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Contact Process under renewal cures — An overview of recent results

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Dedicated to the memory of Antonio Galves

Abstract. We review some recent results on the contact process with *renewal cures*, concerning sufficient conditions on the (tail of the) *intercure* distribution for either the occurrence or absence of *persistent survival* of the infection.

Keywords: Contact process; Renewal process; Presence/Absence of persistent survival

2020 Mathematics Subject Classification: 60K35; 60K05; 82B43

1 Introduction

This article gives an account of recent results on the contact process with renewal cures in which the author was directly involved. It corresponds to the topics addressed in his talk on the XXV Brazilian School of Probability held in August, 2022 at the University of Campinas.

The contact process was introduced by Ted Harris in 1974 [9] as a model for the spread of an infection over a connected graph. This is a well

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known, much studied process, in its initial form, as well as variants, along its long history. See [12], [13] and [3] for early expositions.

In the model proposed by Harris, individuals rest on the sites of $\mathbb{Z}^d$, $d \geq 1$, and each one is initially either *healthy* or *infected*. As time passes, the individuals may get healthy, if they are infected; and, while infected, they may infect their nearest neighbors. The mechanism for that goes as follows. Following the renowned *graphical construction* proposed by Harris in [10], let us consider a timeline for each individual. Along each of these timelines we put *cure* marks at random, according to Poisson point processes of rate 1, independent and identically distributed one from the other; an individual who, immediately before such a mark is infected, gets healthy at the mark, and remains so until eventually getting infected again; and a cure mark has no effect on a healthy individual. This is the mechanism for getting healthy.

Infections take place also by putting marks on timelines, but now the timelines are associated with pairs of nearest neighbor individuals. On each of those timelines, we put *infection* marks, also according to Poisson point processes, but of rate λ, independent from pair to pair; as soon as we find a mark at a given time for a pair of neighbors, then, provided one of the individuals involved is infected, and the other is not, the healthy individual becomes infected at that time. The cure and infection Poisson processes are assumed independent, so that almost surely there is no coincidence of cure and infection marks in time, and so the evolution is almost surely well defined. Figures 1.1 to 1.3 provide a diagram with the structure and evolution of the process.

Suppose that we initially have a single infected individual (sitting, say, at the origin of $\mathbb{Z}^d$). A natural question then poses itself: can the initial infection survive forever (with positive probability)? Harris answered in the affirmative in his introductory paper, provided λ is large enough; he also proved that *for positive values of* λ which are close enough to 0, the probability of *survival* is 0 (in other words, *extinction* of the infection is almost sure).

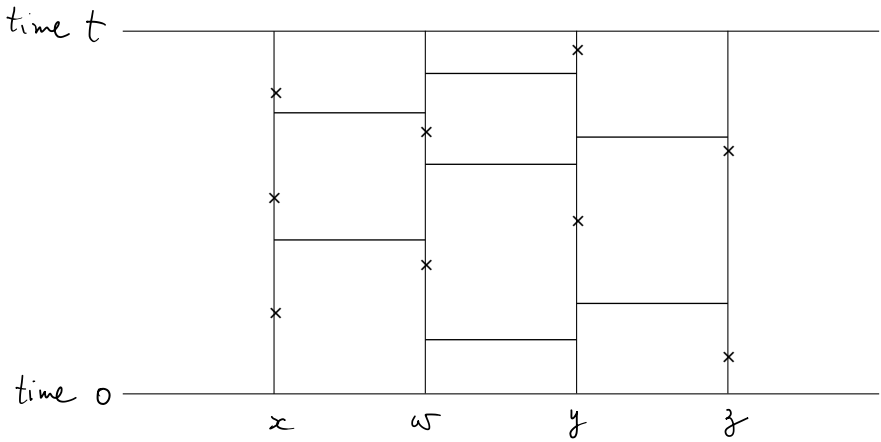

Figure 1.1: Illustration of the timelines of cures and infections; explicit lines appear for sites (x, w, y, z), with crosses $(\times)$ representing cure marks; infection marks (over implicit timelines) are represented by horizontal segments joining timelines of bond endpoints.

We point out that the latter aspect of the Harris contact process may not take place in a setting where the intercure marks are placed according to a generic renewal process, extending the Poissonian picture. In the extended setting, it may conceivably be the case that we have *persistent survival* of the infection, i.e., the probability of survival of the infection (started from a single individual) is positive for *every* $\lambda > 0$. ([14] introduced this terminology in the study of (fully Poissonian) contact processes with random rates; the same phenomenon also comes up in (fully Poissonian) contact processes on scale-free random graphs [2]).

This extension of the original contact process is the object of analysis of the papers on which the present article is based, namely [6], [8], [5] and [7]. It is the results of those papers that we will expound in the present article. Those results provide sufficient conditions for either the presence and, separately, absence of persistent survival in roughly two situations:

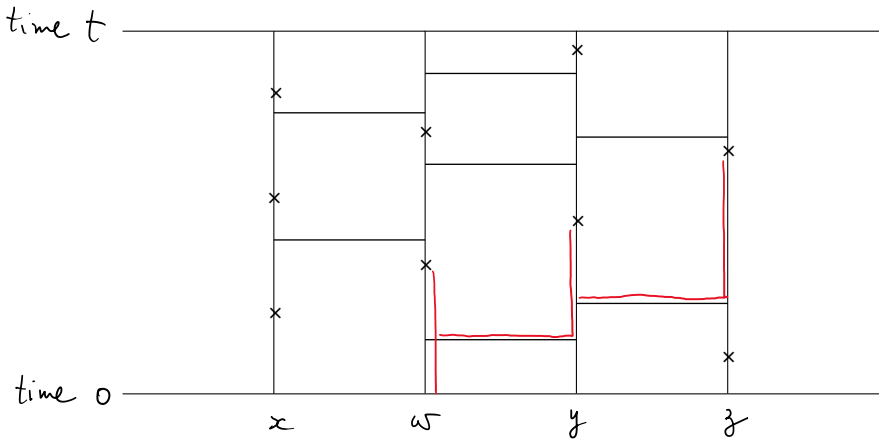

Figure 1.2: Illustration of the evolution of the infection started with a single infected individual (at w); *infection paths* are depicted starting at w at time 0 and moving along timelines and across infection marks, and stopping at cure marks. In this case, the infection (globally) stops before time t.

for infinite, and for finite graphs.[1]

A moment's reflection suggests[2] that a heavy tail of the intercure distribution may play a crucial role. Indeed, assuming heavy tails, as well as some regularity, we first show occurrence of persistent survival in infinite graphs, and next, prove a complete convergence theorem and one other result on the long time behavior of the distribution of the process. This is presented in Section 2 and corresponds to results of [6, 7].

Section 3 is devoted to sufficient conditions for extinction at small positive λ on infinite graphs, translating roughly into a bit more than

[1] [11] considers the model where both cure and infection marks come from renewal processes, neither necessarily Poissonian, and, among other results on this and a related process, finds conditions for the occurrence/absence of what might be called *persistent extinction*, namely, almost sure extinction for every positive value of a *scaling parameter* for the *cure* renewal processes which plays the role of the rate in the Poissonian case.

[2] correctly, as it turns out

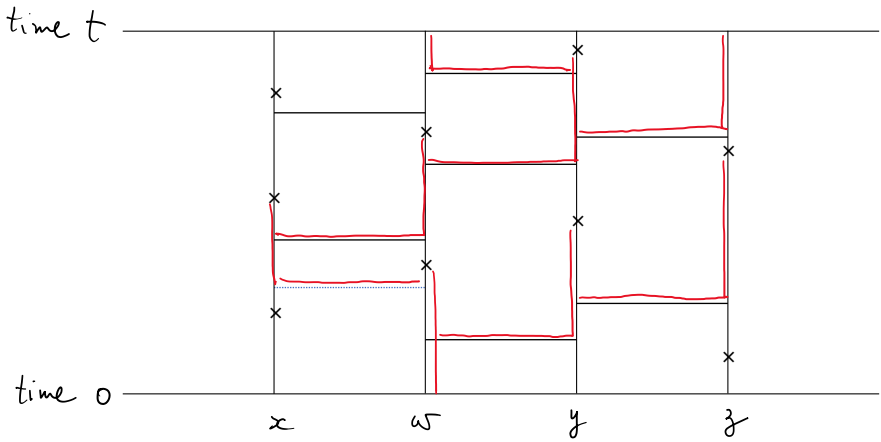

Figure 1.3: In this illustration (with the extra, dotted infection mark with respect to the diagram of Figure 1.2), we find that the infection has survived up to time t.

integrability of the intercure distribution. It refers to a result of [7][3].[4]

Finally, in Section 4, we describe results of [5] for finite graphs, and intercure distributions attracted to a heavy tailed stable law, which provide bounds[5] on the size of the graph, in terms of the index of the stable law, above which persistent survival occurs, and below which it does not.

This text may be seen as an extended, improved, written version of the talk alluded to at the beginning of this introduction. In the next sections, we state precisely the results mentioned above. Arguments for them will be outlined informally, describing ideas and approaches, with few details, often in a simpler, or rough, approximate, even possibly somewhat

[3] which appeared in weaker forms in [8]

[4] Heavy vs. light tails (of the interinfection distribution, in this case) also come up in the results of [11] concerning occurrence vs. absence of persistent extinction; recall Footnote 1, and see Theorem 1.4 in [11].

[5] which turn out to be, perhaps surprisingly, quite sharp

misleading form, and appealing amply to pictures. This will hopefully shed light on actual proofs, and guide the reader to the respective papers.

2 Persistent survival in infinite graphs

Let T denote a random variable distributed as the intercure distribution of the renewal processes underlying our model, and let μ denote its distribution.

We start, on Theorem 2.1, by stating sufficient conditions on the tail of μ for the occurrence of persistent survival, which could be crudely summarized as a heavy tail condition plus some regularity.

Under the same conditions, adding some more regularity on the tail of μ, we establish, in Theorem 2.4, a *complete convergence theorem*, namely the existence, and description, of the limit in distribution of the process (started from an arbitrary configuration of healthy and infected individuals) as time diverges.

Under considerably more strict conditions on the regularity of the tail of μ (asking that it is attracted to a stable law), we derive, in Theorem 2.5, a *closeness to determinism* result, which, for almost every realization of the cure marks of the process, and conditioned on survival of the infection, gives information on the asymptotic distribution of the status of a given individual as time diverges.

In all of these results, the heavy tail of μ plays a central role, by giving rise to larger and larger intercure intervals which provide hubs for the spread of the infection, whatever its intensity. We will see in Section 3 that with (little more than) integrability of T, persistent survival is absent.

The three theorems mentioned above will be stated in the next subsections, one in each.

2.1 Occurrence of persistent survival in infinite graphs

Let us describe the conditions on the distribution of T under which persistent survival can be claimed to hold. They are: there exist $M > 1$

and $\beta < 1$ such that

A) $E(T; T \leq t) \leq t\, P(t < T < Mt)$ for all $t > M$;

B) $P(M^r < T < M^{r+1}) \leq M P(M^{r+1} < T < M^{r+2})$ for all $r > M$;

C′) $P(T > t) > t^{-\beta}$ for all $t > M$.

As anticipated above, we have a heavy tail condition (C′) — notice that it implies that $E(T) = \infty$ —, in addition to regularity tail conditions (A and B) on μ. One may readily check that distributions attracted to stable laws of indices in (0,1) satisfy A-C′).

Theorem 2.1 (Theorem 1 in [6])**.** *If T satisfies A-C′), then persistent survival occurs.*

Indeed, Theorem 1 in [6] is stated and proved under the more stringent condition: there exist $M > 0$ and $\beta \in (1/2, 1)$ such that

C) $t^{-\beta} < P(T > t) < t^{-(1-\beta)}$ for all $t > M$,

replacing C′), but as pointed out in [7], the upper bound in C) may be removed with essentially the same argument — see Remark 3.6 in [7].

Remark 2.2. In [7], we also provide an example of a distribution for T which is attracted to a stable law of index 1, and for which persistent survival takes place — see Section 5 in [7] —, thus extending the domain of μ's for which persistent survival takes place from the conditions of Theorem 2.1.

As anticipated at the introduction, we will not repeat or summarize the proof of the above result, which may be found in [6, 7], but rather will present an informal/partial/rough argument assuming T is attracted to a stable law.

Let us assume

$$P(T > t) = \mu(t, \infty) = L(t)/t^{\alpha}, \tag{2.1}$$

where L is a slowly varying function at infinity, and $\alpha \in (0,1)$. The occurrence of persistent survival may be seen to follow, in this case[6], from the combination of a sequence of events, which constitute what we may call a *tunnelling event*, which has positive probability for every $\lambda > 0$, and on which we find an infinite infection path from the origin to infinity, implying the result.

With the help of Figure 2.1, we sequentially describe the constituent events of the tunnelling event. Without loss, we may take our (infinite) graph to be $\mathbb{N}$.

Definition 2.3. Let $L_0 = 0$ and for $n \geq 1$, $L_n = \inf\{x > L_{n-1} : Z_{2^{n-1}}(x) > 2^n\}$, where for $x \in \mathbb{N}$ and $t > 0$, $Z_t(x)$ denotes the residual waiting time after time t, i.e., the time elapsed between t and the next cure mark in the timeline of x; let us also define $Y_t(x)$, the age at/spent waiting time till time t, i.e., the time elapsed prior to t since the last cure mark before t in the timeline of x.

From the well known theory of distributions attracted to stable laws (satisfying (2.1)) — see, e.g., [4] —, we have that $Z_{2^n}(x)$ and $Y_{2^n}(x)$ are both of order 2^n. It follows that

1. the increments of L_n are random variables *of order 1* (i.e., their distributions depend weakly on n, or converge as $n \to \infty$ to a continuous distribution); and that

2. for every $\lambda > 0$ fixed, we may find, with probability *exponentially close to 1* as n diverges, an increasing staircase pattern of infection bonds from L_n to L_{n+1}, as shown in red in Figure 2.1, effectively carrying the infection from the intercure interval of L_n containing 2^{n-1}, if it is infected, to that of L_{n+1} containing 2^n.

Let us consider the event illustrated in this picture. In the intersection of those events over n, which thus has a positive probability, there is

[6] but the general case is treated quite similarly, at least in a broad sense

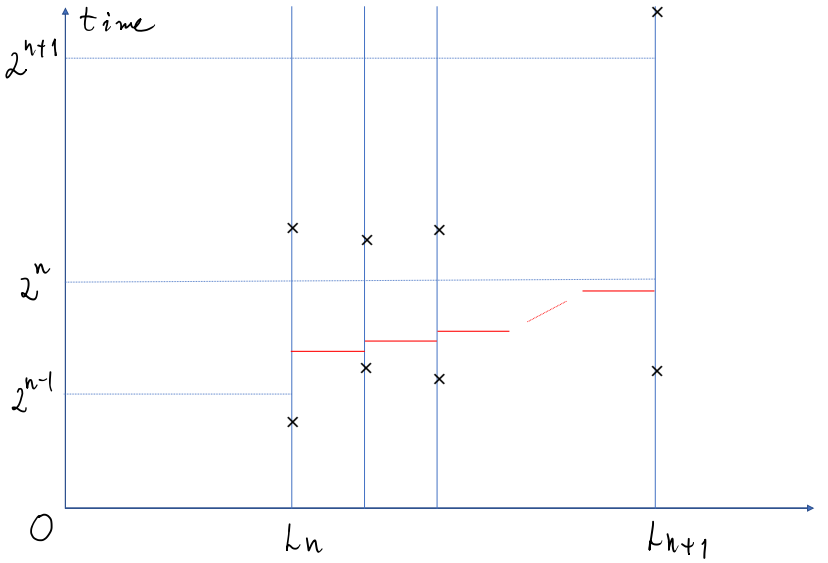

Figure 2.1: Patterns of cure marks (crosses) and staircase of infection bonds (in red) propagating the infection from L_n to L_{n+1} during the time period $[2^{n-1}, 2^n]$.

an infection path connecting the origin to infinity, and the argument for persistent survival is complete.

2.2 Complete convergence theorem

Let ξ_t denote the configuration of healthy and infected individuals of our process at time $t \geq 0$, where for $x \in \mathbb{Z}^d$

$$\xi_t(x) = \begin{cases} 0, & \text{if the individual at } x \text{ is healthy;} \\ 1, & \text{if the individual at } x \text{ is infected.} \end{cases} \tag{2.2}$$

Theorem 2.4 (Theorem 1.2 in [7]). *Suppose μ satisfies conditions A,B,C. Then, given any initial condition ξ_0, we have that ξ_t converges in distribution, as $t \to \infty$, to*

$$P(\tau < \infty)\delta_{\underline{0}} + P(\tau = \infty)\delta_{\underline{1}}, \tag{2.3}$$

where $\tau = \inf\{t > 0 : \xi_t \equiv 0\}$ *is the extinction time of the infection;* $\delta_{\underline{0}}$ *and* $\delta_{\underline{1}}$ *represent the Dirac measure on the configuration with all sites healthy and all sites infected, respectively.*

Let us remark that in the above result, we are not making any assumptions on ξ_0, and the mentioned distribution, as well as P, are conditional ones (on ξ_0).

We again, as with Theorem 2.1, explain the ideas for the proof of Theorem 2.4 in the simpler setting of T attracted to a stable law, i.e., satisfying (2.1). We will be perhaps even a bit rougher than before, and rely on a variation of Figure 2.1, in the belief that things will be quite clear.

We only need to figure out what goes on with the process when $\tau < \infty$, and, separately, when $\tau = \infty$. In the former case, by definition, $\xi_t \equiv 0$ for all t large enough (more precisely, if $t \geq \tau$).

We recall from the argument laid out above for the occurrence of persistent survival that the probability of the event depicted in Figure 2.1 occurring for all $n \geq n_0 \geq 0$ may be taken arbitrarily close to 1 by taking n_0 large enough — that follows from the above mentioned exponentiality.

It should be intuitively clear that in the event where $\tau = \infty$, we find that the interval $\{L_{n+1}\} \times [2^n, 2^{n+1}]$ is infected with probability close to 1 when n is large.

It is also the case that for $t \in [2^n, 2^{n+1}]$, since $Y_t(x) \approx t \approx 2^n$ and $L_{n+1} \approx n$ (recall the meaning of $Y_\cdot(\cdot)$ in Definition 2.3 above), we find that the event illustrated in Figure 2.2 is highly probable as $n \sim \infty$.

It follows from the above points that in the event where $\tau = \infty$, (by taking the intersection of the relevant events) we have that the origin, as well as its neighbors, are all infected at time t with high probability, provided t is large. This concludes the idea for Theorem 2.4.

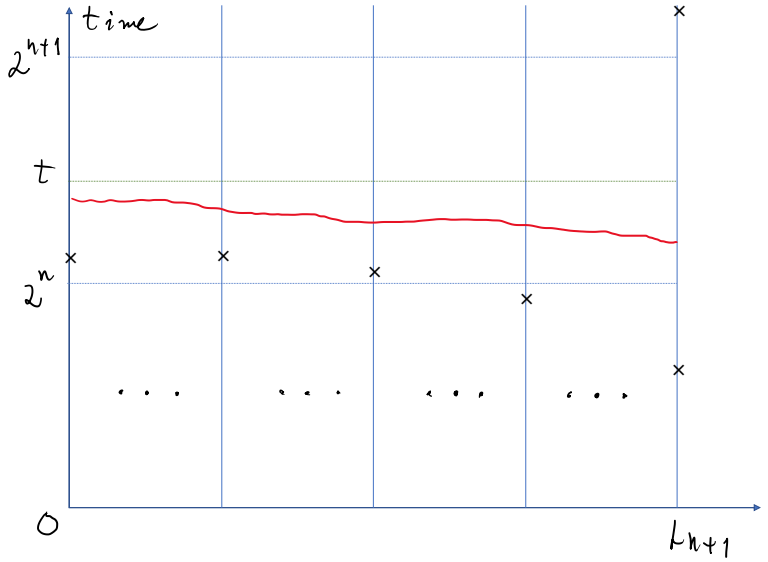

Figure 2.2: Illustration of an event where there exists an infection path from $\{L_{n+1}\} \times (t - m_n, t)$ to $\{0\} \times (t - m_n, t)$ (increasing from right to left, represented in red), where $m_n = m_n(t) = \min_{0 \le x \le L_{n+1}} Y_t(x)$.

2.3 Closeness to determinism

The picture drawn above allows for going further in understanding the long time behavior, when there occurs survival of the infection, of the distribution of the status of any fixed individual.[7] For the results of this subsection, we take our graph to be $\mathbb{Z}^d$, and we require also T to be attracted to a stable law. We may indeed obtain results for almost every realization of the cure marks.

Let $\mathcal{G}$ denote the σ-field generated by the renewal processes and the extinction random time τ.[8]

Theorem 2.5 (Theorem 4.1 in [7]). *If T is attracted to an α-stable law,*

[7] When the infection dies out, this is, of course, a straightforward issue.

[8] $\mathcal{G}$ includes *all* the information on the renewal processes; information on the infection marks and initial condition are also present in $\mathcal{G}$, but *only* as encoded in τ.

$0 < \alpha < 1$, *then for all* $x \in \mathbb{Z}^d$*:*

1. *If* $\alpha < 1/2$ *and further regularity conditions hold on the tail of* μ*, we have on* $\{\tau = \infty\}$ *that*

$$\lim_{t\to\infty} \left| P(\xi_t(x) = 0 \mid \mathcal{G}) - e^{-2d\lambda Y_t(x)} \right| = 0 \ \ a.s.;$$

2. *If* $\alpha > 1/2$ *and* $F(t) > 0 \ \forall \ t > 0$*, we have on* $\{\tau = \infty\}$ *that*

$$\overline{\lim_{t\to\infty}} \left| P(\xi_t(x) = 0 \mid \mathcal{G}) - e^{-2d\lambda Y_t(x)} \right| > 0 \ \ a.s.,$$

where $Y_t(x)$ *is the age of the renewal process in* x *at time* t*.*

Remark 2.6.

1. The regularity conditions alluded to in the first item of the above statement refer to those required for the validity of the Strong Renewal Theorem, which are quite technical, so we do not spell them out here — see Theorem 1.4 in [1].

2. In Theorem 4.4 of [7] we give more precise behavior of $P(\xi_t(x) = 0 \mid \mathcal{G})$ for large t as a function of $Y_t(x)$ when $\alpha > 1/2$ — see Remark 2.7 below.

Here is again a rough account of the idea for the above result, illustrated in Figure 2.3. Let us assume that $\tau = \infty$ and that t is large. If $Y_t(x)$ is large, then the (large) interval $\big(t - Y_t(x), t\big)$ will be infected (with high probability), and the result follows in this case; if instead $Y_t(x)$ is of order 1, then under the conditions of item (i) of Theorem 2.5, with high probability all the $2d$ neighbors of x will have large values of $Y_t(\cdot)$; it readily follows that in order for x to not be infected at time t, there must be no infection mark on the time interval $\big(t - Y_t(x), t\big)$ in any timeline of the pair of x and one of such neighbors; the result readily follows in case (i).

Now, when $\alpha > 1/2$, and under the other condition in (ii), then we are able to find (exceptional) arbitrarily large t for which $Y_t(x)$ is of order 1

and the timeline of a neighbor y of x shows a pattern of many cure marks close together, spanning a time interval I containing $\big(t - Y_t(x), t\big)$ such that $I \cap (t, \infty)$ is long, in such a way that infection marks on the timeline of the pair (x, y), if any, with high probability occur between cure marks of y spanning a time interval where y is always healthy (provided x is healthy at the infection mark in question) and thus that infection mark will not transmit the infection from y to x. The upshot is that, with high probability, in order to check that x is healthy at time t, we only need to look at infection marks between x and its other neighbors (but y).

Remark 2.7.

As anticipated in the second item of Remark 2.6 above, we may be more precise in the above paragraph. Indeed we may argue that for $k = 1, \dots, d$, there is a collection of d (explicit) disjoint ordered open subintervals of $(1/2, 1)$, $I_1, \dots, I_d$, whose closures cover $(1/2, 1)$, such that for $\alpha \in I_k$, we have that

$$\overline{\lim_{t\to\infty}} \Big(P(\xi_t(x) = 0 \mid \mathcal{G}) - e^{-(2d-k)\lambda Y_t(x)} \Big) = 0.$$

The reasoning for this is a refinement of the one in the above paragraph, where we are able to say that we may find (at arbitrarily large times) *exactly* k neighbors of x with the close packed pattern of cures around the time interval $\big(t - Y_t(x), t\big)$.

3 Extinction in infinite graphs

In this section we present conditions for the absence of persistent survival of the contact process with renewal cures on $\mathbb{Z}^d$, i.e., such that we may find $\lambda > 0$ for which the infection started at the origin dies out almost surely. As one may surmise from the previous discussion and results, we need a moment condition; indeed, a first moment might be thought to be enough, but we require a bit more.

Theorem 3.1 (Theorem 1.1 in [7]). *If* $E(Te^{\theta\sqrt{\log T}}) < \infty$ *for some* $\theta > 4\sqrt{d \log 2}$, *then* $P(\textit{survival}) = 0$ *for* $\lambda > 0$ *close enough to* 0.

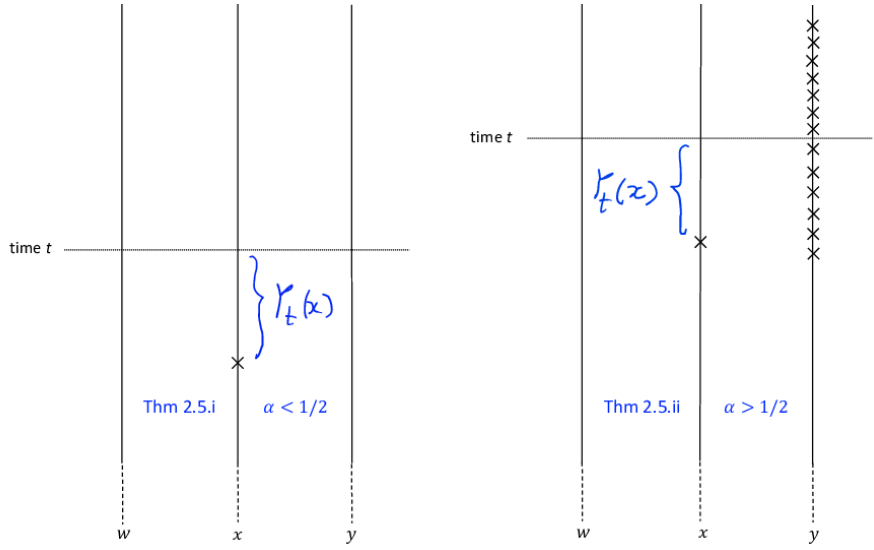

Figure 2.3: Depiction of events behind Theorem 2.5; on the left, case (i); case (ii) on the right.

This result supersedes earlier results with the same conclusion appearing in [8], under the more restrictive conditions: either

1. $E(T^2) < \infty$ (Theorem 1 in [8]), or
2. $E(T^\alpha) < \infty$ for some $\alpha > 1$ *and* $d = 1$ *and* T has a continuous distribution such that $f(t)/(1 - F(t))$ is decreasing in $t > 0$, where f and F are, respectively, the density and distribution functions of T (Theorem 2 in [8]).

Condition (1) allows for a *supermartingale argument* or *comparison to a branching process* where the second moment of T implies a uniform bound on the first moment of the length of intervals between cures arrived at by infection marks/bonds, in a growth construction of the intercure intervals infected by the origin.

Condition (2) allows for a multiscale argument involving crossings of rectangles by infection paths. Besides the $\alpha > 1$ moment, whose improvement in Theorem 3.1 is quite slim, the other conditions were put in place

to ensure a positive association property for the crossing events, which then may be combined in a way that requires $d = 1$. The refinement of the latter aspect in Theorem 3.1, essentially preserving the same approach, dispenses, by a subtle change in the definition of the crossing events, the above mentioned positive association and $d = 1$ requirements. We next roughly and pictorially explain the ideas of our argument for the proof of Theorem 3.1. As anticipated, the argument is based on a multiscale analysis of crossing of rectangle events. We will consider rectangles of several scales, for the definiteness of which, in terms of the present exposition, we would need a slightly stronger moment condition on T, namely that $E(Te^{\theta \frac{\log T}{\log \log T}}) < \infty$ for some large θ. The ideas are visually easier to explain, and perhaps to more readily grasp, in the one dimensional case, but it should be quite clear that it works for all d.

Let n_0 be a large fixed integer, to be more precisely chosen later. For $n \geq n_0$, let $\mathcal{R}_n = \mathcal{B}_n \times \mathcal{H}_n$ be the two dimensional rectangle with base $\mathcal{B}_n$ at the x-axis centered at the origin and of length 2^n and height $2^{n \log n}$. We start by relating the probability of survival of the infection started at the origin in the contact process to crossings of such rectangles by infection paths, as follows.

Let A_n denote the event that there exists either an infection path inside $\mathcal{R}_n$ starting anywhere at $\mathcal{B}_n \times \{0\}$ and ending at $\mathcal{B}_n \times \{2^{n \log n - 1}\}$ — this may be seen as a *time half-crossing* of $\mathcal{R}_n$ — or an infection path inside $\mathcal{R}_n$ starting anywhere on the left hand side of $\mathcal{R}_n$ and ending on the right hand side of $\mathcal{R}_n$, or the other way around — this may be seen as a *space* (full) *crossing* of $\mathcal{R}_n$. See Figure 3.1.

Let $P_n = \sup P(A_n)$, with the sup taken over renewal starting points before time 0.

With τ denoting, as in the Section 2, the extinction time of the process started with a single infected individual at the origin, we have that if $\tau > 2^{n \log n}$, then there must be an infection path inside $\mathcal{R}_n$ starting at the origin and exiting $\mathcal{R}_n$ either at the top, or at the left hand side, or at the right hand side. Let us denote the event where such a path exists by S_n;

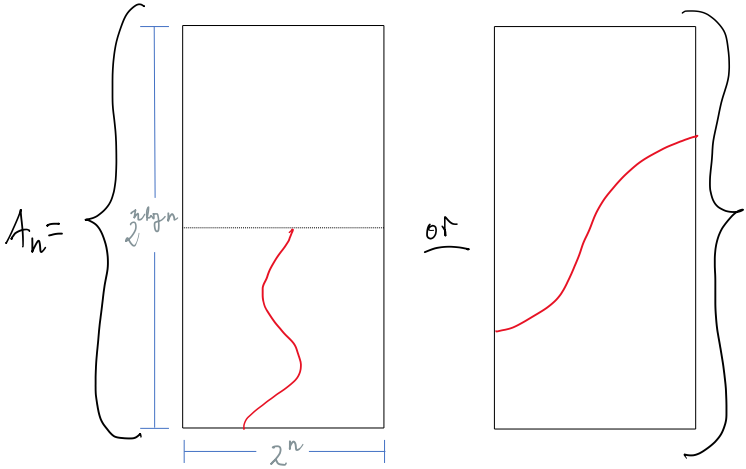

Figure 3.1: Illustration of the event A_n.

see Figure 3.2. It follows that

$$P(\tau > 2^{n \log n}) \leq P(S_n). \tag{3.1}$$

Since one of the instances of S_n is a time (full) crossing of $\mathcal{R}_n$, and by the symmetry of the model, we have that

$$P(S_n) \leq P_n + 2P(S'_n), \tag{3.2}$$

where S'_n is the event depicted in Figure 3.3, corresponding to there being an infection path from the origin exiting $\mathcal{R}_n$ from the right hand side. We next relate the latter probability to P_{n-1}.

In S'_n, we quite clearly have a space crossing of the rectangle $\mathcal{R}'_n$ which has the same height and half the base of $\mathcal{R}_n$. In order to find a relationship with crossings of (a rectangle congruent to) $\mathcal{R}_{n-1}$ leading to a relationship of $P(S_n)$ (and thus $P(\tau > 2^{n \log n})$) to P_n, we partition $\mathcal{R}'_n$ into rectangles congruent to $\mathcal{R}_{n-1}$, as indicated in Figure 3.4. We then note that $S'_n \subset S''_n$, where S''_n is the event where there is either a space crossing or a time half crossing of one of the at most n^κ, with $\kappa < 1$ (as can be readily checked,

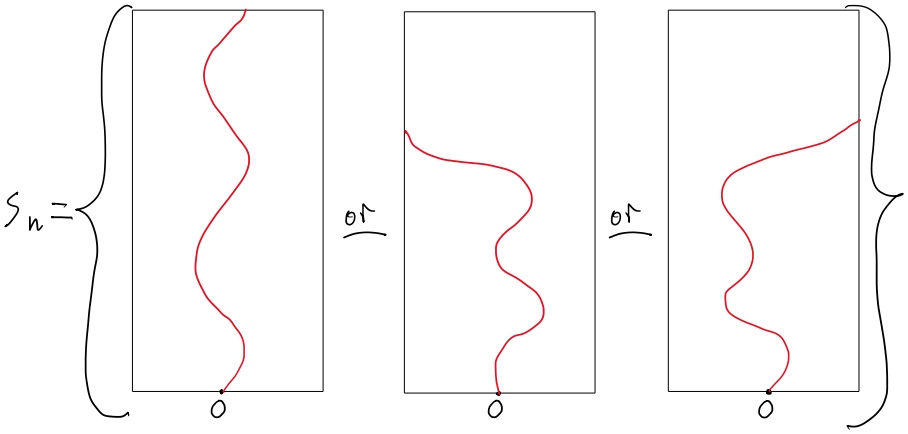

Figure 3.2: Illustration of the event S_n, where the infection path starting at the origin is represented in red.

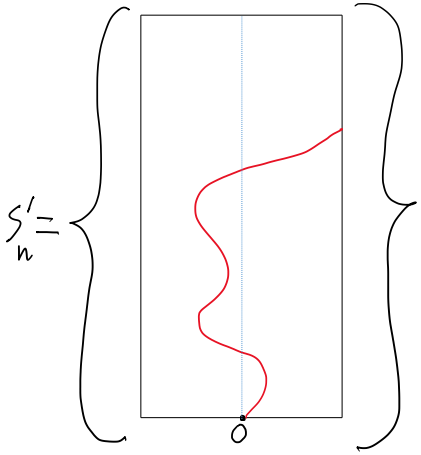

Figure 3.3: Illustration of event S'_n

if we additionally take n_0 large enough), subrectangles resulting from the partition, as respectively illustrated in Figure 3.4 (in three instances for the latter case).

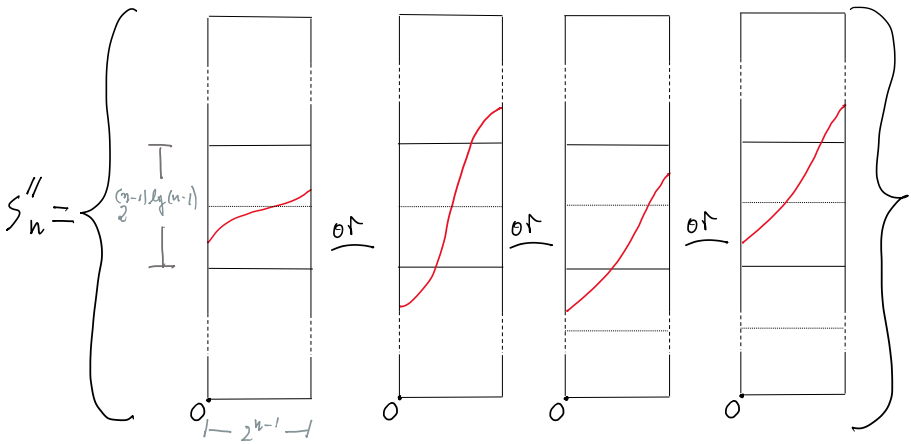

Figure 3.4: Illustration of event S''_n

It follows from that, (3.1) and (3.2) (again taking n_0 large enough) that

$$P(\tau > 2^{n \log n}) \leq P_n + nP_{n-1}, \tag{3.3}$$

and thus, making $Q_n = nP_n$, it is enough to establish the following result.

Proposition 3.2. *If $\lambda > 0$ is close enough to 0, then*

$$Q_n \to 0 \text{ as } n \to \infty. \tag{3.4}$$

In order to argue Proposition 3.2, we set up a 2-step recursion, relating P_n (and thus Q_n) to P_{n-1} and P_{n-2} (and thus Q_n to Q_{n-1} and Q_{n-2}), the iteration of which plus the use of $\lambda > 0$ small after the last step yields (3.4).

The recursion step will be based on considering the two kinds of crossings entering the event A_n, the time kind, and the space kind. We spell out the first kind, leaving the second kind, which is similar, if simpler, aside.

Let us concentrate on the event A'_n contained in A_n that there exists a time half crossing of $\mathcal{R}_n$ (corresponding to the left hand side rectangle in Figure 3.1). In order to relate A'_n to crossing events corresponding to

A_{n-1} and A_{n-2}, we proceed, similarly as above, to partition $\mathcal{R}_n$ into eight rectangles, denoted $\mathcal{R}_n^1$ to $\mathcal{R}_n^8$, with the same base $\mathcal{B}_n$ (and thus height $2^{n\log n-3}$). Then, each of the 4 bottom subrectangles of the partition must exhibit a time full crossing. Let us focus on $\mathcal{R}_n^1$, and the event C_n^1 where it is (fully) time crossed.

The next step is to decompose C_n^1 as indicated in Figure 3.5.

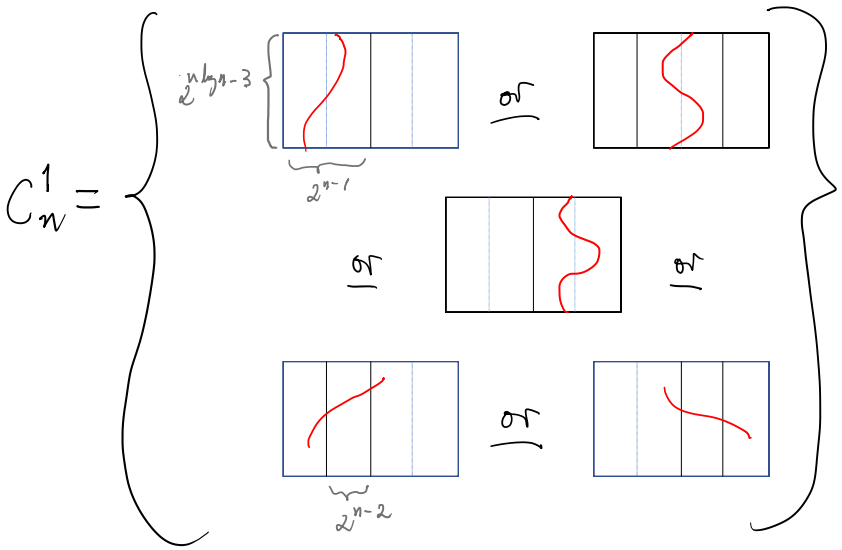

Figure 3.5: Illustration of the decomposition of C_n^1 into 5 distinct crossing events

We note that in this decomposition we find 3 subevents where there is a full time crossing of a fixed rectangle congruent to $\mathcal{R}_{n-1}$ (corresponding to the two top rectangles and the middle rectangle in Figure 3.5), as well as 2 subevents where there is a *spatial crossing* of a fixed $2^{n-2} \times 2^{n\log n-3}$ rectangle (corresponding to the two bottom rectangles in Figure 3.5).

The next step is to estimate the sup of the probability of the latter events, with the sup taken, as before, over renewal starting points before the time corresponding to the bottom of the rectangle. Let us consider one of the 2 latter events only. (The other 4 events may be similarly

treated.) We may reason similarly as in the argument to get (3.3) above (by partitioning the respective rectangle into at most $n^{2\kappa}$, $\kappa < 1$, subrectangles with base 2^{n-2} and height $2^{(n-2)\log(n-2)}$), to find the upper bound $n^{2\kappa}P_{n-2}$ for the number of such subrectangles. We thus have that

$$\sup P(C_n^1) \leq 3P_{n-1} + 2n^{2\kappa}P_{n-2}, \tag{3.5}$$

and the same bound clearly holds also for $\sup P(C_n^3)$, with C_n^3 defined as C_n^1, except that we replace $\mathcal{R}_n^1$ by $\mathcal{R}_n^3$.

Up to now, we have not used the moment condition of Theorem 3.1. We will use it in the following estimation. Let G_n denote the event that there exists a timeline segment in $\mathcal{R}_n^2$ with no cure mark. Our moment condition may be checked to imply that

$$\sup P(G_n) \leq e^{-cn} \tag{3.6}$$

for all large n, where c is a positive constant.

Now, relying on the independence properties of the renewal processes giving the cure marks in our model, which roughly speaking gives a decoupling of C_n^3 from C_n^1 on G_n^c, we readily get that

$$\sup P(A_n') \leq e^{-cn} + (3P_{n-1} + 2n^{2\kappa}P_{n-2})^2 \leq e^{-cn} + n^{2\kappa}(Q_{n-1} \vee Q_{n-2})^2. \tag{3.7}$$

With a similar, if simpler, reasoning, we find a similar bound for the probability of the event A_n'' where there is a space crossing of $\mathcal{R}_n$ (the simplicity comes from the fact that in the spatial direction we have extra independence; the term of $\sup P(G_n)$ is absent in this case).

The bounds

$$P_n \leq e^{-cn} + n^2(Q_{n-1} \vee Q_{n-2})^2 \tag{3.8}$$

and thus

$$Q_n \leq ne^{-cn} + n^3(Q_{n-1} \vee Q_{n-2})^2 \tag{3.9}$$

follow.

Setting up a recursion for Q_n, stopped at the appropriate n_0, and taking $\lambda > 0$ small enough, depending on n_0, we finally find that

$$Q_n \leq e^{-c'n} \tag{3.10}$$

for all large n, with c' a positive constant; (3.4) follows.

Remark 3.3. The subtlety, alluded to at the beginning of this section, that allowed the improvement of the multiscale analysis of [8], refers to considering half time crossings in the definition of A_n. The argument in [7], while essentially preserving the approach of [8], dispenses with the need to, roughly speaking, obtain full time crossings from half time crossings — which appear inevitably in the decomposition illustrated in Figure 3.4 —, a need which in turn, on the one hand, originates the demand for a positive association property of crossing events in our model, allowing for the application of the Harris-FKG inequality, and, on the other hand, restricts application to one spatial dimension.

Remark 3.4. The result of this section and the one of Subsection 2.1 raise the issue of a sharp condition on the distribution of T for persistent survival. A natural conjecture is that $E(T) = \infty$ is such a condition.

One related question, suggested by our conditions for Theorem 3.1, and the example alluded to in Remark 2.2, is whether the case where $P(T > t) \sim 1/t$ shows persistent survival or not.

These questions seem to be beyond the reach of our current methods of analysis of this model.

(Notice that if $P(T > t) \sim L(t)/t$ with $L(t) = e^{-\theta\sqrt{\log t}}$ for some $\theta > 4\sqrt{d \log 2}$, then T satisfies Theorem 3.1. On the other hand, the example referred to in Remark 2.2 has the same form, with $L(t) = \text{const}\, e^{\frac{\log t}{\log\log t}}$ — see Section 5 of [7]. Both L's are slowly varying at ∞, but while the first one decays quite quickly to 0 as $t \to \infty$, the second one increases to ∞ correspondingly.)

4 Finite graphs

We now look at finite (connected) graphs, and the occurrence or not of (persistent) survival under heavy tails. We do not expect survival to take place in these cases without heavy tails, even at large λ, at least not if we have some regularity on the tail of the distribution of T.

We will indeed consider in this section only the case of μ attracted to an α-stable law, $0 < \alpha < 1$. Our results give thresholds for the size of the graph above which we have persistent survival, and below which the process dies out for any $\lambda > 0$.

Let V be the vertex set of our finite, connected graph, and let $k = |V|$ denote the cardinality of V.

Theorem 4.1 (Theorem 2.3 in [5]). *Suppose that T is attracted to an α-stable law, $\frac{1}{2} < \alpha < 1$. For all $\lambda > 0$*

1. $P(\textit{survival}) = 0$, *if* $k < v^- := 2 + \frac{2\alpha-1}{(1-\alpha)(2-\alpha)}$;
2. $P(\textit{survival}) > 0$, *if* $k > v^+ := \frac{1}{1-\alpha}$.

We first note that if $\alpha \leq 1/2$, then (1) is trivially satisfied; and that (2) also holds under additional regularity conditions (allowing for the validity of the Strong Renewal Theorem, which is required for our argument in all cases — see first item of Remark 2.6 above and Remark 2.1.3 in [5]).

Secondly, it may be readily checked that $v^+ - v^- < 1$ for all $\alpha \in (\frac{1}{2}, 1)$; thus, if $[v^-, v^+] \cap \mathbb{N} = \emptyset$, then the above criteria determine the situation for all V; otherwise, the situation is undetermined for exactly one value of k.

Remarkably, we have that the geometry of the graph plays no role in this result, and neither does the particular positive value of λ.[9] One might say that above v^+ the model behaves as in infinite volume, and below v^-, as if it were light tailed.

This result rules out the possibility of survival (for any $\lambda > 0$) in finite graph for T's attracted to a 1-stable distribution, even when $E(T) = \infty$.

[9] But either could be relevant in undetermined cases.

(Recall from Remark 2.2 that in this case, we may have persistent survival in an infinite graph.)

We will next, in separate subsections, argue each case of the theorem, again in broad and rough strokes (perhaps even more so than in the previous sections).

4.1 Extinction

We will give an approximate description of the structure of the argument for extinction, incomplete but hopefully shedding light on the main mechanism we took advantage of, while attempting to broadly, if imprecisely, discuss some of the left out points.

For illustration sake, let us henceforth focus on the graph with vertex set $V = \{1, 2, \dots, k\}$ and nearest neighbor edges.

For $x \in V$, let $T_{x,i}$, $i = 1, 2, \dots$ denote the succesive intercure times at the timeline of x. Let $X_1 = \max\{T_{x,1} : x \in V\}$, $\mathcal{X}_1 = \text{argmax}\{T_{x,1} : x \in V\}$, and for $n \geq 1$ let $X_{n+1} = \max\big\{Z_{S_n}(x) : x \in V \setminus \{\mathcal{X}_n\}\big\}$, $\mathcal{X}_{n+1} = \text{argmax}\big\{Z_{S_n}(x) : x \in V \setminus \{\mathcal{X}_n\}\big\}$, where $Z_\cdot(\cdot)$ is the residual time defined in Definition 2.3, and $S_n = X_1 + \cdots + X_n$. See Figure 4.1.

The result follows readily once we show that X_n a.s. does not diverge as $n \to \infty$: this implies that we will infinitely often see time intervals spanned by X_n with lengths of order 1, one of which will be sooner or later a.s. be free of infection bonds, which will then mean extinction of the infection at the end of that interval (or sooner).

To argue that property, we assume that X_{n+1} is bounded in distribution by X_n times the maximum, say Z, of $Y_1, \dots, Y_{k-1}$, a random vector with i.i.d. coordinates with common density $\text{const}/(t^\alpha(1+t))$, $t > 0$.

(This bound makes sense for large values of the ages $Y_{S_n}(\cdot)$ — see Definition 2.3 — by well known results on the behavior of residual times of distributions attracted to a stable law of index < 1, to the effect that $Y_t(\cdot)/t \approx Z$ in distribution when $t \sim \infty$ — see e.g. [4]. In the actual argument in Section 3 of [5] we make adjustments in the above definitions, introducing a threshold t^* above which we may use an approximate version

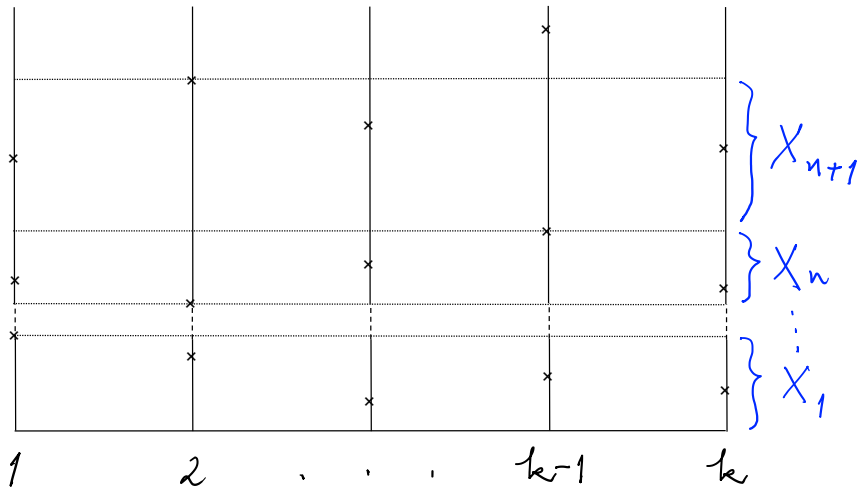

Figure 4.1: Illustration of the construction of X_n, $n \geq 1$, where $\mathcal{X}_1 = 1$, $\mathcal{X}_{n-1} = 2$, $\mathcal{X}_n = k - 1$, $\mathcal{X}_{n+1} = 2$

of that fact.)

It would follow (from this rough argumentation), that we might bound the distribution of X_n by $\prod_{i=1}^{n} Z_i$, where $Z_1, Z_2, \ldots$ are i.i.d. copies of Z. It may be shown now that under the conditions of (1) in Theorem 4.1, we have that $E(\log Z) < 0$ — see Proposition 5.1 in [5]. It follows that X_n cannot diverge.

(Notice that the domination proposed in the above paragraph implies that X_n vanishes in probability as $n \to \infty$. The above product indeed makes an appearance in the actual proof of the result in [5], even if a less direct one, and its asymptotic behavior is crucial in the development of that argument, yielding a non divergent behavior of (the actual version of) X_n, even if we do not show that it vanishes — the adoption of the above

mentioned threshold t^* precludes that.)[10]

4.2 Survival

The argument for survival is not dissimilar to the one for Theorem 2.1 above. We again describe the main ideas for it, broadly and roughly.

Let n_0 be a large fixed integer and for $n \geq n_0$, set $b_n = \gamma \log n$ and let

$$t_n = \sum_{j=n_0}^{n} (j^\epsilon - b_j^{m+1}) \sim n^{1+\epsilon} \text{ as } n \sim \infty,$$

where γ and m are large (integer) constants (to be chosen more precisely later) and $\epsilon > 0$ is small.

The main steps of the argument are as follows.

1. Showing that a.s. for all large n, at least one $x \in V$ whose timeline has no cure marks in $\big(t_n, t_n + (n+1)^\epsilon\big)$;

2. then, a.s. for all large n, we see the (potential) transmission of the infection within $\big(t_{n+1}, t_n + (n+1)^\epsilon\big)$;

3. finally, the infection survives initially with positive probability.

See Figure 4.2 for an illustration of the above objects (where time runs in the horizontal direction).

To show the first item, let D_n denote the event $\{\max_{x \in V} Z_{t_n}(x) > n^\epsilon\}$. Then

$$P\big(D_n^c\big) \approx \Big\{P\Big(\frac{Z_{t_n}(1)}{t_n} \leq n^{-1}\Big)\Big\}^k \approx \Big(\int_0^{n^{-1}} x^{-\alpha}\, dx\Big)^k \approx n^{-k(1-\alpha)}, \quad (4.1)$$

[10] The approximate argument outlined above can be made exact in the model where we replace the renewal cure processes by their scaling limit, namely α-stable subordinators (keeping the Poissonian infection mechanism intact); we would need to change the condition of an infected individual at the origin at time 0, by the same condition at time 1 — otherwise, the infection would be immediately extinguished —, and have $X_1 = 1$ instead.

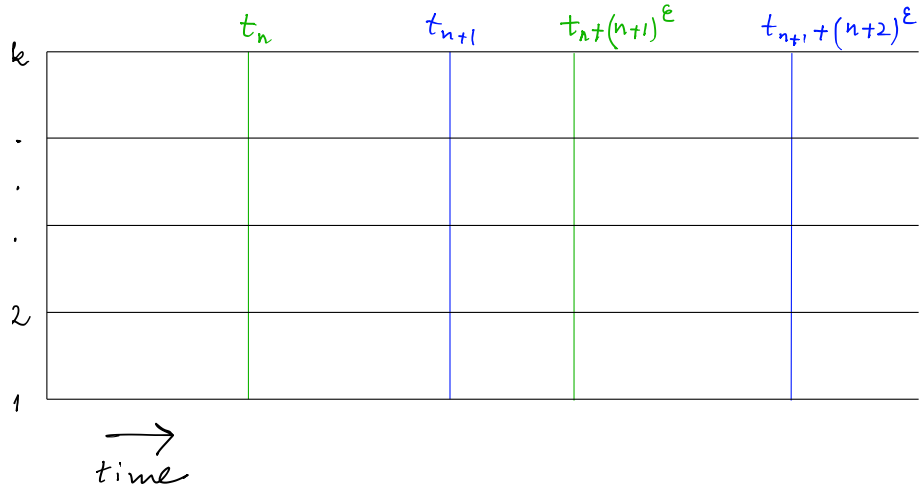

Figure 4.2: Timelines of $V = \{1, \dots, k\}$

as $n \sim \infty$, where the second $\approx$ sign comes from the well known asymptotics of the distribution of $Z_t(1)/t$ as $t \sim \infty$ for interrenewal distributions attracted to a stable law of index in $(0, 1)$.

Arguing more rigorously, in Proposition 4.2 of [5], we get that $P\big(D_n^c\big) \le n^{-\beta}$, with $\beta = k(1 - \alpha - 3\epsilon) > 1$, provided ϵ is small, given the condition on k in item 2 of Theorem 4.1. Borel-Cantelli now implies the first item of the argument.

For the second item, for $n \ge n_0$ and $j = 0, \dots, b_n^m - 1$, let $E_{n,j}$ denote the event where there is an 'up and down' pattern of infection bonds in $V \times \big(t_{n+1} + jb_n, t_{n+1} + (j+1)b_n\big)$, as illustrated in Figure 4.3.

We may readily check that for each j

$$P\big(E_{n,j}^c\big) \le 1 - \big(1 - e^{-\frac{\lambda}{\ell}\gamma \log n}\big)^\ell = 1 - \Big(1 - \frac{1}{n^\rho}\Big)^\ell \sim \frac{\ell}{n^\rho}, \tag{4.2}$$

where $\ell = 2k - 1$, and $\rho = \frac{\lambda}{\ell}\gamma > 1$, provided γ is large. It follows that

$$P\big(\cup_{j=0}^{b_n^m - 1} E_{n,j}^c\big) \le \frac{1}{n^{\rho'}}, \tag{4.3}$$

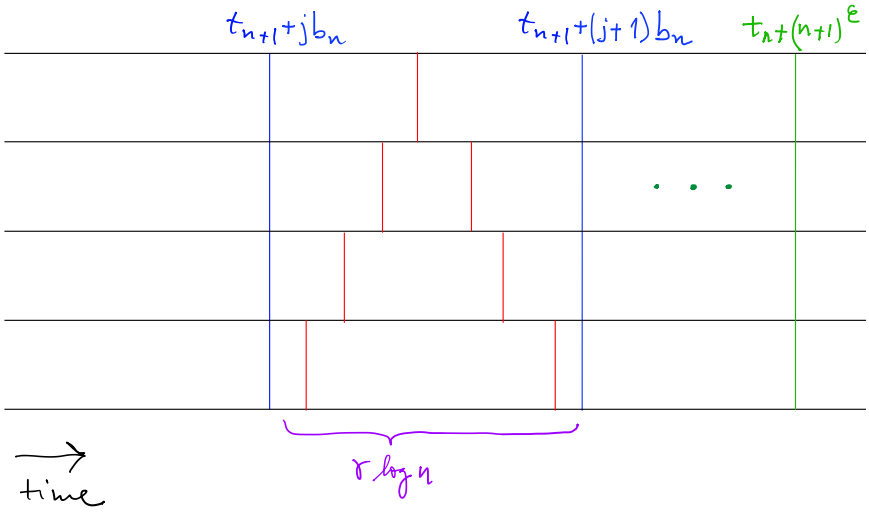

Figure 4.3: Illustration of event $E_{n,j}$, with infection bonds represented in red. (Time runs in the horizontal direction.)

with $\rho' > 1$. Borel-Cantelli now implies that $E_n := \cap_{j=0}^{b_n^m - 1} E_{n,j}$ occurs for every large enough n almost surely.

In order to conclude the argument for the second item, let us now consider the event $C_{n,j}^x$ where the timeline of x is void of cure marks during $(t_{n+1}+jb_n, t_{n+1}+(j+1)b_n)$, and set $C_{n,j} = \cap_{x\in V} C_{n,j}^x$ and $C_n = \cup_{j=0}^{b_n^m - 1} C_{n,j}$.

We claim that C_n occurs for every large enough n almost surely. This will be seen to follow from the following fairly straightforward (conditional) bound on the tail of the distribution of $Z_t(x)$. Let $t_0 > 0$ be given, and let $\mathcal{F}_{t_0}^x$ be the σ-algebra generated by the renewal process at x up time t_0. Then for all t large enough

$$P\big(Z_{t_0}(x) > t \big| \mathcal{F}_{t_0}^x\big) \geq \frac{1}{t^{\alpha+\epsilon}} \tag{4.4}$$

— see Lemma 4.2 in [5]. It follows that

$$\begin{aligned} P\big(C_n^c\big) &= \Big(1 - P\big(\cap_{x\in V}\,\{Z_{t_n}(x) > b_n\}\big)\Big) \\ &\quad \times \prod_{j=1}^{b_n^m-1} \Big(1 - P\big(\cap_{x\in V}\,\{Z_{t_n+jb_n}(x) > b_n\}\big|\,\cap_{i=0}^{j-1} C_{n,i}^c\big)\Big) \\ &= \Big(1 - \Big[P\Big(Z_{t_n}(1) > b_n\}\Big)\Big]^k\Big) \\ &\quad \times \prod_{j=1}^{b_n^m-1} \Big(1 - \Big[P\Big(Z_{t_n+jb_n}(1) > b_n\}\big|\,\cap_{i=0}^{j-1} (C_{n,i}^1)^c\Big)\Big]^k\Big) \\ &\le \Big(1 - \frac{1}{b_n^{k(\alpha+\epsilon)}}\Big)^{b_n^m} \le e^{-b_n^{m-k(\alpha+\epsilon)}} \le e^{-b_n} \le \frac{1}{n^\gamma}, \end{aligned}$$

where the first inequality follows from (4.4), and the third one holds as soon as $m > k\alpha + 1$ and $\epsilon > 0$ is small enough. Borel-Cantelli thus implies the claim, and the second item follows from this and the above argued similar occurrence of E_n.

The third item is quite clear (depending only on initial configurations, which can be arranged with positive probability to transmit the infection along any fixed finite time interval). Combining this with items 1 and 2, we get survival.

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This article is dedicated to the memory of Antonio Galves, who first taught the author about the contact process.

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