from municipality i to j, and γ is a global scalar governing infections caused by traveling (Hufnagel et al., 2004). γ_{ij}/γ is the probability that a traveler in i will choose the route ij.

Each intensity in Eq. (A.1) is the parameter required to specify the exponential distribution that yields the time steps for the corresponding transition. The model is now in all respects in place. To simulate, we need to take each transition in order and so we are interested in knowing the time Δt until the next transition, given the current state. The time, one can easily show, is incidentally also exponentially distributed with parameter Q equal to the sum of the D intensities in Eqs. (A.1) and (A.2),

$$
\Delta t \in \mathrm{Exp}(Q), \tag{A.4}
$$

$$
Q = \sum_{i=1}^{M} \left(Q_i^{\mathcal{L}_1} + \cdots + Q_i^{\mathcal{L}_\kappa} + Q_i^{\mathcal{I}_k} + \cdots + Q_i^{\mathcal{I}_\lambda} + Q_i^{\mathcal{R}}\right). \tag{A.5}
$$

To determine which transition occurs at this time, we compare the intensities among themselves. The probability of a transition is proportional to the relative value of the corresponding intensity, simply the intensity normalized by Q. In each pass through the main loop of the algorithm, we find Q, pick a random time step from the exponential distribution specified by Q as a parameter, randomly pick a transition according to the relative

value of the intensities, and update the state vectors and the intensities according to the new state and start again. The simulation proceeds this way until there are either no more infectious or latent persons or until an arbitrarily chosen time limit is reached, whichever comes first. In our case, we chose 60 days as by this time a substantial majority of simulated scenarios will have developed into epidemics. Recall that the object of interest in not the final size, but any delay in time of the epidemics.

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