

General epidemiological models: law of large numbers and contact tracing

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Abstract

We study a class of individual-based, fixed-population size epidemic models under general assumptions, e.g., heterogeneous contact rates encapsulating changes in behavior and/or enforcement of control measures. We show that the large-population dynamics are deterministic and relate to the Kermack–McKendrick PDE. Our assumptions are minimalistic in the sense that the only important requirement is that the basic reproduction number of the epidemic R_0 be finite, and allow us to tackle both Markovian and non-Markovian dynamics. The novelty of our approach is to study the “infection graph” of the population. We show local convergence of this random graph to a Poisson (Galton–Watson) marked tree, recovering Markovian backward-in-time dynamics in the limit as we trace back the transmission chain leading to a focal infection. This effectively models the process of contact tracing in a large population. It is expressed in terms of the Doob h -transform of a certain renewal process encoding the time of infection along the chain. Our results provide a mathematical formulation relating a fundamental epidemiological quantity, the generation time distribution, to the successive time of infections along this transmission chain.

Keywords: Crump–Mode–Jagers process with interaction; functional law of large numbers; Kermack–McKendrick model; non-Markovian epidemic model; contact-tracing; local weak convergence; infection graph.

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1 Introduction

1.1 General individual-based epidemic model

In the present article, we study an extension of the general epidemiological framework introduced in [18] to model the COVID-19 epidemic. Let us briefly recall the main features of this model.

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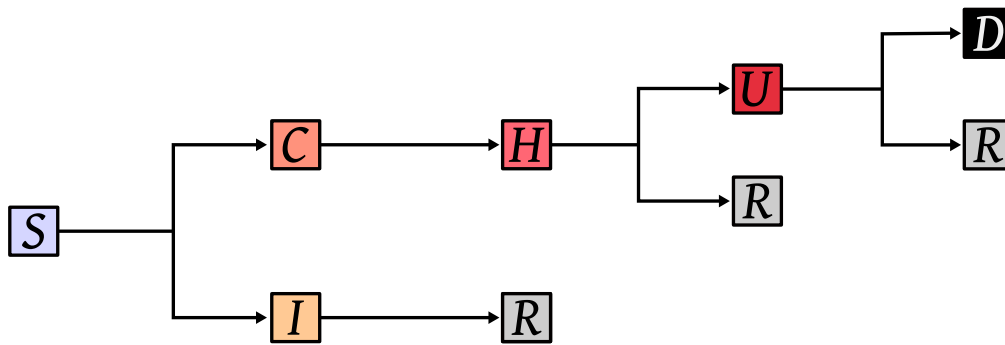


Figure 1: An example of compartmental model for the COVID-19 epidemic. The compartments are: S , susceptible; I , mildly infected; C , severely infected; H , hospitalized; U , admitted to intensive care unit; R , recovered; and D , dead.

At time $t = 0$, we consider a population made of susceptible individuals, that have never encountered the disease, and infected individuals. Each infected individual is supposed to belong to one *compartment*, that models the stage of the disease of this individual. Classical examples of compartments are the exposed compartment (E), where the individual is infected but not yet infectious, the infectious compartment (I), and the recovered compartment (R), once the individual has become immunized. In the case of the COVID-19 epidemic, it might be relevant to add a hospitalized (H) and an intensive care unit (U) compartment, as monitoring the number of individuals in these states is typically important for policy-making. See Figure 1 for an example of compartmental model used for the COVID-19 epidemic. We denote by S the set of all compartments. For the sake of simplicity, we will also assume that S is finite.

We encode the compartment to which individual x belongs as a stochastic process $(X_x(a); a \geq 0)$ valued in S , that we call the *life-cycle process*. The random variable $X_x(a)$ gives the compartment to which x belongs at *age of infection* a , that is, a unit of time after its infection. Moreover, individual x is endowed with a point measure \mathcal{P}_x on \mathbb{R}_+ that we call the *infection point process*, which is assumed to be simple. The atoms of \mathcal{P}_x encode the age at which x makes infectious contacts with the rest of the population. We think of the pair (\mathcal{P}_x, X_x) as describing the course of the infection of individual x . We make the fundamental assumption that the pairs (\mathcal{P}_x, X_x) are i.i.d. for distinct individuals in the population.

In [18] we assumed that susceptibles are in excess, and that any infectious contact leads to a new infection. The resulting population is then distributed as a Crump–Mode–Jagers (CMJ) process. In the current work, we consider an extension of this model that takes into account the saturation due to the finite pool of susceptibles in the population. More precisely, we consider a population of finite fixed size N . Each infectious contact is made with an individual uniformly chosen in this population, and it results in a new infection only if the targeted individual is susceptible. Finally, we model the impact of control measures, such as school closure, or national lockdown, with a *contact rate function* $(c(t); t \geq 0)$. This contact rate is such that an infection occurring at time t is only effective with probability $c(t) \in [0, 1]$. With probability $1 - c(t)$, the infection is removed. A formal description of this model is provided in Section 2.

1.2 Law of large numbers for the age structure

A standard way to study compartmental models is to consider the dynamics of the number of individuals in each compartment. If the underlying probabilistic model is

Markovian, this typically gives rise to systems of ODEs of the SIR type in the large population size limit, see [10] for a recent account. Here, we will not keep track of the count of individuals in the various compartments, but we will rather be interested in the age structure of the population. Our main result is a law of large number for the age structure of population, which is the equivalent of Theorem 7 of [18] for our non-linear extension of the model.

We anticipate the notation of Section 2 and denote the empirical measure of ages and compartments in the population at time t as

$$\forall i \in \mathcal{S}, \quad \mu_t^N(\mathrm{d}a, \{i\}) = \sum_{x=1}^N \mathbb{1}_{\{\sigma_x^N \leq t\}} \mathbb{1}_{\{X_x(t-\sigma_x^N)=i\}} \delta_{t-\sigma_x^N}(\mathrm{d}a),$$

where σ_x^N is the infection time of individual x , and the sum runs over all infected individuals at time t . (Note that $t - \sigma_x^N$ is just the age of x at time t .) The measure μ_t^N encodes the ages and compartments of infected individuals at time t . Let us also denote by

$$\forall t \geq 0, \forall i \in \mathcal{S}, \quad Y_t^N(i) = \#\{\text{individuals in } i \text{ at time } t\} = \mu_t^N([0, \infty), \{i\}).$$

The limiting distribution of μ_t^N will depend on the following two quantities:

- The intensity measure of the infection point process defined as

$$\tau(\mathrm{d}a) := \mathbb{E}[\mathcal{P}(\mathrm{d}a)]. \tag{1.1}$$

We assume that τ has a density w.r.t. the Lebesgue measure that we denote by $\tau(a)$ with a slight abuse of notation, and that $R_0 := \tau([0, \infty)) < \infty$. We also assume that there exists $\alpha \in \mathbb{R}$, called the *Malthusian parameter* such that

$$\int_0^\infty e^{-\alpha a} \tau(a) \mathrm{d}a = 1. \tag{1.2}$$

- The one-dimensional marginals of the life-cycle process, denoted by

$$\forall i \in \mathcal{S}, \forall a \geq 0, \quad p(a, i) := \mathbb{P}(X(a) = i).$$

Let $I_0 \in (0, 1)$. At time $t = 0$, we assume that every individual is independently infected with probability I_0 and that its age of infection is chosen independently according to a probability density g on \mathbb{R}_+ . In the following, we define $\mathcal{I}_0^N \subseteq [N]$ as the set of infected individuals at $t = 0$. We will also use the notation $S_0 = 1 - I_0$.

We make the simplifying assumption that the underlying compartmental model is acyclic: we assume that for any two compartments $i, j \in \mathcal{S}$, if j can be accessed from i with positive probability, that is, if the event that we can find $s \leq t$ such that $X(s) = i$ and $X(t) = j$ has positive probability, then i cannot be accessed from j . In other words, the directed graph on \mathcal{S} composed of all edges $i \rightarrow j$ such that j is accessible from i is a directed acyclic graph. This assumption is not very restrictive, most natural compartmental models enjoy this acyclic property. See Figure 1. It is only needed to reinforce the finite-dimensional convergence to a Skorohod one in the following theorem.

Theorem 1.1. *As $N \rightarrow \infty$, the following convergence holds in probability in the Skorohod topology on the space of measure-valued processes,*

$$\left(\frac{1}{N} \mu_t^N(\mathrm{d}a, \{i\}); t \geq 0 \right) \longrightarrow (n(t, a)p(a, i) \mathrm{d}a; t \geq 0)$$

where $(n(t, a); t, a \geq 0)$ is the solution to

$$\begin{aligned} \partial_t n(t, a) + \partial_a n(t, a) &= 0 \\ \forall t \geq 0, n(t, 0) &= c(t)S(t) \int_0^\infty n(t, a) \tau(da) \\ \forall a \geq 0, n(0, a) &= I_0 g(a) \\ \forall t \geq 0, S(t) &= 1 - \int_0^\infty n(t, a) da. \end{aligned} \tag{1.3}$$

This theorem is proved at the end of Section 7, as a consequence of the convergence of the so-called historical process (Theorem 7.1). From Theorem 1.1 we immediately deduce the following result.

Corollary 1.2. For any $i \in \mathcal{S}$, we have

$$\left(\frac{1}{N} Y_t^N(i); t \geq 0 \right) \longrightarrow \left(\int_0^\infty n(t, a) p(a, i) da; t \geq 0 \right) \tag{1.4}$$

in probability in the Skorohod topology.

Remark 1.3. Theorem 1.1 can be easily extended to weaker assumptions on the initial condition. For instance, it is not hard to see from our proof that Theorem 1.1 holds true if we simply assume that

$$\frac{1}{N} \sum_{x=1}^N \mathbb{1}_{\{\sigma_x^N < 0\}} \delta_{-\sigma_x^N}(da) \longrightarrow I_0 g(a) da$$

where the convergence holds in probability for the weak topology.

Remark 1.4 (Infinite reproduction number). The dynamics of the epidemic until some fixed time t does not depend on the potential infections occurring after time t . In particular we can remove all atoms after age t of the point processes of the individuals that are initially susceptible without affecting the process at time t . Similarly, we can remove all atoms after time t of the point processes of the initially infected individuals. Anticipating the notation $\bar{\tau}$ in (1.8) for the intensity measure of the initially infected individuals, the convergence of the process in Theorem 1.1 until time t only requires that $\int_0^t \tau(a) da < \infty$ and $\int_0^t \bar{\tau}(a) da < \infty$. Thus, Theorem 1.1 holds under the weaker assumption that τ is a locally finite measure, and that g decays fast enough so that $\bar{\tau}$ is also locally finite. In particular one could have that $R_0 = \infty$.

This result will follow from the more general Theorem 7.1, and is proved in Section 7. The definition of a solution to (1.3) is provided in Section 3. Theorem 1.1 proves that the age structure of the population converges to a limiting non-linear PDE of the Kermack-McKendrick type [30]. It also entails that the number of individuals in each compartment can be recovered by integrating the one-dimensional marginals $p(a, i)$ against the age structure.

It is interesting to note that the limit of our model is universal. The limiting expression in Equation (1.4) does not depend on the entire distribution of the pair $(\mathcal{P}, (X(a); a \geq 0))$, but only on:

- the mean number of secondary infections $\tau(a)$ induced by an infected individual with age a ;
- the one-dimensional marginals $p(a, i)$ of the life-cycle process.

These are the only individual characteristics that need to be assessed to forecast the dynamics of the epidemic at a large scale. By further writing $\tau = R_0 \nu$ with

$$R_0 = \int_0^\infty \tau(da), \quad \nu(da) = \frac{\tau(da)}{R_0}, \tag{1.5}$$

we see that τ only depends on two well-known epidemiological quantities:

- the basic reproduction number, R_0 , which is the mean number of secondary infections induced by a single individual in an entirely susceptible population;
- the distribution of the generation time, ν , which gives the typical time between the infection of a source individual and that of the recipient individual in an infection pair [19].

Further interesting modeling consequences of Theorem 1.1 are discussed in our earlier work [18].

1.3 Contact tracing: the historical process

We already argued that our approach allows to identify the individual characteristics that impact the large population size dynamics. We identified those parameters as R_0 , the distribution of generation time ν together with the one-dimensional marginals of the life-cycle process. The estimation of those parameters is obviously of paramount importance. One possible approach to estimate the generation time distribution consists in observing the generation times backwards in time using contact tracing [15, 20], i.e., the time between the infection time of an individual (the infectee) and that of his/her infector (rather than the infection time of the individuals he/she infects). In [11], the authors addressed this specific question in a simplified setting. More specifically, they assumed that $c \equiv 1$ and that the susceptibles are in excess so that our microscopic model can be approximated by a Crump–Mode–Jagers process as in our earlier work [18]. They showed that the observation of backward generation times raises two serious issues:

- (i) First, observations of past infections induce a strong observational bias: the backward generation time distribution differs from the actual generation time distribution. In the supercritical case (i.e., when $R_0 > 1$), the backward generation time has density

$$\exp(-\alpha u) R_0 \nu(u) \tag{1.6}$$

where $\alpha > 0$ is the Malthusian parameter of the model defined in (1.2). As a consequence, observations of backward infection times tend to be biased towards lower values.

- (ii) Infection times are difficult to observe. Instead, the onset of symptoms is generally observed. For this reason, the serial interval, which is defined as the time between symptom onsets in the two individuals mentioned above, is often used as a surrogate for the generation time. As discussed in [11], this can induce a second source of significant observational bias.

As already mentioned above, the authors in [11] address the previous bias in the case where $c \equiv 1$ and when susceptibles are in excess. In the present article, we will show if we (1) take into account saturation effect (i.e, when the population is out of the branching process regime), and (2) assume some heterogeneity in the contact rate, then those two components of the dynamics can induce a third source of bias.

In order to provide some intuition of the upcoming results, consider a newly infected individual at time t . Trace backward in time the chain of infection up to time $t = 0$. (The first individual along the chain is the infector of the focal individual, the second is the infector of the primary infector and so on.) Finally, along the chain, report the successive times of infection, see Figure 2. When susceptibles are in excess (branching approximation) Jagers and Nerman [32] showed under mild assumptions that as $t \rightarrow \infty$, the successive time of infections are well approximated by the values of a renewal

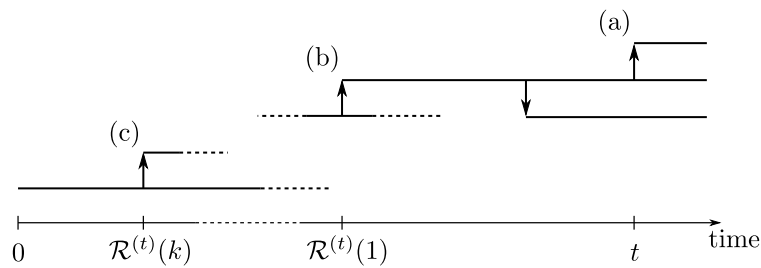


Figure 2: Chain of infection of a focal individual. Horizontal lines represent the lifetime of an infected individual, and vertical arrows represent new infections. Going backwards in time, the following events are recorded: (a) the focal individual i_0 is infected at time t ; (b) its infector, individual i_1 , is infected at time $\mathcal{R}^{(t)}(1)$; (c) going back a chain of k successive infections, individual i_k is infected at time $\mathcal{R}^{(t)}(k)$ by an individual that was already infected at time 0.

process

$$\mathcal{R}^{(t)}(0) = t, \quad \forall k \geq 1, \quad \mathcal{R}^{(t)}(k) = t - \sum_{i=1}^k \xi_i$$

where the ξ_i 's are i.i.d. and distributed according to (1.6).

In the presence of saturation, we show that the chain of infection is given by an h -transform of the renewal process $\mathcal{R}^{(t)}$. Intuitively, the h -transform tends to favor infection at times where there is a large fraction of susceptibles and a high contact rate. When the initial age structure of the population coincides with the “equilibrium” measure of the branching approximation, i.e.,

$$g(u) = \alpha \exp(-\alpha u),$$

the h -transformed process can be reformulated in a simple manner. In Proposition 5.9 we show that it is identical in law to the original renewal process conditioned on survival assuming that at each step k the process is killed with probability $1 - c(\mathcal{R}^{(t)}(k))S(\mathcal{R}^{(t)}(k))$.

In Section 7, we introduce the historical process. Loosely speaking, the historical process is the empirical measure reporting the chain of infections for every individual infected at time $t = 0$ or who eventually gets infected in the future. It is constructed by reporting the successive age of infections along the chain, but also the stages of the life-cycle process for every “ancestor” along the chain, e.g. onset of the symptoms, latency period, etc. In Theorem 7.1, we show that the historical process converges to a deterministic probability law. Loosely speaking, our result shows that the chain of infections for a finite sample of infected individuals are asymptotically independent. Furthermore, for each sampled individual, its chain of infection is distributed in such a way that

- the successive times of infection are expressed in terms of the h -transformed renewal process mentioned above.
- the life cycle of individuals along the chain is biased, and the bias can be expressed as a Palm modification of the original life cycle. This will be made more formal in Section 5.

Going back to the two epidemiological questions (i) and (ii), our results decipher how the epidemiological parameters R_0 and the generation time distribution ν relate (in a non-trivial way) to observables which can be directly collected from contact-tracing data.

1.4 A genealogical dual to the delay equation

The Kermack–McKendrick equation (1.3) can be reformulated in terms of a non-linear delay equation. To ease the exposition, let us consider the case $c \equiv 1$. In Section 5, the general case $c \neq 1$ will be exposed.

If $(n(t, a); t, a \geq 0)$ denotes the solution to (1.3) with $c \equiv 1$, let us define the number of infections between time 0 and t as

$$B(t) = \int_0^t n(s, 0) ds = \int_0^t S(s) \int_0^\infty n(s, a) \tau(da) ds.$$

Then we will derive in Section 3 that B solves the following non-linear delay equation:

$$B(t) = S_0 \left(1 - \exp \left(- \int_0^t \tau(a) B(t-a) da - \int_0^\infty \int_0^t \tau(a+s) g(a) ds da \right) \right), \quad (1.7)$$

where we recall that $S_0 = 1 - I_0$ is the initial fraction of susceptibles.

Our proof of Theorem 1.1 uses a genealogical approach, where we look backwards in time at the set of potential infectors of a focal individual. This approach leads to a genealogical dual to the delay equation that we think to be of independent interest. The dual is built out of the following branching process.

Recall that R_0 stands for the total mass of τ and $\nu = \tau/R_0$. We define the intensity

$$\forall u \geq 0, \quad \bar{\tau}(u) = \int_0^\infty g(a) \tau(a+u) da \quad (1.8)$$

so that the measure $\bar{\tau}(u) du$ is the intensity measure of the infection point process of an individual with initial age distributed as g . Let us further set

$$\bar{R}_0 = \int_0^\infty \bar{\tau}(u) du, \quad \bar{\nu}(du) = \frac{\bar{\tau}(u) du}{\bar{R}_0}. \quad (1.9)$$

The branching process is constructed as follows. Let us assume that individuals in the branching process are either infected (I) or susceptibles (S). Suppose that the population starts from a single (S) individual. Then, at each generation, an (S) individual produces:

- a Poisson($S_0 R_0$) distributed number of (S) individuals;
- a Poisson($(1 - S_0) \bar{R}_0$) distributed number of (I) individuals.

Individuals of type (I) have no offspring. Draw an oriented edge from each individual towards its parent. Assign a length independently to each edge, such that the length of an edge originating from an (S) individual is distributed as ν , and that of an edge coming from a (I) individual is distributed as $\bar{\nu}$.

The previous branching process corresponds to the large population size limit of the set of potential infectors of a fixed individual. Type (I) individuals correspond to individuals that were initially infected. Each edge corresponds to an infectious contact in the population, and the length of that edge is the age of the infector when this contact occurs.

The corresponding object is a rooted *geometric tree*, where edges are endowed with a length. We define the infection path at the root as the (a.s. unique) geodesic connecting the set of infectious individuals (I) to the root. Finally, the time of infection σ^∞ is defined as the length of the geodesic. The following result connects the distribution of σ^∞ to the delay equation.

Proposition 1.5. For any $t \geq 0$, define

$$B(t) = S_0 P(\sigma^\infty \leq t).$$

Then $(B(t); t \geq 0)$ solves the delay equation (1.7).

In Section 5, we will derive a similar dual for the delay equation with $c \neq 1$ — see Proposition 5.4.

1.5 Link with literature

Age-structured models in epidemiology. The idea of studying an epidemic through its age structure dates back to at least the work of Kermack and McKendrick [30]. They introduced a general epidemic model where the infectiousness of an individual can depend on its age of infection, which was formulated as the solution to a delay equation equivalent to (1.3). In the same article [30], the authors noticed that if the infectiousness and the recovery rate are assumed to be constant, Equation (1.3) reduces to a set of non-linear ODEs now known as the SIR model. Even if the work of [30] was primarily devoted to the more general age-structured model, subsequent work on epidemic modeling has mostly focused on extensions of the ODE special case. Nonetheless, the original age-structured model is now receiving renewed attention both in the mathematical literature [37, 13, 23] and in applications [12, 46, 8, 9].

In the probabilistic literature, it is only quite recently that it was proved in [5] that Equation (1.3) describes the large population size limit of a stochastic epidemic model similar to ours. The setting of the main result in [5] is slightly different from that considered in the current work: the process is assumed to be supercritical ($R_0 > 1$) and to start from a single infected individual. After an appropriate time-shift so as to skip the long initial branching phase when there are few infected individuals, [5] prove that the fraction of susceptibles in the population converges to a limiting function $(S(t); t \in \mathbb{R})$. This limit corresponds to the number of susceptible individuals in an extension of (1.3) to the whole real line [13, 40, 41]. Although the law of large numbers considered in [5] is quite similar to our Theorem 1.1, let us outline some important differences.

From a purely technical point of view, [5] work under quite restrictive assumptions on the point process \mathcal{P} , see Assumption 2 on the top of page 7. For instance the simple Markovian SEIR model [10] does not fulfill these assumptions. Also, the model in [5] does not explicitly account for compartments through $(X(a); a \geq 0)$, nor for the contact rate $(c(t); t \geq 0)$. These are two key modeling ingredients in the context of the COVID-19 epidemic. While incorporating compartments would be a direct extension of the proofs in [5], taking into account the contact rate would raise the same serious technical difficulties as in our work, since their proofs rely on a backward-in-time approach similar to ours. Finally, the description of the chain of transmission events leading to a focal infection, which is one of the main contributions of our work, is not considered in [5].

Other age-structured models. There exists a rich literature on age-dependent population processes, not necessarily related to epidemic models. Let us first mention the Crump–Mode–Jagers processes (CMJ), also known as general branching processes [25, 39], from which the formalism of our model is borrowed. In these processes, the birth time of children can depend in a very general way on the age of the parent, but individuals reproduce independently of each other. These processes are good approximations of the early dynamics of an epidemic when susceptible individuals are in excess. We have considered such an approximation in an earlier work [18] and proved a law of large numbers similar to Theorem 1.1 in this context.

Further models have relaxed the assumption that individuals reproduce independently by allowing the birth and death rates to depend on the whole age distribution of the

population [43, 26, 27, 22]. Under a large population size limit, the age structure of these models converges to an extension of the McKendrick–von Foerster equation that generalizes (1.3) [43, 22]. Moreover, several central limit theorems have been derived for this age structure [42, 14]. Although these models allow for a very general dependence between births and the state of the population, they require the age distribution to be a Markov process and our results are not trivially implied by those in the above works. The techniques used to study these models are also quite different from the backward-in-time approach developed here. They require to see the age structure as the solution to a stochastic equation driven by a Poisson measure, or to use martingale techniques which could not be extended to our setting.

Other non-Markovian epidemic models. Finally, there is a recent series of work that considers epidemic models that are non-Markovian [34, 36, 16, 33, 35], but not structured by the age of infection. They derived laws of large numbers and central limit theorems for extensions of the model considered in [34] that can incorporate spatial heterogeneity [36], varying or random infectiosity [16, 33, 35], and applied these models to the COVID-19 epidemic in France [17]. The limiting equations that describe the dynamics of the density of individuals in each compartments are systems of so-called Volterra integral equations. These equations are tightly linked to our PDE representation using the Kermack–McKendrick equation (1.3), as is acknowledged explicitly in [35], Proposition 2.1. All the models with non-vanishing immunity that they consider (SIR, SEIR) can be formulated within our framework. The infection point process \mathcal{P} is obtained by starting either a homogeneous Poisson point process [34], an inhomogeneous Poisson point process [16, 33], or an inhomogeneous Poisson point process with random intensity [35], at age $a = 0$ in the SIR case, or after an exposed phase in the SEIR case. Moreover, the proof techniques they use rely heavily on a representation of their model as the solution to a stochastic equation driven by a Poisson measure, which does not hold in our more general setting. Nevertheless, let us acknowledge that their techniques allow to derive central limit theorems for their models.

1.6 Further discussion

Let us discuss some practical implications and limitations of our results for epidemiological applications, as well as some avenues for future work.

Impact of the initial condition. A major limitation to the practical interest of Theorem 1.1 is that the age structure of the initial population should converge to a known limit, for which a positive fraction of individuals are infected. This means that our result could only be applied once the epidemic has been spreading for a long enough time, and that the initial age structure of the population needs to be prescribed. In practice this age structure can hardly be estimated.

In applications, the large number of individuals observed at $t = 0$ results from the growth of the epidemic out of a few initial individuals. It is thus natural to try to derive a law of large numbers similar to Theorem 1.1 but started from a few infected individuals. Such a result was already derived in [5], for $c \equiv 1$ and under some additional technical assumptions. It was shown that the limit of the age structure then converges to an extension of (1.3) to the whole real line $t \in \mathbb{R}$. This extension is unique up to a shift [13], and does not depend on any initial age structure, solving the above issue. We expect that a similar result holds in our setting with $c \neq 1$. However, in this case, the solutions to (1.3) on the real line are neither unique nor shift-invariant. It is a more delicate issue to describe these solutions, and to understand which one of them is selected by the

initial randomness of the stochastic process. This relates to existing work on dynamical systems perturbed by a small noise and started near an unstable equilibrium [6, 4].

Speed of convergence and deviations. Theorem 1.1 provides a rigorous justification to the use of deterministic age-structured epidemic models, as limits of a large class of stochastic individual-based models. For the purpose of applications, it would be desirable to understand quantitatively how accurate this approximation is, that is, to derive a speed of convergence of the stochastic model to its deterministic limit. An even more important question for statistical applications would be to characterize the deviation of the stochastic system from the limit. We have derived our law of large numbers under a minimal first moment assumption (1.1) on the infection point process. We expect that a central limit theorem similar to those obtained in [34, 14, 42] should hold under a second moment assumption on \mathcal{P} . This would entail that the fraction of individuals in the various compartments remains at a distance of order $1/\sqrt{N}$ from the deterministic limit, and would provide a natural limiting expression for the likelihood of the process. Note that, since we do not assume that \mathcal{P} is a Poisson or a Cox process, the correlation structure of the limiting Gaussian process should be different from than in [34, 14, 42] and more similar to the co-variance structure of a Crump–Mode–Jagers process, described for instance in [28].

1.7 Outline

The rest of this paper is organized as follows. A formal description of the model is provided in Section 2, and the Kermack–McKendrick PDE is studied in Section 3.

Section 4 contains the graph construction of our model, as well as a rigorous definition of the ancestral process mentioned in Section 1.3. Our proofs rely on showing the local weak convergence of the graph of potential infectors to a limiting Poisson tree. Section 5 describes this limiting tree, and provides a characterization of the transmission chain leading to the infection of individual in terms of the h -transform of a renewal process. Finally, the convergence to the Poisson tree is carried out in Section 6 and our law of large numbers is proved in Section 7.

2 Description of the model

In the following, we will consider an epidemic model in which individuals' life trajectories are represented by independent stochastic processes. We distinguish between two types of individuals:

- Susceptible individuals that have never been infected before.
- Infected individuals that have been infected in the past. We emphasize that the meaning of infected is a bit broader than usual. For instance, a recovered or dead individual is considered as infected. To each infected individual, we associate an age. The age is the time elapsed since the beginning of the infection.

There are N individuals in the population. To each individual $x \in [N]$, we associate a pair of processes (\mathcal{P}_x, X_x) describing respectively the process of secondary infections and the successive stages of the disease experienced by the focal individual x . More precisely:

- The *life-cycle process*, denoted by $(X_x(a); a \geq 0)$, is a random process valued in \mathcal{S} where $X_x(a)$ is the stage of the disease (e.g., exposed, death, etc.) of x at age a .
- The *infection point process* \mathcal{P}_x is a point measure describing the ages of potential infections.

Let us denote by $\mathcal{X}_x := (\mathcal{P}_x, X_x)$. We will always assume that $(\mathcal{X}_x; x \in [N])$ are i.i.d. and denote by $\mathcal{X} = (\mathcal{P}, X)$ their common distribution. The state space of \mathcal{X} is denoted by \mathcal{X} .

Remark 2.1. Note that we allow for non-trivial dependence between the life-cycle and the infection process. Examples of such dependence can be that a deceased individual is not infectious anymore, a hospitalized individual may have a reduced potential of infection due to quarantine, etc.

We suppose that at $t = 0$, every individual is independently infected with probability I_0 . Let \mathcal{I}_0^N be the set of initially infected individuals. For each $x \in \mathcal{I}_0^N$ we need to prescribe an age, or equivalently, an infection time. We assume that, conditional on \mathcal{I}_0^N , the ages of the initial individuals $(Z_x; x \in \mathcal{I}_0^N)$ are i.i.d. with common distribution g . We also set $Z_x = 0$ for $x \notin \mathcal{I}_0$ for convenience. Let us denote by $(\sigma_x^N; x \in \mathcal{I}_0^N)$ the infection time of the initial infected, that is, $\sigma_x^N = -Z_x$.

The epidemic now spreads as follows. Suppose that, at some time t_0 , we have defined a set $\mathcal{I}_{t_0}^N \subseteq [N]$ of infected individuals at time t_0 , and a vector $(\sigma_x^N; x \in \mathcal{I}_{t_0}^N)$ of infection times. Let t_1 be the first atom after t_0 of the point measure

$$\sum_{x \in \mathcal{I}_{t_0}^N} \sum_{a \in \mathcal{P}_x} \delta(\sigma_x^N + a).$$

If there is no such atom, the infection stops. Otherwise, let U be uniformly chosen in $[N]$, independent of the rest: it is the first individual that comes in contact with any of the infected individuals after time t_0 . If $U \in \mathcal{I}_{t_0}^N$, then nothing happens, and we carry out the same procedure for the next atom t_2 . If $U \notin \mathcal{I}_{t_0}^N$, then, with probability $1 - c(t_1)$, the infection is ineffective in which case nothing happens and we consider the next infection time t_2 . Otherwise, set $\mathcal{I}_{t_1}^N = \mathcal{I}_{t_0}^N \cup \{U\}$ and $\sigma_U^N = t_1$, and continue the procedure as if starting from time t_1 with the initial infected set $\mathcal{I}_{t_1}^N$. This inductive procedure will be reformulated in terms of an infection graph in Section 4.1.

3 Kermack-McKendrick PDE

In this section we provide our definition of the solution to the Kermack-McKendrick equation (1.3). We start with a formal resolution of the PDE using the method of characteristics.

Let I_0 be the initial density of infected individuals and g the initial age profile of the population. First, note that if n is solution of the PDE, then for every pair (t, a) of non-negative numbers, $s \mapsto n(t - s, a - s)$ is constant on $(0, t \wedge a)$. This yields

$$\forall t, a \geq 0, \quad n(t, a) = \begin{cases} I_0 g(a - t) & \text{when } a > t \\ b(t - a) & \text{when } a \leq t, \end{cases} \tag{3.1}$$

with

$$\forall t \geq 0, \quad b(t) := n(t, 0)$$

is the number of new infections at time t . Moreover,

$$\begin{aligned} \dot{S}(t) &= - \int_0^\infty \partial_t n(t, a) da = \int_0^\infty \partial_a n(t, a) da \\ &= -b(t) = -c(t)S(t) \int_0^\infty \tau(a)n(t, a) da. \end{aligned}$$

As a result, since $S(0) = S_0$, we have

$$\begin{aligned} S(t) &= S_0 \exp \left(- \int_0^t c(s) \int_0^\infty \tau(a)n(s, a) \, da \, ds \right) \\ &= S_0 \exp \left(- \int_0^t c(s) \left(\int_0^s \tau(a)b(s-a) \, da + I_0 \int_s^\infty \tau(a)g(a-s) \, da \right) ds \right) \\ &= S_0 \exp \left(- \int_0^t c(s) \left(\int_0^s \tau(s-a)b(a) \, da + I_0 \bar{\tau}(s) \right) ds, \right) \end{aligned}$$

where $\bar{\tau}(s)$ was defined in (1.8), so necessarily

$$B(t) := \int_0^t b(s) \, ds = S_0 - S(t)$$

solves the nonlinear delay equation

$$B(t) = S_0 \left[1 - \exp \left(- \int_0^t c(s) \left(\int_0^s \tau(s-a) B(da) + I_0 \bar{\tau}(s) \right) ds \right) \right] \tag{3.2}$$

where $B(da) = b(a) \, da$ is the Stieltjes measure associated to the nondecreasing map B . This motivates the following definition of a solution to the Kermack–McKendrick equation.

Definition 3.1. We say that $(n(t, a); t, a \geq 0)$ is a weak solution to (1.3) if there exists a nonnegative function $(b(t); t \geq 0)$ such that:

1. the functions n and b are related through (3.1);
2. the function $B(t) := \int_0^t b(s) \, ds$ solves the delay equation (3.2).

If a nondecreasing function B satisfies (3.2), then we have the following inequality:

$$B(t+u) - B(t) \leq S_0 \int_t^{t+u} c(s) \left(\int_0^s \tau(s-a) B(da) + I_0 \int_0^\infty \tau(a+s)g(a) \, da \right) ds. \tag{3.3}$$

The previous inequality readily entails that B is absolutely continuous, and thus that we can find b such that $B(t) = \int_0^t b(s) \, ds$. Therefore, existence and uniqueness of solutions to (1.3) reduce to existence and uniqueness of nondecreasing solutions to (3.2), which is provided by the following result.

Lemma 3.2. There is a unique nondecreasing, nonnegative solution to (3.2).

Proof. Let us denote by E the set of all nondecreasing, nonnegative, càdlàg functions on $[0, \infty)$. Recall the definition of the Malthusian parameter α from (1.2). Fix some $\gamma > \alpha \vee 0$, so that we have

$$\int_0^\infty e^{-\gamma t} \tau(t) \, dt < 1. \tag{3.4}$$

Define

$$E_\gamma = \{f \in E : \int_0^\infty e^{-\gamma t} f(t) \, dt < \infty\}.$$

We endow E_γ with the metric

$$d_\gamma(f, g) = \int_0^\infty e^{-\gamma t} |f(t) - g(t)| \, dt$$

which makes (E_γ, d_γ) a complete metric space. As any solution to (3.2) is bounded and continuous, it is sufficient to show existence and uniqueness of the solution in E_γ .

We introduce the operator $\Phi: E_\gamma \rightarrow E_\gamma$ such that

$$\begin{aligned} \Phi f(t) = S_0 & \left(1 - \exp \left(- \int_0^t c(s) \left(\int_0^s \tau(s-a) f(da) \right) ds \right. \right. \\ & \left. \left. - I_0 \int_0^\infty \left(\int_0^t c(s) \tau(a+s) ds \right) g(a) da \right) \right), \end{aligned}$$

where $f(da)$ denotes the Stieltjes measure associated to f . Note that $\Phi f \in E_\gamma$, since it is clear that Φf is bounded, continuous, nonnegative and nondecreasing. Let us show that Φ is a contraction. We have, for $f_1, f_2 \in E_\gamma$,

$$\begin{aligned} d_\gamma(\Phi f_1, \Phi f_2) & \leq S_0 \int_0^\infty e^{-\gamma t} \left| \int_0^t c(s) \left(\int_0^s \tau(s-a) f_1(da) - \int_0^s \tau(s-a) f_2(da) \right) ds \right| dt \\ & \leq \int_0^\infty e^{-\gamma t} \left(\int_0^t \tau(s) |f_1(t-s) - f_2(t-s)| ds \right) dt \\ & = d_\gamma(f_1, f_2) \int_0^\infty e^{-\gamma t} \tau(t) dt. \end{aligned}$$

By (3.4) we know that $\int_0^\infty e^{-\gamma t} \tau(t) dt < 1$, showing that Φ is a contraction. The Banach fixed point theorem therefore shows that there exists a unique $B \in E_\gamma$ such that $\Phi B = B$, ending the proof. \square

4 Graph of infection

4.1 Infection graph

Recall the infection model defined in Section 2, and the notation $(\mathcal{P}_x; x \in [N])$ for the infection point processes. Recall that $(Z_x; x \in [N])$ are i.i.d. random variables with law

$$Z_x \sim \delta_0(da)(1 - I_0) + I_0 g(a) da, \tag{4.1}$$

and that we have defined $\mathcal{I}_0^N = \{x : Z_x > 0\}$ for the set of initially infected individuals, and $(\sigma_x^N = -Z_x; x \in \mathcal{I}_0^N)$ for their infection (or birth) time. Intuitively, Z_x encodes the age of infection of individual x at time $t = 0$. Susceptible individuals have age 0, whereas the age of an infected individual is chosen according to the density g . Define the shifted infection measure

$$\widehat{\mathcal{P}}_x = \sum_{a \in \mathcal{P}_x} \mathbb{1}_{\{a - Z_x \geq 0\}} \delta(a - Z_x) \tag{4.2}$$

Note that if x is susceptible (i.e., $Z_x = 0$), we have $\widehat{\mathcal{P}}_x = \mathcal{P}_x$. Vertices with $Z_x = 0$ will be said of type susceptible (S). Vertices with $Z_x > 0$ will be said of type infected (I).

Recall that each atom of a point process $\widehat{\mathcal{P}}_x$ encodes a potential infectious contact, which is targeted to a uniformly chosen individual in the population. We enrich the infection point processes by adding the information about the label of the target individual. Formally, we consider a sequence of i.i.d. random variables $(U_{x,i}; x \in [N], i \in \mathbb{N})$ uniformly distributed on $[N]$. Define

$$\forall x \in [N], \quad \widehat{\mathcal{N}}_x := \sum_{a_i \in \widehat{\mathcal{P}}_x} \delta(a_i, U_{x,i}) \tag{4.3}$$

where $a_1 < a_2 < \dots$ in the sum are the atoms of $\widehat{\mathcal{P}}_x$ listed in increasing order. We now build a graph out of the family $(\widehat{\mathcal{N}}_x; x \in [N])$ that records the potential infections in the population.

Definition 4.1 (Infection graph). *The infection graph built from the i.i.d. collection of triplets $(\widehat{N}_x, X_x, Z_x; x \in [N])$ is the random oriented geometric marked graph $\mathcal{G}^N = (V^N, E^N)$ with $V^N = [N]$ and*

$$E^N = \bigcup_{x \in [N]} \bigsqcup_{(a,j) \in \widehat{N}_x} \{(i,j)\},$$

where the second union is a disjoint union, meaning that for each pair (i, j) we allow for multiple edges from i to j in the infection graph. The marks and edge lengths are defined as follows.

1. Each edge $e = (i_e, j_e)$ corresponds to an atom (a_e, j_e) of the point process \widehat{N}_{i_e} defined in (4.3). The age a_e will be referred to as the length of edge e .
2. For each vertex $x \in V^N$, we define the mark at x as

$$m_x := (Z_x, \mathcal{X}_x),$$

where the initial infection age Z_x is defined in (4.1).

Remark 4.2. As stated in the theorem, \mathcal{G}^N is an oriented geometric marked graph. By geometric, we mean that edges have lengths. The orientation is dictated by the direction of potential infections, and the meaning of an edge (i, j) is that individual j is potentially infected by i . Finally, the first coordinate of the marking allows to distinguish between infected and susceptible individuals at time $t = 0$.

A path in \mathcal{G}^N is a set of edges $\pi = (e_1, \dots, e_n)$ such that, $j_{e_k} = i_{e_{k+1}}$, with the notation (i_e, j_e) for the origin and target vertices of the edge e . The length of a path $|\pi|$ is defined as

$$|\pi| = \sum_{e \in \pi} a_e.$$

The genealogical (or topological) distance is defined as the number of edges composing the path (n in our specific example). For $k \leq n$, we define the k -truncation of π as

$$\tau_k \pi := (e_1, \dots, e_k).$$

We say that π is a path from i to j if $i_{e_1} = i$ and $j_{e_n} = j$. A path in \mathcal{G}^N from i to j corresponds to a potential infection chain between i and j . The length of the path is the time elapsed between the infection of i and j . The genealogical distance is the number of infectors along the chain.

It turns out the infection graph that we have constructed corresponds to a directed version of a configuration model. The (undirected) configuration model is a well-studied random graph model where, starting from a prescribed number of half-edges for each of the N vertices (D_1, \dots, D_N) , a random graph is obtained by pairing these half-edges uniformly at random [45, Section 2.2.2]. Let us make this connection explicit.

For $x \in [N]$, denote by D_x^{out} (resp. D_x^{in}) the out-degree (resp. in-degree) of vertex x in \mathcal{G}^N . Clearly, $D_x^{\text{out}} = |\widehat{\mathcal{P}}_x|$ and, since each out-edge is pointing towards a uniformly chosen vertex, conditional on $(D_x^{\text{out}})_x$,

$$(D_1^{\text{in}}, \dots, D_N^{\text{in}}) \sim \text{Multinomial}\left(\sum_{x \in [N]} D_x^{\text{out}}; \left(\frac{1}{N}, \dots, \frac{1}{N}\right)\right). \tag{4.4}$$

Suppose that $(D_x^{\text{out}}, D_x^{\text{in}})_x$ are prescribed. We can construct a graph with this given sequence of in and out degrees in the following way:

1. attach to each vertex $x \in [N]$ a number D_x^{out} of out half-edges, and a number D_x^{in} of in half-edges; and

2. pair each out half-edge to a different in half-edge.

If the pairing in the second step is made uniformly among all possibilities, the resulting random graph is called a *directed configuration model* with degree sequence $(D_x^{\text{out}}, D_x^{\text{in}})_x$.

In the infection graph it is not hard to see that, again because each out-edge is pointing towards a uniformly chosen vertex, conditional on $(D_x^{\text{out}}, D_x^{\text{in}})_x$ the pairing of in- and out-edges in \mathcal{G}^N is made uniformly. Furthermore, conditional on $(D_x^{\text{out}}, D_x^{\text{in}})_x$, the marks $(m_x)_x$ are independent, and m_x has the distribution of (Z, \mathcal{X}) conditional on $|\widehat{\mathcal{P}}_x| = D_x^{\text{out}}$. We record this connection as a proposition for later use.

Proposition 4.3. *Let (V^N, E^N) be a directed configuration model on N vertices with degree sequence $(D_x^{\text{out}}, D_x^{\text{in}})_x$ where $(D_x^{\text{out}})_x$ are i.i.d. and distributed as $|\widehat{\mathcal{P}}|$ and $(D_x^{\text{in}})_x$ are distributed as (4.4). Conditional on (V^N, E^N) , mark the vertices and add edge lengths in such a way that:*

1. the marks $(m_x)_x$ are independent and

$$m_x \sim (Z, \mathcal{P}, X) \text{ conditional on } |\widehat{\mathcal{P}}| = D_x^{\text{out}};$$

2. if (e_i) are the edges going out of x , then $a_{e_i} = A_i$, where $(A_i; i \leq |\widehat{\mathcal{P}}_x|)$ is a uniform permutation of the atoms of $\widehat{\mathcal{P}}_x$.

Then $(V^N, E^N, (m_x)_x, (a_e)_e)$ is distributed as the infection graph \mathcal{G}^N .

4.2 Infection process

Conditional on a realization of the infection graph (V^N, E^N) , we attach an additional independent random variable s_e uniform on $[0, 1]$ to every edge $e \in E^N$ of the graph. This random variable will encode what we will call the contact intensity of edge e . Roughly speaking, if the contact occurs at time t , this contact translates into an infection iff two conditions are satisfied. First, the contact intensity should be strong enough in the sense that $s_e \leq c(t)$ (see (4.5) below). Secondly, the target individual should not have been infected before (see (4.6) below). We make this more precise in the next definition; see also Figure 3.

Definition 4.4 (Active geodesic). *Let $\pi = (e_1, \dots, e_n)$ be a path with $i_{e_1} \in \mathcal{I}_0^N$. The path is said to be active iff*

$$\forall k \leq n, \quad s_{e_k} \leq c(|\tau_k \pi|). \tag{4.5}$$

For every $x \notin \mathcal{I}_0^N$, let $\Xi^N(x)$ be the set of active paths from \mathcal{I}_0^N to x . The path is said to be the active geodesic from \mathcal{I}_0^N to x iff

$$\forall k \leq n, \quad \tau_k \pi = \arg \min_{\pi' \in \Xi^N(j_{e_k})} |\pi'|. \tag{4.6}$$

Finally, we define the infection time of x — denoted by σ_x^N — as the length of the active geodesic from \mathcal{I}_0^N to x , with the convention that $\sigma_x^N = \infty$ if the geodesic does not exist.

Remark 4.5. 1. Since τ has a density, there is at most one path satisfying the minimization problem (4.6).

2. If $c \equiv 1$, then any path in the infection graph is active, so that our definition coincides with the usual definition of a geodesic on a geometric graph. In particular, (4.6) just states that if $\pi = (e_1, \dots, e_n)$ is the geodesic from \mathcal{I}_0^N to x , then the truncated path $\tau_k \pi$ is the geodesic from \mathcal{I}_0^N to j_{e_k} . Thus, when $c \equiv 1$, all the information about the infection process is contained in the infection graph and the extra variables s_e do not play any role.

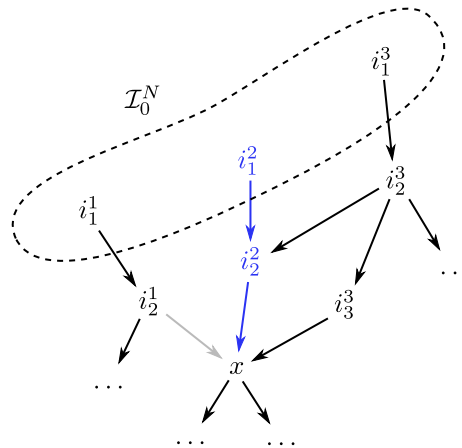


Figure 3: Ancestors of individual x in \mathcal{G}^N . In this picture, we have four potential infection paths from \mathcal{I}_0^N to x : $\pi^1 = (e_1^1, e_2^1)$, $\pi^2 = (e_1^2, e_2^2)$, $\pi^3 = (e_1^3, e_2^3, e_3^3)$ and $\pi^4 = (e_1^3, e_*, e_2^2)$, where we write $e_k^\ell = (i_k^\ell, i_{k+1}^\ell)$ and $e_* = (i_2^3, i_2^2)$. Assume first that π^1 is the shortest path, but that $s_{e_2^1} > c(|\pi^1|)$ — the edge is grayed out in the figure. Then π^1 is not an active path. Now let us assume that π^2 is active and that $|\tau_1 \pi^2| = a_{e_2^2} < a_{e_1^3} + a_{e_*} = |\tau_2 \pi^4|$. This means that π^4 cannot be the active geodesic. Finally, if π^2 and π^3 are two active paths and $|\pi^2| < |\pi^3|$, then π^2 (in blue) is the active geodesic from \mathcal{I}_0^N and $\sigma_x^N = |\pi^2|$.

4.3 The ancestral path

Definition 4.6 (Infection and ancestral paths).

- Let us consider x of type (S) such that $\sigma_x^N < \infty$ and write $\pi = (e_1, \dots, e_n)$ with $e_k = (i_k, j_k)$ for the active geodesic from \mathcal{I}_0^N to x . We define the infection path \mathcal{R}_x^N from x to \mathcal{I}_0^N as

$$\mathcal{R}_x^N(0) = \sigma_x^N, \quad \forall \ell \in [n], \quad \mathcal{R}_x^N(\ell) = \sigma_x^N - \sum_{k=0}^{\ell-1} (a_{e_{n-k}} + Z_{i_{n-k}}). \quad (4.7)$$

Finally, we define the ancestral process as

$$\mathcal{A}_x^N := \left(\mathcal{R}_x^N(\ell) \mathcal{X}_{v_\ell} \right)_{\ell=0}^n, \quad \text{where} \quad v_k = \begin{cases} i_{n+1-k} & \text{if } \ell \neq 0 \\ x = j_n & \text{if } \ell = 0, \end{cases}$$

to be the vector recording the information along the chain of infection (age of infection, infection measure, life-cycle).

- If x is of type (S) but $\sigma_x^N = \infty$, then \mathcal{A}_x^N is defined as the empty sequence.
- If x is of type (I) , then $\mathcal{A}_x^N := (\sigma_x^N, \mathcal{X}_x)$.

In words, the random path \mathcal{R}_x^N is obtained by tracing backward in time the chain of infection from the focal individual x to the set of initially infected individuals. The increments of the path are given by the successive age of infection. $\mathcal{R}_x^N(\ell)$ is the time of infection of the ℓ -th ancestor along the chain; the variable \mathcal{X}_{v_ℓ} encodes its infection and life-cycle processes. (Assuming that individuals are ranked from the focal individual x to the initial (I) individual.) Note that in the sum (4.7), all the $Z_{i_{n-k}}$ terms are equal to 0, except when $k = n - 1$. In words, the oldest ancestor is the only type (I) individual along the chain of infection. For $k = n - 1$, the corresponding increment is decomposed into two parts: (1) the undershoot a_{e_1} and (2) the overshoot Z_{i_1} corresponding to the age of infection of the (I) individual at time $t = 0$. See Figure 4.

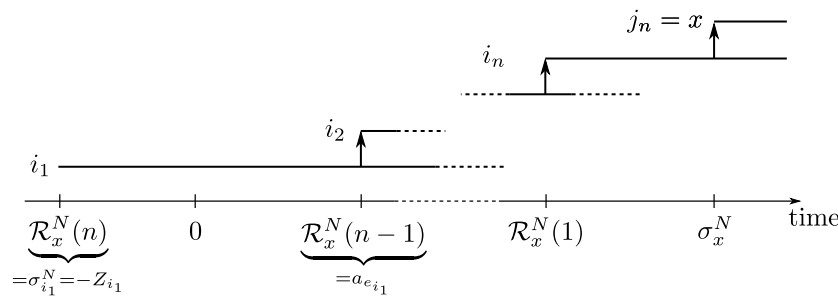


Figure 4: Infection path of individual x .

We close this section by a brief description of the topology underlying the set of ancestral paths. Let \mathcal{M} refer to the set of locally finite positive measures on $[0, \infty)$, and equip \mathcal{M} with a metric $d_{\mathcal{M}}$ that induces the vague topology [29, Section 4.1]. We denote by $d_{\mathcal{S}}$ the Skorohod metric on the set of càdlàg processes valued in \mathcal{S} denoted by $\mathbb{D}(\mathcal{S})$. The space $\mathbb{R}_+ \times \mathcal{M} \times \mathbb{D}(\mathcal{S})$ is equipped with the sup metric ρ defined as

$$\forall (x, y, z), (x', y', z'), \quad \rho((x, y, z), (x', y', z')) = \max(|x - x'|, d_{\mathcal{M}}(y, y'), d_{\mathcal{S}}(z, z')).$$

Each ancestral path is valued in the space

$$\bigcup_{n=0}^{\infty} \left(\mathbb{R}_+ \times \mathcal{M} \times \mathbb{D}(\mathcal{S}) \right)^n$$

equipped with the metric \mathcal{D} defined as follows

$$\mathcal{D}\left((m_1, \dots, m_n), (m'_1, \dots, m'_{n'})\right) = \begin{cases} 1 & \text{if } n \neq n' \\ 1 \wedge \max(\rho(m_1, m'_1), \dots, \rho(m_n, m'_{n'})) & \text{if } n = n'. \end{cases}$$

4.4 Local weak convergence

We introduce the notion of *local weak convergence* [1, 7]. Intuitively, a sequence of graphs converges in the local weak sense if the local structure around a typical vertex (meaning a uniformly chosen vertex) converges in distribution to a random limit. We make this definition precise.

A pointed oriented geometric marked (pogm) graph G is characterized by five coordinates $G = (V, E, (a_e), (m_x), \emptyset)$, respectively the set of vertices, the set of edges, $(a_e)_{e \in E}$ the lengths of the edges, $(m_x)_{x \in V}$ the set of marks, and $\emptyset \in V$ the pointed vertex. We let \mathcal{H} denote the set of pogm graphs, and equip it with a metric $d_{\mathcal{H}}$ so that $(\mathcal{H}, d_{\mathcal{H}})$ is a Polish space. A graph isomorphism ϕ between two *finite* pogm graphs $G = (V, E, (a_e), (m_x), \emptyset)$ and $G' = (V', E', (a'_e), (m'_x), \emptyset')$ is a bijection from V to V' such that

1. $(u, v) \in E$ iff $(\phi(u), \phi(v)) \in E'$.
2. ϕ maps the reference vertex of G to the reference vertex in G' .

By convention, we set $\min(\emptyset) = \infty$ in the following. Let $G = (V, E, (a_e), (m_x), \emptyset)$, $G' = (V', E', (a'_e), (m'_x), \emptyset')$ be two elements of \mathcal{H} . Define

$$d(G, G') = \min \left\{ 1, \min_{\phi} \left(\max_{e \in E} |a_e - a'_{\phi(e)}| \vee \max_{x \in V} |\rho(m_x, m'_{\phi(x)})| \right) \right\}$$

where the minimum is taken over all possible graph isomorphisms between the two graphs (in the sense prescribed above, that is, we only consider the isomorphisms

preserving the pointed vertex). If there is no such isomorphism between G and G' , we set $d(G, G') = 1$.

For $G \in \mathcal{H}$ and $y \in G$, the topological (or *genealogical*) distance to the reference vertex x is defined as

$$\inf\{n : \text{there exists a path } (y = x_0, \dots, x_n = x) \text{ in } G\}.$$

For every $r \in \mathbb{N}^*$, we denote by $[G]_r$, the subgraph induced by the vertices at a topological distance to the origin, that is, to the pointed vertex, less than r . For two elements $G, G' \in \mathcal{H}$, we define the (pseudo-)distance $d_{\mathcal{H}}$ as follows

$$d_{\mathcal{H}}(G, G') = \sum_r 2^{-r} d([G]_r, [G']_r).$$

The metric $d_{\mathcal{H}}$ naturally induces a notion of local convergence on (equivalence classes of) \mathcal{H} . Using standard arguments, we can see that $(\mathcal{H}, d_{\mathcal{H}})$ is a Polish space.

Given an oriented geometric marked graph G^N of size N , for $x \in [N]$ define $G^N(x)$ as the subgraph of G^N induced by all vertices y with an oriented *valid* path from y to x (including x itself), where we call a path $(y = y_0, y_1, \dots, y_k, x)$ valid if and only if for all $1 \leq i \leq k$, the node y_i is of type (S) . $G^N(x)$ is therefore the graph that contains all potential chains of infection leading to the infection of node x from an initially infected individual.

We treat $G^N(x)$ as an element of \mathcal{H} , with x as the reference vertex. We can construct a measure on \mathcal{H} out of the graph G^N by assigning the root x uniformly at random:

$$P(G^N) = \frac{1}{N} \sum_{x \in [N]} \delta_{G^N(x)}.$$

If the graph G^N is random, $P(G^N)$ is a *random* measure. The following definition is taken from Definition 3.6 in [21].

Definition 4.7 (Local weak convergence). *We say that a sequence of random pogr graphs $(G^N)_N$ converges in probability in the local weak sense to a random graph $G \in \mathcal{H}$ if*

$$P(G^N) \longrightarrow \mathcal{L}(G)$$

in probability for the weak topology on measures on \mathcal{H} , and where $\mathcal{L}(G)$ is the law of G .

We end this section with a direct consequence of the various definitions.

Lemma 4.8. *Consider a metric space E and a functional $\Phi: \mathcal{H} \rightarrow E$. Suppose that for all $N \geq 1$, G^N is a random pogr graph of size N , and that (G^N) converges in probability in the local weak sense to some other pogr graph G . If Φ is continuous on a set $A \subseteq \mathcal{H}$ such that $\mathbb{P}(G \in A) = 1$, then*

$$\frac{1}{N} \sum_{i=1}^N \delta_{\Phi(G^N)} \longrightarrow \mathcal{L}(\Phi(G))$$

in probability for the weak topology on measures on E , and where $\mathcal{L}(\Phi(G))$ is the law of $\Phi(G)$.

Proof. For a probability measure P on \mathcal{H} , let $P \circ \Phi^{-1}$ denote the push-forward measure of P by Φ . Clearly

$$\frac{1}{N} \sum_{i=1}^N \delta_{\Phi(G^N)} = P(G^N) \circ \Phi^{-1}.$$

According to the continuous mapping theorem, the result is proved if we can show that the mapping $P \mapsto P \circ \Phi^{-1}$ is continuous at $P = \mathcal{L}(G)$ for the weak topology. Let $P^N \rightarrow \mathcal{L}(G)$ weakly, and $\tilde{G}^N \sim P^N$. Another application of the continuous mapping theorem shows that $\Phi(\tilde{G}^N) \rightarrow \Phi(G)$ in distribution, showing the result. \square

5 A limiting Poisson random tree

5.1 Palm infection measures

Recall that $\mathcal{X}_x = (\mathcal{P}_x, X_x)$ is the pair encoding the infection and the life-cycle process and \mathcal{P} is a point process where each atom represents a potential infection event. Define $|\mathcal{P}| := \int d\mathcal{P}(a)$ which is interpreted as the total number of potential infections (or *contacts*) along the course of infection. We define a triplet of random variables $(W, \mathcal{P}^*, X^*) \equiv (W, \mathcal{X}^*)$ valued in $\mathbb{R}_+ \times \mathcal{M} \times \mathcal{S} \equiv \mathbb{R}_+ \times \mathcal{X}$ such that for every bounded continuous function f

$$\begin{aligned} \mathbb{E}\left(f(W, \mathcal{P}^*, X^*)\right) &= \frac{1}{R_0} \mathbb{E}\left(\int f(a, \mathcal{P}, X) d\mathcal{P}(a)\right) \\ &= \frac{1}{R_0} \mathbb{E}\left(|\mathcal{P}| \times \int \frac{1}{|\mathcal{P}|} f(a, \mathcal{P}, X) d\mathcal{P}(a)\right) \end{aligned}$$

In words, we first bias the pair $\mathcal{X} = (\mathcal{P}, X)$ by $|\mathcal{P}|$. Conditional on the resulting biased pair $\mathcal{X}^* = (\mathcal{P}^*, X^*)$, the r.v. W is obtained by picking an atom of the infection measure \mathcal{P}^* uniformly at random.

Definition 5.1 (Campbell and Palm measures). *The law of (W, \mathcal{X}^*) is the Campbell's measure associated to \mathcal{X} [3]. The Palm measure at $a \in \mathbb{R}_+^*$ is defined as the distribution of the random pair \mathcal{X}^* conditioned on the event $\{W = a\}$. We will use the notation $\mathcal{X}^{(a)}$ for a random variable with the Palm measure at a . See again [3] for a precise definition of this conditioning.*

Recall that τ is the intensity measure of \mathcal{P} defined in (1.1), and that we can write it as $\tau = R_0\nu$, where the total mass R_0 and the probability measure ν are defined in (1.5). The next result is standard from Palm measure theory.

Lemma 5.2. *The random variable W is distributed according to ν .*

5.2 Definition of the Poisson tree

Recall that we have defined $\bar{\tau}$ in (1.8) by

$$\forall u \geq 0, \quad \bar{\tau}(u) = \int_0^\infty g(a)\tau(a+u) da,$$

where g is the initial age density of infected individuals, and that we write $\bar{\tau} = \bar{R}_0\bar{\nu}$ where \bar{R}_0 is the mass of $\bar{\tau}$ and $\bar{\nu}$ the renormalized probability measure, see (1.9). Let us now consider a pair of random variables $(\bar{W}, Z) \in \mathbb{R}_+^2$ with joint density

$$\forall w, z > 0, \quad G(w, z) = \frac{1}{\bar{R}_0} g(z)\tau(w+z). \tag{5.1}$$

In particular, the first coordinate is distributed according to $\bar{\nu}$.

We now construct a Poisson marked random tree \mathcal{H} in two consecutive steps. (This extends the construction of Section 1.4 to the case $c \neq 1$.) First, the graph structure of \mathcal{H} depends on the two positive real parameters S_0R_0 , I_0R_0 , and second the random edge lengths and the marks are assigned through two probability distributions ν , $\bar{\nu}$ and the Palm measures described in the previous section.

Step 1. Graph structure. The graph structure is given by a Poisson Galton–Watson tree with two types:

- Start from a root \emptyset of type (S) .
- Susceptible (S) nodes have independent $\text{Poisson}(S_0 R_0)$ susceptible (S) offspring, and $\text{Poisson}(I_0 \bar{R}_0)$ infected (I) offspring.
- (I) nodes have no offspring.

In the following, let us consider the edges of the tree as being *oriented towards the root*.

Step 2. Decoration. Given the tree structure with distinguished (S) and (I) vertices, we now assign a marking $m_i = (Z_i, \mathcal{X}_i)$ to every vertex i , and a length a_e to every edge e as follows. If $i = \emptyset$, then $m_\emptyset = (0, \mathcal{X}_\emptyset)$ where \mathcal{X}_\emptyset is distributed as \mathcal{X} . For every $i \neq \emptyset$, there exists a unique oriented edge $e = (i, j)$ originated from i and

- If $i \in (I)$, let (a_e, Z_i) be chosen according to the density G . If $i \in (S)$, then $Z_i = 0$ and a_e is chosen according to ν .
- Conditional on (a_e, Z_i) , the variable \mathcal{X}_i has the Palm measure $\mathcal{X}^{(a_e + Z_i)}$ evaluated at $a_e + Z_i$.

Remark 5.3.

- If $e = (i, j)$ with $i \in (S)$ then (a_e, \mathcal{X}_i) has the Campbell measure introduced in Definition 5.1.
- If $i \in (I)$, then a_e is distributed according to $\bar{\nu}$.

The random tree \mathcal{H} will correspond to the local limit of the pogm graph $\mathcal{G}^N(x)$ conditioned on $\{Z_x = 0\}$. Let us now consider the infection process on \mathcal{H} introduced in Section 4.2. Conditional on \mathcal{H} , we endow each oriented edge e with a uniform random variable s_e (the intensity of the contact). As pointed out in Definition 4.4, those r.v.'s allow to determine whether a path is active or not and to determine the active geodesic at the root.

Define σ^∞ as the length of the active geodesic in \mathcal{H} from the set of (I) leaves to the root \emptyset . The following key result connects the distribution of σ^∞ to the delay equation.

Proposition 5.4. *Define*

$$\forall t \geq 0, \quad B(t) := S_0 \mathbb{P}(\sigma^\infty \leq t).$$

Then B solves the delay equation (3.2).

Proof. As we have assumed that τ has a density w.r.t. the Lebesgue measure, it is clear that this also holds for the distribution of σ^∞ . We denote its density by f . Let K , resp. \bar{K} , be the number of type (S) , resp. type (I) , children of the root of \mathcal{H} . Let $(\mathcal{H}_1, \dots, \mathcal{H}_K)$ denote the subtrees attached to the root \emptyset which are growing out of the type (S) children of the root. Let $(\sigma_1^\infty, \dots, \sigma_K^\infty)$ be the corresponding infection times, σ_i^∞ being obtained by determining the length of the active geodesic from the vertices of type (I) to the root in the tree \mathcal{H}_i . Moreover, let (W_1, \dots, W_K) and $(\bar{W}_1, \dots, \bar{W}_{\bar{K}})$ be the lengths of the edges ending at \emptyset and starting from an (S) and an (I) children respectively. (Recall that the edges of the Poisson tree are directed towards the root.) Finally, with a slight abuse of notation, let s_i be the contact intensity on the edge with length W_i . Let \bar{s}_i be defined analogously. Define

$$\chi_i := \mathbb{1}_{\{s_i \leq c(W_i + \sigma_i^\infty)\}}, \quad \bar{\chi}_i := \mathbb{1}_{\{\bar{s}_i \leq c(\bar{W}_i)\}}.$$

By definition of the active geodesic, we have that

$$\sigma^\infty = \left(\min_{1 \leq i \leq K} \{ \chi_i(W_i + \sigma_i^\infty) + (1 - \chi_i) \times \infty \} \right) \wedge \left(\min_{1 \leq i \leq \bar{K}} \{ \bar{\chi}_i \bar{W}_i + (1 - \bar{\chi}_i) \times \infty \} \right), \quad (5.2)$$

with the convention $0 \times \infty = 0$. Define $G(t) = \mathbb{P}(\sigma^\infty > t)$. Let W and \bar{W} be distributed according to ν and $\bar{\nu}$ respectively. By the branching property, conditional on K and \bar{K} , $(\sigma_i^\infty, W_i, \chi_i)_i$ and $(\bar{W}_i, \bar{\chi}_i)_i$ are two independent collections of i.i.d. random variables. Moreover

$$\begin{aligned} \mathbb{P}(\chi_i(W_i + \sigma_i^\infty) + (1 - \chi_i) \times \infty \leq t) &= \mathbb{E}(c(W + \sigma^\infty) \mathbb{1}_{\{\sigma^\infty + W \leq t\}}) \\ \mathbb{P}(\bar{\chi}_i \bar{W}_i + (1 - \bar{\chi}_i) \times \infty \leq t) &= \mathbb{E}(c(\bar{W}) \mathbb{1}_{\{\bar{W} \leq t\}}) \end{aligned} \quad (5.3)$$

Using these expressions, (5.2) and the branching property we have

$$\begin{aligned} G(t) &= \mathbb{E} \left\{ \left(1 - \mathbb{E}(c(\sigma^\infty + W) \mathbb{1}_{\{\sigma^\infty + W \leq t\}}) \right)^K \left(1 - \mathbb{E}(c(\bar{W}) \mathbb{1}_{\{\bar{W} \leq t\}}) \right)^{\bar{K}} \right\} \\ &= \mathbb{E} \left\{ \left(1 - \int_0^t \int_0^{t-a} c(a+s) f(s) \, ds \, \nu(da) \right)^K \left(1 - \int_0^t c(s) \bar{\nu}(ds) \right)^{\bar{K}} \right\} \\ &= \exp \left(- S_0 \int_0^t \int_0^{t-a} c(a+s) f(s) \tau(a) \, ds \, da \right. \\ &\quad \left. - I_0 \int_0^t g(a) \int_a^\infty c(u-a) \tau(u) \, du \, da \right), \end{aligned}$$

where, in the last equality, we have used the generating function of a Poisson distribution. It now follows that $B(t) = S_0(1 - G(t))$ satisfies (3.2). \square

5.3 The infection path conditioned on its length

Let us consider the infection process on \mathcal{H} as described in the previous section. For every realization in $\{\sigma^\infty < \infty\}$, define \mathcal{R}^∞ to be the infection path from \emptyset to the (I) leaves in \mathcal{H} , and let \mathcal{A}^∞ be the ancestral process defined analogously to Definition 4.6. In this section, we ask the following question: *conditional on the active geodesic to be of length t , what is the distribution of the vector of infection times along the geodesic?* In order to give an answer to this question, we start with some definition.

Let us consider \mathcal{R}^∞ to be the infection path from \emptyset to the (I) leaves in \mathcal{H} — see Definition 4.6. Our aim is to provide a description of \mathcal{R}^∞ conditional on $\{\sigma^\infty = t\}$. Define the process $\hat{R}^{(t)} \equiv \hat{R}$ as the \mathbb{R} -valued, nonincreasing Markov chain, started from t and stopped upon reaching $(-\infty, 0]$, with transition kernel $Q(x, y)$ defined for all $x > 0$ by

$$\begin{aligned} \forall y \geq x, \quad Q(x, y) &:= 0 \\ \forall y < x, \quad Q(x, y) &:= \frac{S(x)c(x)b(y)}{b(x)} \tau(x - y), \end{aligned}$$

where b is extended to the negative half-line with $b(-t) := I_0 g(t)$. The fact that Q defines a transition kernel follows from the renewal equation for b , which is obtained by differentiating (3.2) with respect to t :

$$\forall t \geq 0, \quad b(t) = c(t)S(t) \int_{-\infty}^t b(a)\tau(t - a)da. \quad (5.4)$$

Define

$$\hat{L}^{(t)} := \hat{L} = \inf \{ k : \hat{R}_k^{(t)} \leq 0 \}.$$

In the next proposition, we slightly abuse notation and identify $\hat{R}^{(t)}$ with its finite-length restriction to $[\hat{L}]$.

Proposition 5.5. *Let \mathcal{R}^∞ be the infection path from \emptyset to the (I) leaves. Conditional on $\{\mathcal{R}^\infty(0) = \sigma^\infty = t\}$,*

$$\mathcal{R}^\infty = \hat{R}^{(t)} \quad \text{in law.}$$

Proof. Recall that $\sigma^\infty = \mathcal{R}^\infty(0)$ is a random variable valued in $\mathbb{R}_+ \cup \{\infty\}$. By Proposition 5.4, the density of the random variable σ^∞ on \mathbb{R}_+ is given by $S_0^{-1}b(t)$. Let F be the joint probability density of the random pair $(\mathcal{R}^\infty(1), \mathcal{R}^\infty(0) - \mathcal{R}^\infty(1))$, and define

$$\forall t \geq 0, \forall x \leq t, \quad F^{(t)}(t-x) := \frac{F(x, t-x)}{S_0^{-1}b(t)},$$

so that $F^{(t)}$ corresponds to the density of the increment $\mathcal{R}^\infty(0) - \mathcal{R}^\infty(1)$ conditioned on $\{\mathcal{R}^\infty(0) = t\}$. Since \mathcal{H} is a Poisson random tree it is sufficient to understand the first step of the infection path, i.e., we need to show that

$$F^{(t)}(t-x) = \frac{c(t)S(t)b(x)\tau(t-x)}{b(t)}. \tag{5.5}$$

We use the same notation as in the proof of Proposition 5.4 and we distinguish between two cases.

Case 1: $x \in [0, t]$. In this case, the first individual along the geodesic is of type (S) . Let us work conditional on (K, \bar{K}) and compute the density $F(x, t-x)$. Fix a child $i \leq K$ of type (S) of the root \emptyset . By construction of the tree \mathcal{H} , the active geodesic leading to i and the length of the edge e_i going out of i toward the root are independent. Their joint density at $(x, t-x)$ is $S_0^{-1}b(x)\nu(t-x)$ by Proposition 5.4. For individual i to be part of the active geodesic leading to \emptyset , the edge e_i needs to be active, which occurs with probability $c(t)$, and the shortest active path going through any of the other children of the root must be longer than t . Using the expression (5.3), the probability of the latter event is

$$\left(\mathbb{E}(c(W + \sigma^\infty)\mathbb{1}_{\{\sigma^\infty + W \leq t\}})\right)^{K-1} \times \left(\mathbb{E}(c(\bar{W})\mathbb{1}_{\{\bar{W} \leq t\}})\right)^{\bar{K}}.$$

Summing over all K children of type (S) yields that

$$\begin{aligned} F^{(t)}(t-x) &= \frac{c(t)S_0^{-1}b(x)\nu(t-x)}{S_0^{-1}b(t)} \\ &\quad \times \mathbb{E}\left\{\mathbb{1}_{\{K \geq 1\}} K \left(1 - \mathbb{E}(c(W + \sigma^\infty)\mathbb{1}_{\{W + \sigma^\infty \leq t\}})\right)^{K-1} \left(1 - \mathbb{E}(c(\bar{W})\mathbb{1}_{\{\bar{W} \leq t\}})\right)^{\bar{K}}\right\} \\ &= \frac{c(t)b(x)\nu(t-x)}{b(t)} \times S_0 R_0 \\ &\quad \times \mathbb{E}\left\{\left(1 - \mathbb{E}(c(W + \sigma^\infty)\mathbb{1}_{\{W + \sigma^\infty \leq t\}})\right)^K \left(1 - \mathbb{E}(c(\bar{W})\mathbb{1}_{\{\bar{W} \leq t\}})\right)^{\bar{K}}\right\} \\ &= \frac{c(t)b(x)\tau(t-x)}{b(t)} \\ &\quad \times S_0 \mathbb{E}\left\{\left(1 - \mathbb{E}(c(W + \sigma^\infty)\mathbb{1}_{\{W + \sigma^\infty \leq t\}})\right)^K \left(1 - \mathbb{E}(c(\bar{W})\mathbb{1}_{\{\bar{W} \leq t\}})\right)^{\bar{K}}\right\} \end{aligned}$$

where in the second line, we used the fact that K is Poisson($S_0 R_0$) (so that the size-biased version of K is identical in law to $K + 1$). In the third line, we used the relation $\tau(u) = R_0 \nu(u)$. In Proposition 5.4, we showed that

$$S_0 \mathbb{E}\left\{\left(1 - \mathbb{E}(c(V + \sigma^\infty)\mathbb{1}_{\{V + \sigma^\infty \leq t\}})\right)^K \left(1 - \mathbb{E}(c(\bar{V})\mathbb{1}_{\{\bar{V} \leq t\}})\right)^{\bar{K}}\right\} = S_0 - B(t) = S(t).$$

This shows (5.5).

Case 2: $x \leq 0$. On this event the first vertex along the transmission chain is of type (I) . We use the same argument as in the case $x > 0$. Let $i \leq \bar{K}$ be a child of \emptyset of type (I) . Again, for this individual to be in the active geodesic, all paths going from an (I) individual to the root and not going through i need to be longer than t , and the edge from i to \emptyset needs to be active. In this case, $\mathcal{R}^\infty(1) = -Z_i$ and $\mathcal{R}^\infty(0) = \bar{W}_i$, where (\bar{W}_i, Z_i) have joint density G defined in (5.1). Thus the density of $(\mathcal{R}^\infty(1), \mathcal{R}^\infty(0) - \mathcal{R}^\infty(1))$ at $(x, t - x)$ is $G(t, -x) = g(-x)\tau(t - x)/\bar{R}_0$. This together with (5.3) lead to

$$\begin{aligned} F^{(t)}(t - x) &= \frac{c(t)g(-x)\tau(t - x)/\bar{R}_0}{S_0^{-1}b(t)} \\ &\quad \times \mathbb{E}\left\{ \left(1 - \mathbb{E}(c(W + \sigma^\infty)\mathbb{1}_{\{W + \sigma^\infty \leq t\}})\right)^K \mathbb{1}_{\{\bar{K} \geq 1\}} \right. \\ &\quad \left. \times \bar{K} \left(1 - \mathbb{E}(c(\bar{W})\mathbb{1}_{\{\bar{W} \leq t\}})\right)^{\bar{K}-1} \right\} \\ &= \frac{c(t)b(x)\tau(t - x)/\bar{R}_0}{I_0 S_0^{-1}b(t)} \times I_0 \bar{R}_0 \\ &\quad \times \mathbb{E}\left\{ \left(1 - \mathbb{E}(c(W + \sigma^\infty)\mathbb{1}_{\{W + \sigma^\infty \leq t\}})\right)^K \left(1 - \mathbb{E}(c(\bar{W})\mathbb{1}_{\{\bar{W} \leq t\}})\right)^{\bar{K}} \right\} \\ &= \frac{c(t)b(x)\tau(t - x)}{b(t)} \\ &\quad \times S_0 \mathbb{E}\left\{ \left(1 - \mathbb{E}(c(W + \sigma^\infty)\mathbb{1}_{\{W + \sigma^\infty \leq t\}})\right)^K \left(1 - \mathbb{E}(c(\bar{W})\mathbb{1}_{\{\bar{W} \leq t\}})\right)^{\bar{K}} \right\} \\ &= \frac{c(t)S(t)b(x)\tau(t - x)}{b(t)}. \end{aligned} \quad \square$$

5.4 Harmonic transform

In this section, we prove that the path $\hat{R}^{(t)}$ is the h -transform of a renewal process stopped upon reaching $(-\infty, 0]$. Throughout this section, we assume the existence of a unique Malthusian parameter $\alpha \in \mathbb{R}$ such that

$$\int \exp(-\alpha a)\tau(a) da = 1.$$

We define the probability density on \mathbb{R}_+^*

$$\forall a > 0, \quad r(a) := \exp(-\alpha a)\tau(a).$$

Let (Y_i) be a sequence of i.i.d. random variables with probability density r . Let $t > 0$ and define the renewal process $R^{(t)} \equiv R$ as follows

$$\forall k \geq 1, \quad R_k^{(t)} = t - \sum_{i=1}^k Y_i, \quad R_0^{(t)} = t.$$

We couple the renewal process R with a random variable $K^{(t)} \equiv K$ valued in $\mathbb{N} \cup \{\infty\}$ such that conditional on R ,

$$\forall j \geq 0, \quad \mathbb{P}(K = j | R) = \ell(R_0) \cdots \ell(R_{j-1})(1 - \ell(R_j)),$$

with $\ell(x) = \mathbb{1}_{\{x > 0\}}S(x)c(x) + \mathbb{1}_{\{x \leq 0\}}$.

Remark 5.6. Recall that c and S are valued in $[0, 1]$. Think of K as a killing time for the process R , i.e., at site $x > 0$, R dies with probability $1 - S(x)c(x)$, or makes a transition according to the distribution r with the remaining probability. Since by definition $\ell(x) = 1$ for all $x \leq 0$, if R reaches a negative state without being killed, it can no longer be killed.

Consider the filtration $(\mathcal{F}_k; k \geq 0)$ where

$$\mathcal{F}_k = \sigma((R_0, \chi_0), \dots, (R_k, \chi_k)), \quad \text{where } \chi_k = \mathbb{1}_{\{K \geq k\}},$$

and define the reaching time of $(-\infty, 0]$ as $L := \inf\{k : R_k \leq 0\}$.

Lemma 5.7. Define

$$M_k := b(R_{k \wedge L})e^{-\alpha R_{k \wedge L}} \chi_k.$$

The process $(M_k; k \geq 0)$ is a martingale with respect to the filtration $(\mathcal{F}_k; k \geq 0)$.

Proof. Let us compute the conditional expectation $\mathbb{E}(M_{k+1} | \mathcal{F}_k)$ for a realization on the event $A_k := \{R_k > 0, K \geq k\}$. The martingale property is obviously satisfied for any realization on the complementary event. Using the renewal equation (5.4) for b , we have

$$\begin{aligned} \mathbb{1}_{A_k} \mathbb{E}(M_{k+1} | \mathcal{F}_k) &= \mathbb{1}_{A_k} \mathbb{E}\left(b(R_{k+1})e^{-\alpha R_{k+1}} \mathbb{1}_{\{K \geq k+1\}} \mid \mathcal{F}_k\right) \\ &= \mathbb{1}_{A_k} S(R_k)c(R_k) \int_0^\infty b(R_k - a)e^{-\alpha(R_k - a)} \tau(a)e^{-\alpha a} da \\ &= \mathbb{1}_{A_k} e^{-\alpha R_k} S(R_k)c(R_k) \int_0^\infty b(R_k - a)\tau(a) da \\ &= \mathbb{1}_{A_k} b(R_k)e^{-\alpha R_k}. \quad \square \end{aligned}$$

Proposition 5.8. Let $h(s, u) := b(s)e^{-\alpha s u}$ and consider the h -transform of the two dimensional process (R, χ) . Then the process \hat{R} is the first coordinate of the h -transformed process.

Proof. On the one hand, the previous lemma implies that h is a harmonic function for the bivariate process (R, χ) . On the other hand, the transition kernel \hat{Q} for the h -transformed process can be rewritten explicitly as

$$\begin{aligned} \forall x, y, \quad \hat{Q}\left((x, 1), (y, 0)\right) &:= 0 \\ \forall x \leq y, \forall \epsilon \in \{0, 1\}, \quad \hat{Q}\left((x, 1), (y, \epsilon)\right) &:= 0 \\ \forall x > y, \quad \hat{Q}\left((x, 1), (y, 1)\right) &:= \frac{b(y)e^{-\alpha y}}{b(x)e^{-\alpha x}} S(x)c(x) \tau(x - y)e^{-\alpha(x - y)} \\ &= \frac{b(y)S(x)c(x)}{b(x)} \tau(x - y) \end{aligned}$$

It is now straightforward to check that \hat{R} is identical in law with the first coordinate of the h -transformed process. □

Let P be the law of the bivariate path (R, χ) stopped at $L = \inf\{k : R_k \leq 0\}$. Let \hat{P} be the law of h -transform $(\hat{R}, \hat{\chi})$ stopped at $\hat{L} = \inf\{k : \hat{R}_k \leq 0\}$. Then $\hat{P} \ll P$ and the Radon–Nikodym derivative is given by

$$\frac{d\hat{P}}{dP} = \frac{b(R_L) \exp(-\alpha R_L)}{b(t) \exp(-\alpha t)} \chi_L.$$

This immediately entails the following result.

Proposition 5.9. Assume that $g(t) = \alpha \exp(-\alpha t)$. Then \hat{P} is obtained by conditioning the renewal process R on not being killed before time L , and $b(t)$ can be written:

$$b(t) = \alpha e^{\alpha t} P(R^{(t)} \text{ is not killed before time } L).$$

Remark 5.10. Consider the linearized version of the Kermack–McKendrick equation

$$\begin{aligned} \partial_t n(t, a) + \partial_a n(t, a) &= 0 \\ \forall t \geq 0, n(t, 0) &= c(t) \int_0^\infty n(t, a) \tau(da) \\ \forall a \geq 0, n(0, a) &= I_0 g(a) \end{aligned}$$

obtained from (1.3) by assuming $S(t) = 1$. This can be thought as the age structure of a population where susceptibles are in excess. One can check that if $g(a) = \alpha \exp(-\alpha t)$, then $b_{\text{lin}}(t) := n(0, t) = \alpha e^{\alpha t}$. As a consequence, Proposition 5.9 can be rewritten as

$$b(t) = b_{\text{lin}}(t) P(R^{(t)} \text{ is not killed before time } L).$$

We close this section by a brief discussion on the previous result. In [18], we considered a “linearized” version of the present model by making the simplifying assumption that susceptible individuals are always in excess (branching assumption), so that the epidemic is described by a Crump–Mode–Jagers process. When $c \equiv 1$ and $R_0 > 1$, the process is supercritical. Starting from a single infected individual, there is a positive probability of non-extinction and conditional on this event, the number of infected grows exponentially at rate $\alpha > 0$. Further, it is well known from the seminal work of Jagers and Nerman [32] that under mild assumptions,

1. the age structure of the population converges to the exponential profile $g(t) = \alpha \exp(-\alpha t)$ mentioned in Proposition 5.9.
2. the infection path — interpreted as the ancestral line in the work of Jagers and Nerman — is well described by the renewal process R . More precisely, if one sample an infected individual at a large time t , its infection path converges to the renewal process R .

We can draw two conclusions out of those observations. As a consequence of the first item, the age structure $g(t) = \alpha \exp(-\alpha t)$ could be interpreted as the age structure emerging from a single infected individual in the past (provided that the initial fraction of infected individuals in our model is small). The second conclusion is that the effect of the conditioning in Proposition 5.9 encodes the effect of the saturation and the contact rate c on the genealogy. Recall that in the absence of saturation (branching approximation) and full contact rate ($c \equiv 1$), the infection path is distributed as the renewal process. When those effects are taken into account, Proposition 5.9 indicates that the law of the infection path is twisted in such a way that infection paths with infection occurring at low susceptible frequency (i.e. low values of S) and high contact rates c are favored. This is consistent with the intuition that ancestral infections tend to be biased towards periods when many infections occurred.

6 Convergence of the infection graph

We show in this section that the Poisson random tree \mathcal{H} constructed previously corresponds to the local weak limit of (S) vertices in the infection graph \mathcal{G}^N . This entails that the empirical distribution of any continuous functional of the graph in the local topology converges to the law of the corresponding functional for \mathcal{H} . In particular we will deduce our two main results, the convergence of the age structure and that of the historical process, by viewing the age of an individual x and its transmission chain as functionals of the active geodesic in \mathcal{G}^N leading to x . The key result of this section is the following.

Proposition 6.1. *The sequence of infection graphs $(\mathcal{G}^N)_N$ converges in probability in the local weak sense to a random pogm tree \mathcal{T} such that*

- with probability I_0 , \mathcal{T} is made of a single (I) vertex \emptyset , whose mark $(Z_\emptyset, \mathcal{X}_\emptyset)$ is distributed as $Z_\emptyset \sim g(a)da$ and $\mathcal{X}_\emptyset \sim (\mathcal{P}, X)$;
- with probability S_0 , \mathcal{T} is distributed as the random tree \mathcal{H} of Section 5.2.

In other words, the tree \mathcal{H} constructed in Section 5.2 corresponds to the law of \mathcal{T} , conditioned on starting from an (S) vertex.

Lemma 6.2. *For each N , let $(X_i^N; i \leq N)$ be some exchangeable random variables in some Polish state space, and (X_1, X_2) be two independent random variables with distribution $\mathcal{L}(X)$. Then*

$$(X_1^N, X_2^N) \rightarrow (X_1, X_2) \iff \frac{1}{N} \sum_{i=1}^N \delta_{X_i^N} \rightarrow \mathcal{L}(X)$$

where the two convergence are in distribution.

Proof. By exchangeability, for any continuous bounded ϕ, ψ ,

$$\mathbb{E} \left[\left(\frac{1}{N} \sum_{i=1}^N \phi(X_i^N) \right) \left(\frac{1}{N} \sum_{j=1}^N \psi(X_j^N) \right) \right] = \mathbb{E}[\phi(X_1^N)\psi(X_2^N)] + O\left(\frac{1}{N}\right). \quad (6.1)$$

If the random measure converges, then by dominated convergence

$$\mathbb{E} \left[\left(\frac{1}{N} \sum_{i=1}^N \phi(X_i^N) \right) \left(\frac{1}{N} \sum_{j=1}^N \psi(X_j^N) \right) \right] \longrightarrow \mathbb{E}[\phi(X_1)]\mathbb{E}[\psi(X_2)]$$

showing that the pair (X_1^N, X_2^N) converges in distribution to (X_1, X_2) . Conversely, using again (6.1), the convergence of (X_1^N, X_2^N) entails that

$$\mathbb{E} \left[\frac{1}{N} \sum_{i=1}^N \phi(X_i^N) \right] = \mathbb{E}[\phi(X_1^N)] \rightarrow \mathbb{E}[\phi(X_1)], \quad \mathbb{E} \left[\left(\frac{1}{N} \sum_{i=1}^N \phi(X_i^N) \right)^2 \right] \longrightarrow \mathbb{E}[\phi(X_1)]^2.$$

These two estimates prove that $\frac{1}{N} \sum_{i=1}^N \phi(X_i^N)$ converges in distribution to $\mathbb{E}[\phi(X_1)]$, which in turn shows that the measure $\frac{1}{N} \sum_{i=1}^N \delta_{X_i^N}$ converges to $\mathcal{L}(X)$, see for instance [29, Theorem 4.11]. \square

Proof of Proposition 6.1. We prove the result in three steps. First, we show the local weak convergence of the graph structure (without the marking) towards a limiting Galton–Watson tree $\tilde{\mathcal{T}}$. We make use of known results on the local weak convergence of configuration models. Then we show that $(\mathcal{G}^N)_N$ (with the marking) converges to the tree \mathcal{T} obtained by marking $\tilde{\mathcal{T}}$ appropriately. Finally we prove that the law of the limiting tree $\tilde{\mathcal{T}}$, conditional on starting from an (S) vertex and after removing all edges pointing towards an (I) vertex, is distributed as the Poisson tree \mathcal{H} .

Step 1. Recall that the infection graph \mathcal{G}^N can be constructed as a directed configuration model, see the notation in Proposition 4.3. We will use the known fact that the local weak limit of a configuration model is a Galton–Watson tree [45, Section 2.2.2]. We make use of a version of this result for directed graphs derived in [21, Proposition 6.2].

The local weak convergence in [21] is derived for a different class of oriented graphs than the pogm graphs introduced in this work. Namely, edges have no lengths and the vertices are marked with their out-degrees. Accordingly, let us denote by $\tilde{\mathcal{G}}^N$ the

oriented marked graph obtained by replacing the marks $(m_x)_x = (Z_x, \mathcal{X}_x)_x$ by the mark $(\tilde{m}_x)_x = (|\widehat{\mathcal{P}}_x|)_x$ and removing the edge lengths. Recall the notation $(D_x^{\text{out}})_x$ and $(D_x^{\text{in}})_x$ for the collection of in and out degree in $\tilde{\mathcal{G}}^N$. Three conditions need to be checked on this degree sequence to obtain the local weak convergence of $\tilde{\mathcal{G}}^N$, see Condition 6.1 of [21],

(a) for any positive bounded function ϕ , in probability,

$$\frac{1}{N} \sum_{x \in [N]} \phi(D_x^{\text{out}}, D_x^{\text{in}}) \longrightarrow \mathbb{E}[\phi(\mathcal{D}^{\text{out}}, \mathcal{D}^{\text{in}})];$$

(b) we have

$$\frac{1}{N} \sum_{x \in [N]} D_x^{\text{out}} \longrightarrow \mathbb{E}[\mathcal{D}^{\text{out}}], \quad \frac{1}{N} \sum_{x \in [N]} D_x^{\text{in}} \longrightarrow \mathbb{E}[\mathcal{D}^{\text{in}}]$$

in probability, and $\mathbb{E}[\mathcal{D}^{\text{out}}] = \mathbb{E}[\mathcal{D}^{\text{in}}]$; and

(c) for any positive bounded function ϕ , if $L^N = D_1^{\text{out}} + \dots + D_N^{\text{out}}$,

$$\frac{1}{L^N} \sum_{x \in [N]} D_x^{\text{out}} \phi(D_x^{\text{out}}, D_x^{\text{in}}) \longrightarrow \mathbb{E}[\phi(\mathcal{D}^{\star\text{out}}, \mathcal{D}^{\star\text{in}})]$$

in probability (note that we have removed a $1/N$ factor compared to [21] that should not appear);

for some random pair $(\mathcal{D}^{\text{out}}, \mathcal{D}^{\text{in}})$, and where $(\mathcal{D}^{\star\text{out}}, \mathcal{D}^{\star\text{in}})$ is obtained by size-biasing $(\mathcal{D}^{\text{out}}, \mathcal{D}^{\text{in}})$ by its first coordinate.

We check condition (a) by computing the second moment of the empirical distribution of degrees. Since the in-degrees follow the multinomial distribution (4.4), we have that

$$(D_1^{\text{in}}, D_2^{\text{in}}) \sim \text{Multinomial}(D_1^{\text{out}} + D_2^{\text{out}} + \sum_{i=3}^N D_i^{\text{out}}; (\frac{1}{N}, \frac{1}{N}))$$

so that, conditional on $(D_1^{\text{out}}, D_2^{\text{out}})$,

$$(D_1^{\text{in}}, D_2^{\text{in}}) \longrightarrow (D_1^{\text{in}}, D_2^{\text{in}})$$

which are independent Poisson random variables with mean $\mathbb{E}[|\widehat{\mathcal{P}}|] = S_0 R_0 + I_0 \bar{R}_0$. Using Lemma 6.2 proves that (a) holds where $(\mathcal{D}^{\text{out}}, \mathcal{D}^{\text{in}})$ are independent r.v. with $\mathcal{D}^{\text{out}} \sim |\widehat{\mathcal{P}}|$ and $\mathcal{D}^{\text{in}} \sim \text{Poisson}(S_0 R_0 + I_0 \bar{R}_0)$.

Point (b) is a direct application of the law of large numbers

$$\frac{1}{N} \sum_{i=1}^N D_i^{\text{out}} \longrightarrow \mathbb{E}[\mathcal{D}^{\text{out}}],$$

and point (c) follows from (a) and (b): using point (a) with $\phi(d, d') = \mathbb{1}_{\{(d, d') = (k, k')\}}$ we have that

$$\frac{1}{N} \text{Card}\{x : (D_x^{\text{out}}, D_x^{\text{in}}) = (k, k')\} \longrightarrow \mathbb{P}(\mathcal{D}^{\text{out}} = k, \mathcal{D}^{\text{in}} = k'),$$

in probability, and combining this with point (b) we have

$$\frac{k}{L^N} \text{Card}\{x : (D_x^{\text{out}}, D_x^{\text{in}}) = (k, k')\} \longrightarrow \frac{k}{\mathbb{E}[\mathcal{D}^{\text{out}}]} \mathbb{P}(\mathcal{D}^{\text{out}} = k, \mathcal{D}^{\text{in}} = k'), \quad (6.2)$$

in probability. This shows (c) for our specific choice of ϕ . For a general ϕ , up to extracting a subsequence, let us assume that a.s. (6.2) holds, for all $k, k' \geq 0$. Scheffé's lemma shows that this pointwise convergence can be reinforced to a convergence in $\ell_1(\mathbb{N} \times \mathbb{N})$, which readily entails (c).

Therefore, Proposition 6.2 in [21] shows that $\tilde{\mathcal{G}}^N$ converges in probability in the local weak sense towards a marked Galton–Watson tree $\tilde{\mathcal{T}}$ where each vertex u has:

1. a $\text{Poisson}(S_0 R_0 + I_0 \bar{R}_0)$ number of offspring (with edges oriented from the children towards the parents); and
2. an independent mark \tilde{m}_u distributed as $|\widehat{\mathcal{P}}|$ for the root and as the size-biasing of $|\widehat{\mathcal{P}}|$ for other vertices.

Note that in this tree there is no distinction between (I) and (S) vertices since part of the marking has been removed.

Step 2. We now show that \mathcal{G}^N (with the full marking) converges to a tree obtained by marking the limiting Galton–Watson tree as in Proposition 4.3. Let $x, y \in [N]$, and let $\tilde{\mathcal{G}}^N(x)$ and $\tilde{\mathcal{G}}^N(y)$ be the subgraphs of $\tilde{\mathcal{G}}^N$ induced by all the vertices with an oriented path to x and y respectively. By construction, each vertex in \mathcal{G}^N (and thus each vertex in $\tilde{\mathcal{G}}^N(x)$ and $\tilde{\mathcal{G}}^N(y)$) is indexed by an element of $[N]$ that we call its *label*. Recall the notation $[G]_r$ for the ball of radius r of a pogm G around the pointed vertex. Let B_r^N be the event “the labels of the vertices in $[\tilde{\mathcal{G}}^N(x)]_r$ and $[\tilde{\mathcal{G}}^N(y)]_r$ are distinct”. On this event Proposition 4.3 shows that, conditional on the unmarked graphs $[\tilde{\mathcal{G}}^N(x)]_r$ and $[\tilde{\mathcal{G}}^N(y)]_r$, the marks $(m_u)_u$ of the vertices in $[\mathcal{G}^N(x)]_r$ and $[\mathcal{G}^N(y)]_r$ are independent and

$$m_u \sim (Z, \mathcal{P}, X) \text{ conditioned on } |\widehat{\mathcal{P}}| = \tilde{m}_u. \tag{6.3}$$

Furthermore the lengths of the edges going out of u are sampled uniformly among the atoms of $\widehat{\mathcal{P}}_u$. Now, the first part of the proof and Lemma 6.2 show that $(\tilde{\mathcal{G}}^N(x), \tilde{\mathcal{G}}^N(y))$ converges in distribution to two independent copies $(\tilde{\mathcal{T}}_1, \tilde{\mathcal{T}}_2)$ of the limit Galton–Watson tree. Provided that $\mathbb{P}(B_r^N) \rightarrow 1$, this shows that in distribution

$$([\mathcal{G}^N(x)]_r, [\mathcal{G}^N(y)]_r) \rightarrow ([\mathcal{T}_1]_r, [\mathcal{T}_2]_r)$$

where the tree \mathcal{T}_i is obtained out of $\tilde{\mathcal{T}}_i$ by adding marks and edge lengths as in (6.3) and removing edges pointing to an (I) vertex. In turn, Lemma 6.2 proves that \mathcal{G}^N converges to \mathcal{T}_1 in probability in the local weak sense. It remains to show that $\mathbb{P}(B_r^N) \rightarrow 1$. This result is actually shown as a step in the proof of Proposition 6.2 from [21] that we have used in our Step 1. More precisely, the proof of [21, Lemma 6.4] shows that, with probability going to 1, the balls of radius r of two uniformly chosen vertices in the directed configuration model do not intersect, which is the result we need here. Let us explain heuristically why we expect this result to hold. The ball $[\mathcal{G}^N(x)]_r$ can be constructed by exploring the graph starting from x , following the in-edges in reverse direction, and pairing them with out-edges. Each time a new in-edge is explored, it is paired with an out-edge chosen uniformly from the unpaired out-edges in the graph. Since the total number of edges explored in $[\mathcal{G}^N(x)]_r$ and $[\mathcal{G}^N(y)]_r$ is negligible w.r.t. the total number of edges in \mathcal{G}^N (and since no vertex in \mathcal{G}^N has a number of out-edges of order N) the probability that the same vertex is explored both in $[\mathcal{G}^N(x)]_r$ and $[\mathcal{G}^N(y)]_r$ vanishes as $N \rightarrow \infty$. This argument is made rigorous in the proof of [21, Lemma 6.4].

Step 3. Let \mathcal{T} be distributed as the local weak limit of \mathcal{G}^N from the previous step. Our last task is to connect the distribution of \mathcal{T} to that of the Poisson tree \mathcal{H} from Section 5.2. Let us first take care of the root \emptyset . By definition of $\tilde{\mathcal{T}}$, $\tilde{m}_\emptyset \sim |\widehat{\mathcal{P}}|$ and conditional on \tilde{m}_\emptyset , $m_\emptyset \sim (Z, \mathcal{X})$ conditioned on $|\widehat{\mathcal{P}}| = \tilde{m}_\emptyset$. This readily shows that the mark of the root is distributed as (Z, \mathcal{X}) , so that in particular it is of type (I) and (S) with probability I_0 and S_0 respectively.

We now turn to some non-root vertex $u \in \tilde{\mathcal{T}}$. Recall that its mark \tilde{m}_u has the size-biased distribution of $|\widehat{\mathcal{P}}|$ and that $m_u = (Z_u, \mathcal{X}_u)$ is obtained as in (6.3). Let A_u be the length of its unique out-edge, which is uniformly chosen among the atoms of $\widehat{\mathcal{P}}_u$. We

have

$$\begin{aligned} \mathbb{E}\left[\phi(A_u, Z_u, \mathcal{X}_u)\right] &= \mathbb{E}\left[\mathbb{E}\left[\frac{1}{|\widehat{\mathcal{P}}|} \int \phi(a, Z, \mathcal{X}) \widehat{\mathcal{P}}(da) \mid |\widehat{\mathcal{P}}| = \tilde{m}_u\right]\right] \\ &= \frac{1}{\mathbb{E}[|\widehat{\mathcal{P}}|]} \mathbb{E}\left[\int \phi(a, Z, \mathcal{X}) \widehat{\mathcal{P}}(da)\right] \\ &= \frac{1}{S_0 R_0 + I_0 \bar{R}_0} S_0 \mathbb{E}\left[\int \phi(a, 0, \mathcal{X}) \mathcal{P}(da)\right] \\ &\quad + \frac{1}{S_0 R_0 + I_0 \bar{R}_0} I_0 \mathbb{E}\left[\int_0^\infty g(u) \int_u^\infty \phi(a - u, u, \mathcal{X}) \mathcal{P}(da) du\right]. \\ &= \frac{S_0 R_0}{S_0 R_0 + I_0 \bar{R}_0} \frac{1}{R_0} \mathbb{E}\left[\int \phi(a, 0, \mathcal{X}) \mathcal{P}(da)\right] \\ &\quad + \frac{I_0 \bar{R}_0}{S_0 R_0 + I_0 \bar{R}_0} \frac{1}{\bar{R}_0} \mathbb{E}\left[\int_0^\infty g(u) \int_u^\infty \phi(a - u, u, \mathcal{X}) \mathcal{P}(da) du\right], \end{aligned}$$

where in the first line we have used (6.3) and that A_u is a uniform atom of $\widehat{\mathcal{P}}_u$, in the second line that \tilde{m}_u has the size-biased distribution of $\widehat{\mathcal{P}}$ and in the third line the definition of $\widehat{\mathcal{P}}$ of (4.2). The result now follows upon identifying the terms. The prefactor in each term of the sums corresponds to the probability that $Z_u = 0$ or $Z_u \geq 0$, that is, that vertex u is of type (S) or (I). Since the total number of offspring in $\tilde{\mathcal{T}}$ follows a Poisson distribution with parameter $S_0 R_0 + I_0 \bar{R}_0$, the number of (S) and (I) offspring are independent Poisson random variables with means $S_0 R_0$ and $I_0 \bar{R}_0$ respectively. Moreover

$$\frac{1}{R_0} \mathbb{E}\left[\int \phi(a, \mathcal{X}) \mathcal{P}(da)\right] = \mathbb{E}[\phi(W, \mathcal{X}^*)]$$

where (W, \mathcal{X}^*) has the Campbell measure of Definition 5.1. Thus any (S) individual in \mathcal{T} has an edge length and mark distributed as (W, \mathcal{X}^*) as in \mathcal{H} . Similarly,

$$\begin{aligned} \frac{1}{\bar{R}_0} \mathbb{E}\left[\int_0^\infty g(u) \int_u^\infty \phi(a - u, u, \mathcal{X}) \mathcal{P}(da) du\right] \\ &= \frac{1}{\bar{R}_0} \int_0^\infty g(u) \int_u^\infty \tau(a) \mathbb{E}[\phi(a - u, u, \mathcal{X}^{(a)})] da du \\ &= \int_0^\infty \int_0^\infty G(v, u) \mathbb{E}[\phi(v, u, \mathcal{X}^{(u+v)})] dv du, \end{aligned}$$

where $\mathcal{X}^{(a)}$ has the Palm distribution of \mathcal{X} at a , and G is the probability density defined in (5.1). In the second line we have used the definition of the Palm measure. Identifying the terms, the mark of an (I) individual is obtained as that defined for \mathcal{H} . \square

7 Convergence of the historical process

We can now state and prove our main result. Let us introduce the historical process as the following empirical measure

$$H^N := \sum_{x \in [N]} \mathbb{1}_{\{\sigma_x^N < \infty\}} \delta_{\mathcal{A}_x^N}. \tag{7.1}$$

We also define the historical process at time $t \geq 0$ as the historical process of all individuals infected before time t ,

$$H_t^N := \sum_{x \in [N]} \mathbb{1}_{\{\sigma_x^N \leq t\}} \delta_{\mathcal{A}_x^N}.$$

Theorem 7.1 (Convergence of the historical process). *Let \mathcal{A}^∞ be the limiting ancestral process in the Poisson tree \mathcal{H} and let (σ_0, \mathcal{X}) denote a pair of independent random variables where $-\sigma_0$ is distributed according to the density g .*

(i) *For any $t \geq 0$ we have*

$$\frac{1}{N}H_t^N \longrightarrow S_0\mathbb{P}(\sigma^\infty \leq t)\mathcal{L}(\mathcal{A}^\infty \mid \sigma^\infty \leq t) + I_0\mathcal{L}(\sigma_0, \mathcal{X}).$$

where $\mathcal{L}(\mathcal{A}^\infty \mid \sigma^\infty \leq t)$ is the law of the random variable \mathcal{A}^∞ conditioned on the event $\{\sigma^\infty \leq t\}$, and the convergence is in distribution for the weak topology.

(ii) *If $(c(t); t \geq 0)$ converges as $t \rightarrow \infty$ we have that*

$$\frac{1}{N}H^N \longrightarrow S_0\mathbb{P}(\sigma^\infty < \infty)\mathcal{L}(\mathcal{A}^\infty \mid \sigma^\infty < \infty) + I_0\mathcal{L}(\sigma_0, \mathcal{X}).$$

in distribution for the weak topology.

The convergence result in (ii) is stronger than that in (i), but requires the mild assumption that the contact rate converges. Point (i) of the previous result is sufficient to derive the limit of the age structure of the epidemic, our Theorem 1.1. However, it is not sufficient to prove that the total number of individuals infected during the epidemic converges. This is a very well-studied quantity in epidemic modeling, referred to as the *final size* of the epidemic [31, 2], and our motivation for deriving point (ii) is the following corollary.

Corollary 7.2 (Final size of the epidemic). *Suppose that $(c(t); t \geq 0)$ converges as $t \rightarrow \infty$, then*

$$\frac{1}{N} \sum_{x \in [N]} \mathbb{1}_{\{\sigma_x^N < \infty\}} \longrightarrow 1 - \lim_{t \rightarrow \infty} S(t),$$

in distribution as $N \rightarrow \infty$.

Proof. By Theorem 7.1, point (ii), we have that

$$\frac{1}{N} \sum_{x \in [N]} \mathbb{1}_{\{\sigma_x^N < \infty\}} \longrightarrow S_0\mathbb{P}(\sigma^\infty < \infty) + I_0 = \lim_{t \rightarrow \infty} S_0\mathbb{P}(\sigma^\infty \leq t) + I_0.$$

By Proposition 5.4,

$$S_0\mathbb{P}(\sigma^\infty \leq t) = B(t) = S_0 - S(t),$$

so that

$$\lim_{t \rightarrow \infty} S_0\mathbb{P}(\sigma^\infty \leq t) + I_0 = I_0 + S_0 - \lim_{t \rightarrow \infty} S(t) = 1 - \lim_{t \rightarrow \infty} S(t). \quad \square$$

To prove the convergence of the historical process, we see the ancestral process \mathcal{A}_x^N as a functional of the pogm graph $\mathcal{G}^N(x)$ rooted at x . Provided we can show that the mapping taking a pogm graph to its active geodesic enjoys some appropriate continuity, the convergence of the historical process will follow from the local weak convergence of the infection graph \mathcal{G}^N .

For a deterministic pogm graph G , we can define an infection process by attaching to each edge e a uniform infection intensity s_e which determines if the edge is active or not, as in Section 4.2. It will be convenient to work conditional on (s_e) and to think of these infection intensities as a marking of the edges of the graph. It is straightforward to extend the definitions and results from Section 4.4 to include this marking, and that the convergence of the infection graph in Proposition 6.1 also remains valid for this extended marking: the infection graph \mathcal{G}^N , marked with uniform infection intensities,

converges in the local weak sense to the tree \mathcal{T} , also marked with uniform infection intensities.

For a pogm graph with fixed infection intensities, G , we can define $\mathcal{A}(G)$ as the ancestral process of G , which records the infection times along the active geodesic leading to the pointed vertex, as defined in Section 4.3. We also define $\sigma(G)$ as the length of the corresponding active geodesic. We can now prove that the ancestral process is a continuous functional of the local graph topology.

Lemma 7.3. *Let f be a continuous bounded functional on the space of ancestral paths. Then for any $t > 0$ the map*

$$G \mapsto f(\mathcal{A}(G))\mathbb{1}_{\{\sigma(G) < t\}} \tag{7.2}$$

is continuous at a.e. realization G of the tree \mathcal{T} . If the function $(c(t); t \geq 0)$ converges as $t \rightarrow \infty$, then f is continuous at a.e. realization of $\mathcal{A}(\mathcal{T})$.

Proof. The tree \mathcal{T} is either made of a singled (I) vertex, or is a copy of \mathcal{H} . Clearly, in the former case the result holds so that it remains to show it for almost every realization of \mathcal{H} . For some pogm tree G , if d is the genealogical distance and π_v denotes the unique path from v to the root, let

$$M_r(G) := \min_{\substack{u \in G \\ d(\emptyset, u) = r}} |\pi_u|$$

be the length of the shortest path from a vertex at distance r to the root. We start by showing in Step 1 that, almost surely, either \mathcal{H} is finite, or

$$M_r(\mathcal{H}) \rightarrow \infty \quad \text{as } r \rightarrow \infty. \tag{7.3}$$

Then, in Step 2, we show that (7.2) is continuous for almost all graphs G verifying this property. Under the additional assumption that $(c(t); t \geq 0)$ converges, we prove that f is continuous in Step 3 and Step 4.

Step 1. Let $(V_r; r \geq 0)$ be the process that records the ages of the (S) vertices of the Poisson tree \mathcal{H} , defined as

$$V_r := \sum_{\substack{u \in \mathcal{H}, d(u, \emptyset) = r \\ u \text{ of type } (S)}} \delta_{|\pi_u|},$$

Then $(V_r)_{r \geq 0}$ is a branching random walk with $\text{Poisson}(R_0 S_0)$ offspring distribution, and it follows from general results that, conditional on non-extinction, its minimum drifts to ∞ , see for instance Theorem 5.12 in [38]. As \mathcal{H} is obtained by attaching independently to any unmarked vertex a $\text{Poisson}(I_0 R_0)$ distributed number of (I) leaves, this shows that (7.3) also holds. This completes Step 1.

Step 2. First let us note that the marks (s_e) , representing the infection intensities, are independent of the structure of the tree \mathcal{T} and the lengths of its edges, so \mathcal{T} almost surely satisfies the following property, for all $r \in \mathbb{N}$:

$$\{s_e, e \text{ edge of } \mathcal{T}\} \cap \{c(t), t \in A_r^{\text{pot}}\} = \emptyset, \tag{7.4}$$

where A_r^{pot} is the (a.s. finite) set of lengths of all paths from (I) vertices at distance at most r from the root to (S) vertices. If a tree G satisfies this property, then it is clear that for any sequence $G^N \rightarrow G$, for N large enough, the r -neighborhood of the root in G^N has the same structure as that of G and in this neighborhood, a path from an (I) vertex to an (S) vertex is open in G^N if and only if it is open in G .

Fix some tree G satisfying (7.4), which is either finite or fulfills (7.3), and a sequence G^N converging to G in \mathcal{H} . We need to prove that

$$f(\mathcal{A}(G^N))\mathbb{1}_{\{\sigma(G^N) < t\}} \rightarrow f(\mathcal{A}(G))\mathbb{1}_{\{\sigma(G) < t\}} \tag{7.5}$$

It is readily checked that (7.5) holds if G is a finite tree. Suppose that G is infinite. If $\sigma(G) < \infty$, let r be such that $M_r(G) > \sigma(G)$. In particular there is an active path from an (I) vertex in $[G]_r$ to the root. The convergence $[G^N]_r \rightarrow [G]_r$ entails that, for N large enough, there is also an active path from an (I) vertex in $[G^N]_r$ to its root whose length converges to $\sigma(G)$, and that all other active paths from $G^N \setminus [G^N]_r$ to the root have a length larger than $\sigma(G)$ and thus cannot be the active geodesic. We are back to the case of a finite tree where (7.5) is readily checked. Finally, if $\sigma(G) = \infty$, fix r such that $M_r(G) > t$. The convergence $[G^N]_r \rightarrow [G]_r$ now entails that, for N large enough, there is no active path from an (I) vertex in $[G^N]_r$ to the root, so that $\sigma(G^N) > t$. This shows that in all three cases (7.5) holds and proves the first part of the result. We have also shown that f is continuous at every G that fulfills (7.3) and has an active geodesic, and that if $G^N \rightarrow G$ where G fulfills (7.3) and has no active geodesic, necessarily $\sigma(G^N) \rightarrow \infty$.

We now prove the second part of the result and assume that $(c(t); t \geq 0)$ converges to a limit c_* as $t \rightarrow \infty$. For a pogm graph G with infection intensities on its edges, we denote by G_s the pogm graph obtained by removing from G all edges e with an infection intensity $s_e > s$. We proceed again in two steps. In Step 3 we show that if \mathcal{H}_{c_*} is infinite, then \mathcal{H} has a.s. a geodesic. In Step 4 we consider a pogm graph G such that G_{c_*} is finite, and prove that if $G^N \rightarrow G$ then $\sigma(G^N)\mathbb{1}_{\{\sigma(G^N) < \infty\}}$ is bounded. Before moving to the proof of these two claims, let us show that they are sufficient to prove our result. If G is such that (7.3) holds and has an active geodesic, by Step 2 f is continuous at G . Therefore, by Step 3, f is continuous at a.e. realization G of \mathcal{H} such that G_{c_*} is infinite, or such that G_{c_*} is finite but G has an active path to the root. It remains to consider the case where G_{c_*} is finite and G has no active path. If $G^N \rightarrow G$, by Step 4 $\sigma(G^N)\mathbb{1}_{\{\sigma(G^N) < \infty\}}$ is bounded and by Step 2 $\sigma(G^N) \rightarrow \infty$. Necessarily, $\sigma(G^N) = \infty$ and $f(\mathcal{A}(G^N)) = f(\emptyset) = f(\mathcal{A}(G))$ for N large enough, showing that f is continuous at G . Our only remaining task is now to show the previous two claims.

Step 3. We show that a.e. realization of \mathcal{H} such that \mathcal{H}_{c_*} is infinite has an active geodesic. There are two trivial cases that we easily exclude: if $c_* = 0$, \mathcal{H}_{c_*} cannot be infinite, and if σ^∞ has bounded support, our result is trivial because the epidemic stops a.s. after a finite time. Now, for any s , by standard properties of Poisson random variables, the graph \mathcal{H}_s is again a Galton–Watson tree with Poisson distributed offspring and the graph \mathcal{H} is obtained by grafting independently on each (S) vertex of \mathcal{H}_s :

- a $\text{Poisson}(S_0 R_0(1 - s))$ distributed number of copies of \mathcal{H} ; and
- a $\text{Poisson}(I_0 \bar{R}_0(1 - s))$ distributed number of (I) vertices.

Furthermore, each of these trees is connected to \mathcal{H}_s through a unique edge whose infection intensity is uniform on the interval $(s, 1)$. Note that when s increases, so does the number of edges in \mathcal{H}_s , therefore we have $\{\mathcal{H}_{c_* - \epsilon} \text{ is infinite}\} \subset \{\mathcal{H}_{c_*} \text{ is infinite}\}$ for each $\epsilon > 0$. Furthermore, by studying the extinction probability of these Galton–Watson trees, we readily see that the probability $\mathbb{P}(\mathcal{H}_s \text{ is infinite})$ is a continuous function of s , which implies that

$$\mathbb{P}\left(\{\mathcal{H}_{c_*} \text{ is infinite}\} \setminus \bigcup_{\epsilon > 0} \{\mathcal{H}_{c_* - \epsilon} \text{ is infinite}\}\right) = 0.$$

In other words, up to a null probability event, we have

$$\bigcup_{\epsilon > 0} \{\mathcal{H}_{c_* - \epsilon} \text{ is infinite}\} = \{\mathcal{H}_{c_*} \text{ is infinite}\},$$

therefore without loss of generality, we can consider a realization of \mathcal{H} and an $\epsilon > 0$ such that $\mathcal{H}_{c_* - \epsilon}$ is infinite. Now let T be such that $|c(t) - c_*| < \epsilon/2$ for $t \geq T$. On the

event that $\mathcal{H}_{c_*-\epsilon}$ is infinite, a.s. we can find a subtree G of \mathcal{H} grafted on $\mathcal{H}_{c_*-\epsilon}$ such that $\sigma(G) > T$ and such that the edge connecting G to $\mathcal{H}_{c_*-\epsilon}$ has an infection intensity in $(c_* - \epsilon, c_* - \epsilon/2)$. (We have used that σ^∞ has unbounded support.) Let us write e_1 for this edge and let $\pi = (e_1, \dots, e_n)$ be the unique path in \mathcal{H} leading to the root and extending the active geodesic in G . Since $\sigma(G) > T$, for each edge e_k of π , we have $|\tau_k(e_k)| > T$, so that $c(|\tau_k(e_k)|) > c_* - \epsilon/2$ and the edge is open. Therefore there exists a.s. an active path in \mathcal{H} if $\mathcal{H}_{c_*-\epsilon}$ is infinite.

Step 4. We now consider a pogm tree G such that G_{c_*} is finite and G has no active path. If $G^N \rightarrow G$, we need to show that for N large enough $\sigma(G^N) = \infty$, that is, that G^N has no active path. Since G_{c_*} is finite, there exists r such that $[G_{c_*}]_r = G_{c_*}$. By definition of G_{c_*} , all edges e in G pointing to a leaf vertex of G_{c_*} have an infection intensity $s_e > c_*$. Since $[G^N]_{r+1} \rightarrow [G]_{r+1}$, the same holds true for G^N for N large enough. If π^N is an active path leading from an (I) vertex to the root in G^N , by Step 2, it has to satisfy $|\pi^N| \rightarrow \infty$. However, any path $\pi^N = (e_1^N, \dots, e_n^N)$ ending at the root in G^N with $|\pi^N| \rightarrow \infty$ includes an edge e_k^N pointing to a leaf vertex of G_{c_*} . On one hand, since $|\pi^N| \rightarrow \infty$ we have $c(|\tau_k \pi^N|) \rightarrow c_*$. On the other hand, since $[G^N]_{r+1} \rightarrow [G]_{r+1}$, we have $\liminf_{N \rightarrow \infty} s_{e_k^N} > c_*$, so that this path is not active for large enough N , proving that there exists no such active paths. \square

Proof of Theorem 7.1. For point (i), we have that for any bounded continuous functional f ,

$$\frac{1}{N} \sum_{x=1}^N f(\mathcal{A}_x^N) \mathbb{1}_{\{\sigma_x^N < t\}} \rightarrow \mathbb{E}[f(\mathcal{A}(\mathcal{T})) \mathbb{1}_{\{\sigma(\mathcal{T}) < t\}}]$$

by using Lemma 4.8 with the mapping $\Phi: G \mapsto f(\mathcal{A}(G)) \mathbb{1}_{\{\sigma(G) < t\}}$ (which is a.e. continuous by Lemma 7.3) and the sequence $(G^N)_N$ (which converges in the local weak sense by Proposition 6.1). This in turn proves that

$$\frac{1}{N} \sum_{x \in [N]} \mathbb{1}_{\{\sigma_x^N < t\}} \delta_{\mathcal{A}_x^N} \rightarrow \mathbb{P}(\sigma(\mathcal{T}) < t) \mathcal{L}(\mathcal{A}(\mathcal{T}) \mid \sigma(\mathcal{T}) < t)$$

in probability for the weak topology (see for instance [29, Theorem 4.11]). The proof of the first point is ended by noting that, with probability I_0 we have $\mathcal{A}(\mathcal{T}) = (-Z, \mathcal{X})$, whereas with probability S_0 we have $\mathcal{T} = \mathcal{H}$.

The proof of point (ii) is the same as for point (i), but replacing the map $G \mapsto f(\mathcal{A}(G)) \mathbb{1}_{\{\sigma(G) < t\}}$ by the map $G \mapsto f(\mathcal{A}(G))$ and using the second part of Lemma 7.3 for the continuity. \square

We can now prove Theorem 1.1 using Theorem 7.1. Recall the notation

$$\mu_t^N = \sum_{x \in [N]} \mathbb{1}_{\{\sigma_x^N \leq t\}} \delta_{(t-\sigma_x^N, X_x(t-\sigma_x^N))}$$

for the empirical distribution of ages and compartments at time t , and the notation

$$Y_t^N(i) = \sum_{x \in [N]} \mathbb{1}_{\{\sigma_x^N \leq t, X_x(t-\sigma_x^N) = i\}} = \mu_t^N([0, \infty), \{i\}) \tag{7.6}$$

for the number of individuals in compartment i at time t . Note that $\mu_t^N = \mu_t^N(da, di)$ can be written in terms of H^N as follows

$$\mu_t^N = N \int \mathbb{1}_{\{0 \leq \sigma_0 \leq t\}} \delta_{(t-\sigma_0, X(t-\sigma_0))} H^N(d\pi), \tag{7.7}$$

where $\pi = (\sigma_\ell, \mathcal{X}_\ell)_{\ell=0}^k = (\sigma_\ell, (\mathcal{P}_\ell, X_\ell))_{\ell=0}^k$ denotes a generic ancestral path.

Proof of Theorem 1.1. By Theorem 7.1 and (7.7), we get for fixed t and $i \in \mathcal{S}$,

$$\mu_t^N(\mathrm{d}a, \{i\}) \longrightarrow S_0\mathbb{P}(t - \sigma^\infty \in \mathrm{d}a)\mathbb{P}(X(a) = i),$$

where σ^∞ is the length of the active geodesic in \mathcal{H} , and X is a life-cycle process. Using Proposition 5.4, we can further identify $S_0\mathbb{P}(t - \sigma^\infty \in \mathrm{d}a) = n(t, a) \mathrm{d}a$, proving finite-dimensional convergence of $(\mu_t^N/N; t \geq 0)$. Because of the expressions of $Y_t^N(i)$ in terms of μ_t^N in (7.6), identification of their limit is trivial. All there is to check is tightness of the processes.

The tightness for $(\mu_t^N/N; t \geq 0)$ will follow from that of $(Y_t^N(i)/N; t \geq 0)$. Recall that the compartments of the life-cycle process enjoy an “acyclic orientation” property. See statement before Theorem 1.1. Writing $i \preceq j$ if j can be accessed from i , the process

$$\sum_{j: i \preceq j} \frac{1}{N} Y_t^N(j), \tag{7.8}$$

is nondecreasing in time. Since the finite-dimensional marginals of this nondecreasing process converge to the expression on the RHS of (1.4), tightness follows provided we can show that this limit is continuous, see for instance Theorem 3.37, Chapter VI of [24]. For the continuity, for $t \geq 0$ and $h > 0$, using Hölder inequality

$$\begin{aligned} & \left| \int_0^\infty n(t, a)p(a, i)\mathrm{d}a - \int_0^\infty n(t+h, a)p(a, i)\mathrm{d}a \right| \\ & \leq \int_0^\infty |n(t, a) - n(t+h, a)|\mathrm{d}a \\ & \leq \int_0^h n(t, a) + n(t+h, a)\mathrm{d}a + \int_0^\infty |n(t, a) - n(t, a+h)|\mathrm{d}a. \end{aligned}$$

The first term can be made small using for instance (3.3), whereas for the second term one can use that translation operators are continuous on L^1 . We proceed in a similar way for $h < 0$. The tightness of $Y_t^N(i)/N$ follows by subtracting the processes in (7.8) in an appropriate way.

Let us turn to the tightness of $(\mu_t^N/N; t \geq 0)$. We will use a tightness criterion for measure-valued processes in [44]. This criterion is stated for measures on a compact space, but can be easily adapted to our setting by considering a compactification of $\mathbb{R}_+ \times \mathcal{S}$ and noting that the limit of our sequence (1.4) has no mass at infinity. According to Lemma 3.2 in [44], it is sufficient to check tightness of the processes $(Y_t^N(\phi, i)/N; t \geq 0)$, for $\phi: \mathbb{R}_+ \rightarrow \mathbb{R}$ uniformly continuous and where

$$\forall t \geq 0, \quad Y_t^N(\phi, i) = \int_0^\infty \phi(a)\mu_t^N(\mathrm{d}a, \{i\}).$$

For $s < t$, we have

$$\begin{aligned} & |Y_s^N(\phi, i) - Y_t^N(\phi, i)| \\ & = \left| \sum_{x \in [N]} \phi(s - \sigma_x) \mathbb{1}_{\{X_x(s - \sigma_x) = i, \sigma_x \geq s\}} - \phi(t - \sigma_x) \mathbb{1}_{\{X_x(t - \sigma_x) = i, \sigma_x \geq t\}} \right| \\ & \leq \sum_{x \in [N]} |\phi(s - \sigma_x) - \phi(t - \sigma_x)| + |Y_s^N(i) - Y_t^N(i)|. \end{aligned}$$

Tightness follows from the uniform continuity of ϕ and from the tightness of the sequence $(Y_t^N(i)/N; t \geq 0)$. \square

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