

Geometric Decay of Infection Probabilities for the Anisotropic Contact Process

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Abstract

Consider the *anisotropic* symmetric contact process on a homogeneous tree \mathcal{T}_{2d} of degree $2d \geq 2$ with a single initially infected site at the root vertex of the tree. We show that, for all values of the infection vector λ , each integer $n \geq 1$, and each vertex $x \in \mathcal{T}_{2d}$ at distance n from the root vertex, the probability $\mathbf{P}(x \text{ is ever infected}) = u_x(\lambda)$ satisfies $u_x(\lambda) \leq [\beta_c(\lambda)]^{n-1}$ for some function β_c that we will specify. This geometric decay property governs the growth and dispersal behaviour of the process and lies at the core of the method of Hueter (1998), which applies the thermodynamic formalism and the theory of Gibbs states (Bowen, 1975) to the contact process on trees. We leave open the question as to when (if at all) λ_c is the maximal infection rate among the components of λ .

1 Introduction

The contact process, introduced by T.E. Harris (1978) on \mathbf{Z}^d in the isotropic framework, serves to model epidemics, tumor growth, and competition among a number of applications. This interacting particle model is an example for a stochastic growth process which, along with branching random walks and percolation processes in spaces with hyperbolic geometries, exhibits two qualitatively different survival phases and gives rise to interesting phenomena at the phase transition between those two regimes, global survival and local survival. The anisotropic contact process on an infinite homogeneous tree, which was introduced by the author, is studied in detail near that discontinuous phase transition elsewhere (Hueter, 1998). That analysis imposes a certain symmetry on the parameters of the process. In this note, we will investigate the geometric decay of certain infection probability functions of the symmetric anisotropic process. This decay property governs the growth and dispersal behaviour of the contact process and lies at the core of the approach of Hueter (1998), which applies the thermodynamic formalism and the theory of Gibbs states (Bowen, 1975) to the contact process on trees. Whereas it is a prime feature of the isotropic model

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as well, it is more easily proved in that special case. Before stating and proving our main result in Section 2, to keep the exposition self-contained, we will introduce the background needed on the anisotropic contact process in the remainder of Section 1.

1.1. Anisotropic Contact Process. Anisotropy in the parameter of the process takes into account heterogeneities of the terrain on which an infection lives or a competition takes place. We consider a “symmetric” anisotropic contact process on an infinite homogeneous tree $\mathcal{T}_{2d} = \mathcal{T}$ of degree $2d$. Each vertex x of \mathcal{T} has exactly $2d - 1$ children. The tree \mathcal{T} is *homogeneous* in that for any two vertices x and y there is an isometry that maps x to y . Associated with each of the $2d$ emanating edges is a nonnegative infection rate λ_i . An *anisotropic contact process* on the tree \mathcal{T} is a continuous time Markov process A_t on the set of finite subsets of (the vertex set of) \mathcal{T} that evolves in the following way. Infected sites (members of A_t) recover at rate 1 and upon recovery are removed from A_t . Healthy sites (members of A_t^c) become infected at a rate that equals the sum of the infection rates attached to the edges leading to infected nearest neighbours and upon infection are added to A_t . Under the default probability measure \mathbf{P} , the initial state A_0 is the singleton set $\{1\}$, where 1 is a distinguished element of \mathcal{T} , called the *root* or *root vertex* of the tree. The rates mentioned belong to two independent sets of independent Poisson variables, one set of which is attached to the vertices and the other set of which is attached to the edges of \mathcal{T} , as explained below. The contact process can be shown to enjoy the strong Markov property (Section 1.3). The symmetry assumption that we will further describe (in Section 1.2) guarantees that the same infection rate be used when the infection traverses the edge in either of the two directions. Specifically, the symmetry assumption assures that each infection rate be used twice for the set of edges emanating from each vertex. If the infection rates are all equal, then the contact process is called *isotropic*.

1.2. The Tree and d Generators. From the perspective of an anisotropic contact process, different vertices of the underlying homogeneous tree at distance n from the root look different. Therefore, it is useful to describe and identify each vertex of the tree by a finite address, a finite word from a finite alphabet. Even though to label the $2d$ edges the alphabet needs $2d$ letters in general, in the symmetric setting at hand, d letters will suffice. Thus, let $\mathcal{A}_+ = \{a_1, a_2, \dots, a_d\}$ be a set of d letters, let $\mathcal{A}_- = \{a_1^{-1}, a_2^{-1}, \dots, a_d^{-1}\}$ be the set of formal inverses of the letters in \mathcal{A}_+ , and set $\mathcal{A} = \mathcal{A}_+ \cup \mathcal{A}_-$. The free group \mathcal{G} with generators \mathcal{A}_+ is the set of finite reduced words from the alphabet \mathcal{A} (a word is *reduced* if no letter $a \in \mathcal{A}$ is adjacent to its inverse), where multiplication is concatenation followed by reduction and the group identity 1 is the empty word. There is a natural bijection between \mathcal{G} and the set of vertices of \mathcal{T} , in which $g, h \in \mathcal{G}$ are mapped to adjacent vertices of \mathcal{T} if and only if $gh^{-1} \in \mathcal{A}$. In other words, vertices are uniquely represented by finite reduced words from \mathcal{A} . In the subsequent discussion, we shall not be careful to distinguish between vertices of \mathcal{T} and the words (or group elements) representing them, and we shall refer to \mathcal{G} as the vertex set of \mathcal{T} . For any vertex z , denote by $|z|$ the length of its representative word. Note that $|z|$ as well is the distance from vertex z to vertex 1 in the graph \mathcal{T} . For every integer $n \geq 0$, let \mathcal{G}_n denote the set of all vertices $z \in \mathcal{G}$ at distance n from the root vertex

(i.e. $|z| = n$). For any vertex z of \mathcal{T} , define $\mathcal{T}(z)$ to be the set of vertices v such that the unique word representing z is a prefix of the word representing v .

Now, if $\lambda_{a_1}, \dots, \lambda_{a_d}, \lambda_{a_1^{-1}}, \dots, \lambda_{a_d^{-1}}$ denotes the set of infection rates, then we say that the anisotropic contact process is *symmetric* when $\lambda_{j^{-1}} = \lambda_j$ for each $j \in \mathcal{A}_+$.

1.3. Percolation Structure and Strong Markov Property. The contact process may be constructed via the usual *percolation structure* (Harris, 1978) on $\mathcal{T} \times (0, \infty)$, that is, as a system of independent Poisson processes attached to vertices and ordered pairs of neighbouring vertices. For each vertex $x \in \mathcal{T}$, the Poisson process attached to x has rate 1 and determines the recovery times. Specifically, at every occurrence time, site x recovers if it is infected. For each ordered pair (x, xi) , $i \in \mathcal{A}$, of neighbouring vertices, the Poisson process attached to (x, xi) has rate λ_i , the occurrence times being precisely those times when an infection at x may jump to xi . Occurrences in these Poisson processes are marked on a system of directed rays $\{x\} \times [0, \infty)$ connected to the vertices x of \mathcal{T} so that (A) at each occurrence time t of the Poisson process attached to (x, xi) an *infection arrow* is drawn from (x, t) to (xi, t) and (B) at each occurrence time t of the Poisson process attached to x a *recovery mark* $*$ is attached to (x, t) . There are no simultaneous occurrences of infection arrows and/or recovery marks in the percolation structure. At time t , the contact process now consists of all those vertices y for which there is a (directed) path through the percolation structure, the system of rays and arrows just described, that begins at the root vertex 1 at time 0, ends at (y, t) , and does not pass through any recovery marks $*$.

The contact process exhibits the strong Markov property (see Lalley and Sellke, 1998, Section 2.2). Let G, F_1, F_2, \dots, F_k be pairwise nonoverlapping parts of the tree \mathcal{T} and let S_1, S_2, \dots, S_k be stopping times determined by the percolation structure over G . Then conditional on the percolation structure over G , the post- S_i portions of the percolation structure over the sets F_i are independent, and for each i , the post- S_i percolation structure over F_i has the same distribution as the entire percolation structure over F_i .

2 Infection Probabilities: Geometric Decay

We next define the infection probability functions to get ready for the main result. Let λ refer to the infection vector $\{\lambda_j\}_{j \in \mathcal{A}_+}$. For any vertex $x \in \mathcal{T}$ and every λ , define the probability that x is ever infected,

$$u_x = u_x(\lambda) = \mathbf{P}(x \in A_t \text{ for some } t > 0). \quad (2.1)$$

The strong Markov property together with the monotonicity properties of the contact process and the homogeneity of the process at each vertex implies that

$$u_{xy}(\lambda) \geq u_x(\lambda)u_y(\lambda) \quad (2.2)$$

for each λ and each $x, y \in \mathcal{T}$ such that $|xy| = |x| + |y|$ (no reduction occurs when x and y are concatenated). In view of (2.2), a supermultiplicativity argument shows that, for every

$a \in \mathcal{A}$ and each vertex $x = aa \dots a \in \mathcal{G}_n$, the limit

$$\lim_{|x|=n \rightarrow \infty} u_x(\lambda)^{1/n} = \beta_a = \beta_a(\lambda) \quad (2.3)$$

exists and that $u_x(\lambda) \leq \beta_a(\lambda)^n$ for all $n \geq 0$. Moreover, for every integer k and $x \in \mathcal{G}_k$, by the same means, for each *periodic* sequence $y_n = xx \dots x \in \mathcal{G}_{nk}$, the limit

$$\lim_{n \rightarrow \infty} u_{y_n}(\lambda)^{1/n} = \beta_x(\lambda) \quad (2.4)$$

exists for every λ and $u_{y_n}(\lambda) \leq \beta_x(\lambda)^n$ for every $n \geq 0$. Note that the functions $\beta_x(\cdot)$ are nondecreasing in each infection parameter λ_j . Monotonicity and continuity properties of $\beta_x(\lambda)$ and other functions in λ are discussed in Hueter (1998). We remark that, in the isotropic setting, $u_x(\lambda)$ depends only on the distance of x from the root and that there is just one function $\beta(\lambda)$ with the inequality $u_{|x|}(\lambda) \leq \beta(\lambda)^{|x|}$ being an immediate consequence of (2.2). When assuming anisotropic rates, however, proving a result that mimics similar decay behaviour for the u_x is more subtle but no less valuable in gaining insight into the nature of the process. An example of a corollary is that the expected number of vertices of \mathcal{T} at fixed distance n that are ever to be infected either decays or increases like an exponential function with a power linear in n except when the process is right at the threshold between the two regimes. Those two regimes coincide with the extinction and survival phases of the contact process (for details on this jargon and more consequences of the decay feature, consult Hueter, 1998). Some difficulties arise since, even though periodic words z give rise to limits $u_z^{1/|z|}$ as $|z| \rightarrow \infty$, in general, those limits $u_z^{1/|z|}$ as $|z| \rightarrow \infty$ do not exist. Nevertheless, the entire collection $\{u_x\}_{x \in \mathcal{T}}$ jointly converges in a sense made precise in Hueter (1998). The reason, contained in the subsequent result, is the geometric decay of the $u_x(\lambda)$ on a region of the λ where there exists $\rho < 1$ such that $\max_{a \in \mathcal{A}} \beta_a(\lambda) \leq \rho$.

LEMMA 1 (Geometric Decay of u_x) *Assume that the anisotropic contact process is symmetric. For every λ , each integer $n \geq 2$, and every $x \in \mathcal{G}_n$, we have*

$$u_x(\lambda) \leq [\max_{a \in \mathcal{A}} \beta_a(\lambda)]^{n-1}$$

and $u_x(\lambda) \leq \max_{a \in \mathcal{A}} \beta_a(\lambda)$ for every $x \in \mathcal{G}_1$.

Proof. The proof proceeds by induction over the distance of the vertices of the tree from the root, the single initially infected vertex. There are two driving factors that determine how fast the infection propagates from one vertex to one in the next higher generation and ultimately determines whether a path is more likely than another to ever be infected. Apart from the infection rate λ_i , the probability that the infection ever moves from vertex x (with $|x| = m$) to its child xi (with $|xi| = m + 1$) is influenced, first, by the intensity of infections that spread between the parent xj^{-1} and x and, second, by the intensity of infections between the children xk (with $|xk| = m + 1$), $k \neq i$, and x . The set $O^x = \{t \geq 0 : x \in A_t\}$ of random times during which x is ever infected captures both effects. For every vertex x ,

the set O^x is the union of the collection of intervals O_k^x for $k \geq 1$ (associated with each of the infection events at x), the lengths of each of which are independently and identically distributed (since the recovery rate associated with each vertex equals 1). Observe that a vertex xi with $|xi| = |x| + 1$ ever gets infected if and only if there exists an infection arrow between x and xi at some time $s \in O^x$. In constructing a path in the tree which the infection favours in the sense that the probability that its endpoint is ever infected is maximal among all self-avoiding paths of the same length, we will maximize the duration $\sum_{k \geq 1} |O_k^x|$ of infection for the vertices along a path in a suitable way (see (2.6) below).

We further illustrate the difficulty in finding the favourite path of the infection. Recall that \mathcal{G}_n denotes the set of vertices at distance n from the root. Fix λ . For $x \in \mathcal{T}$, let E_x denote the event that $x \in A_t$ for some $t > 0$, in other words, the event that x is ever infected, and let E_x^c denote its complement, the event that x is never infected. Thus, $\mathbf{P}(E_x) = u_x(\lambda)$. Since λ will be kept fixed throughout the proof, we shall shorter write u_x for $u_x(\lambda)$. Suppose that $b \in \mathcal{G}_1$ satisfies $\mathbf{P}(E_b) = \max_{i \in \mathcal{G}_1} \mathbf{P}(E_i)$. Then it is possible to imagine that, for some $c \neq b^{-1} \in \mathcal{G}_1$,

$$\mathbf{P}(E_{bb}) = \mathbf{P}(E_{bb}|E_b)P(E_b) < \mathbf{P}(E_{cb}) = \mathbf{P}(E_{cb}|E_c)\mathbf{P}(E_c)$$

happens for bb and cb both in \mathcal{G}_2 . For instance, if vertex b is infected less often (in a certain sense), given it is ever infected, than vertex $c \in \mathcal{G}_1$, given c is ever infected, then bb may not have the largest probability to ever be infected among all vertices in \mathcal{G}_2 . It would follow that $\mathbf{P}(E_{bb}|E_b) < \mathbf{P}(E_{cb}|E_c)$. This inequality would be a consequence of the intensity of reinfections of c , given O^c is nonempty, being larger with high probability than the intensity of reinfections of b , given O^b is nonempty, because $\mathbf{P}(E_{cb}|E_c)$ depends only on the distribution of O^c and the infection rate λ_b .

After this passage of motivation, we shall present a candidate for a favourite path of the infection and continue to establish that it indeed is a favourite. Let b and c denote letters in \mathcal{G}_1 that satisfy

$$\mathbf{P}(E_b) = \max_{i \in \mathcal{G}_1} \mathbf{P}(E_i) \tag{2.5}$$

$$\max_{\mathcal{A} \ni i \neq c^{-1}} \mathbf{P}(E_{ci}) = \max_{k \in \mathcal{G}_1} \max_{\mathcal{A} \ni j \neq k^{-1}} \mathbf{P}(E_{kj}). \tag{2.6}$$

Observe that here ci and kj both are in \mathcal{G}_2 . When can c be taken to be b ? We leave this question open, see our comment after the proof.

We note that b and c are not necessarily unique and that there are at most two such letters $b \in \mathcal{G}_1$ and two such letters $c \in \mathcal{G}_1$ because \mathcal{G}_1 can contain b and b^{-1} and c and c^{-1} (The symmetry assumption imposed on the infection rates says that $\lambda_b = \lambda_{b^{-1}}$ and $\lambda_c = \lambda_{c^{-1}}$). In view of the definition of the vector λ , for every n and $x \in \mathcal{G}_n$, we have $xi \in \mathcal{G}_{n+1}$ for at least one letter $i = b \in \mathcal{A}$ and at least one letter $i = c \in \mathcal{A}$ as defined in (2.5) and in (2.6), respectively. More precisely, consider the case that the last letter of x is $b \in \mathcal{A}_+$, then xb^{-1} is in $\mathcal{G}_{|x|-1}$ and xb is in $\mathcal{G}_{|x|+1}$. In contrast, if the last letter of x is $b^{-1} \in \mathcal{A}_-$, then xb^{-1} is in $\mathcal{G}_{|x|+1}$ and xb is in $\mathcal{G}_{|x|-1}$. If the last letter of x is neither b nor

b^{-1} , then both xb and xb^{-1} are in $\mathcal{G}_{|x|+1}$ (no reduction occurs when b or b^{-1} is concatenated to the word x). For the rest of the proof, we will not distinguish between b and b^{-1} , equivalently, whether b is in \mathcal{A}_+ or \mathcal{A}_- and just write b . Similarly, we will refer to c , when we mean either c or c^{-1} .

In order for any vertex $xk \in \mathcal{G}_{|x|+1}$ to ever be infected, it is necessary that xk is infected for a first time. Since the single initially infected vertex is the root vertex, the first infection at xk must arrive from x . We say that xk gets *infected via x* at time $t > 0$, if there is an infection arrow drawn from (x, t) to (xk, t) . Thus, xk must be infected via x at the time of its first infection since the infection can move to xk only if it moves through x . Each vertex $i \in \mathcal{G}_1$ can get infected for the first time only when 1 (the root) is infected. Since, in light of elementary properties of the Poisson distribution, the probability that i gets infected via the root in the interval O_j^1 , $j \geq 1$, is largest for the letter $i \in \mathcal{G}_1$ associated with the largest infection rate $\max_{d \in \mathcal{A}} \lambda_d$, regardless of whether the infection attempt will be a success or not (yet it will be a success at its first attempt), the letter b in (2.5) satisfies $\lambda_b = \max_{d \in \mathcal{A}} \lambda_d$. The same reasoning establishes that the lefthand side of (2.6) equals $\mathbf{P}(E_{cb})$. In fact, if we apply the same arguments to x in place of the root and replace O_j^1 by O_j^x , we obtain

$$\mathbf{P}(E_{xb}|E_x) \geq \mathbf{P}(E_{xk}|E_x) \quad (2.7)$$

for every $x \in \mathcal{T}$, every $xk \in \mathcal{G}_{|x|+1}$, and $xb \in \mathcal{G}_{|x|+1}$. As a consequence,

$$\begin{aligned} u_{xb} = \mathbf{P}(E_{xb}) &= \mathbf{P}(E_{xb}|E_x)\mathbf{P}(E_x) \\ &\geq \mathbf{P}(E_{xk}|E_x)\mathbf{P}(E_x) \\ &= u_{xk}. \end{aligned} \quad (2.8)$$

Moreover, we remark that the property in (2.6) is independent of the number of vertices in \mathcal{G}_2 that take on the maximum value on the lefthand side of (2.6). In other words, the choice of labeling in \mathcal{G}_1 does not affect the choice of c . Combining the observation that $\mathbf{P}(E_{xkj}|E_x)$ depends only on the distribution of O^x and the infection rates λ_k and λ_j with the defining properties in (2.5) and (2.6) leads to

$$\mathbf{P}(E_{xcb}|E_x) \geq \mathbf{P}(E_{xkj}|E_x) \quad (2.9)$$

for each $x \in \mathcal{T}$ and xcb, xkj in $\mathcal{G}_{|x|+2}$.

We now turn to the induction part of the proof. Fix n . Write $z_n = cc \dots c \in \mathcal{G}_n$ for the word of length n that consists of n letters c and write $y_n = z_{n-1}b \in \mathcal{G}_n$ for the concatenation of z_{n-1} and b for $n \geq 1$. Let $y_1 = b$. To complete the proof, we will need to show that

$$u_{y_n} \geq u_x \quad (2.10)$$

for each $x \in \mathcal{G}_n$. For $n = 1$, the defining property for b in (2.5) verifies (2.10). For $n = 2$, the defining property for c in (2.6) guarantees (2.10). Next, assume that for every $1 \leq m \leq n-1$, inequality (2.10) is valid, thus, $u_{y_m} \geq u_x$ for each $x \in \mathcal{G}_m$. We shall compare the infection

probabilities associated with the sequence y_n and any $x = x_1x_2 \dots x_n \in \mathcal{G}_n$. To do so, we will distinguish between the two cases $x_1x_2 \dots x_{n-1} = y_{n-1}$ and $x_1x_2 \dots x_{n-1} \neq y_{n-1}$. First, consider the case when $x_1x_2 \dots x_{n-1} = y_{n-1}$. In particular, $x_{n-1} = b$ and the two words y_n and $x_1x_2 \dots x_{n-1}b$ differ exactly in their second last letters only. Relying on (2.9) and then on (2.8), we collect

$$\begin{aligned}
\mathbf{P}(E_{y_n}) &= \mathbf{P}(E_{y_n}|E_{z_{n-2}})\mathbf{P}(E_{z_{n-2}}) \\
&\geq \mathbf{P}(E_{x_1x_2 \dots x_{n-1}b}|E_{z_{n-2}})\mathbf{P}(E_{z_{n-2}}) \\
&\geq \mathbf{P}(E_{x_1x_2 \dots x_{n-1}x_n}|E_{z_{n-2}})\mathbf{P}(E_{z_{n-2}}) \\
&= \mathbf{P}(E_x|E_{z_{n-2}})\mathbf{P}(E_{z_{n-2}}) \\
&= \mathbf{P}(E_x),
\end{aligned} \tag{2.11}$$

which is as announced in (2.10). Second, we look at the case when $x_1x_2 \dots x_{n-1} \neq y_{n-1}$. Since by virtue of (2.9),

$$\mathbf{P}(E_{x_1x_2 \dots x_{n-2}cb}) \geq \mathbf{P}(E_{x_1x_2 \dots x_{n-2}x_{n-1}x_n}) = \mathbf{P}(E_x),$$

it suffices to prove that (2.10) holds for those x that have $x_{n-1}x_n = cb$. By our induction assumption,

$$\mathbf{P}(E_{z_{n-2}b}) = \mathbf{P}(E_{y_{n-1}}) \geq \mathbf{P}(E_{x_1x_2 \dots x_{n-2}b}) \tag{2.12}$$

for $x_1x_2 \dots x_{n-2}b \in \mathcal{G}_{n-1}$.

As explained earlier, $\mathbf{P}(E_{xi}|E_x)$ depends only on the distribution of O^x and the infection rate λ_i . Of course, $\mathbf{P}(E_x)$ is uniquely determined by the distribution of O^x as well. Therefore, $\mathbf{P}(E_{xi})$ depends only on the distribution of O^x and the infection rate λ_i . In particular, $\mathbf{P}(E_{x_1x_2 \dots x_{n-2}i})$ for $x_1x_2 \dots x_{n-2}i \in \mathcal{G}_{n-1}$ depends only on the distribution of $O^{x_1x_2 \dots x_{n-2}}$ and the infection rate λ_i while $\mathbf{P}(E_{z_{n-2}i})$ for $z_{n-2}i \in \mathcal{G}_{n-1}$ depends only on the distribution of $O^{z_{n-2}}$ and the infection rate λ_i . Since, if we fix $i = c$, thus, the infection rate λ_i for both events and regard $E_{z_{n-2}c} = E_{z_{n-1}}$ and $E_{x_1x_2 \dots x_{n-2}c} = E_{x_1x_2 \dots x_{n-1}}$, we conclude from the validity of the inequality in (2.12) (where the subscripts of the two events end in the letter b instead of c),

$$\mathbf{P}(E_{z_{n-1}}) = \mathbf{P}(E_{z_{n-2}c}) \geq \mathbf{P}(E_{x_1x_2 \dots x_{n-2}c}) = \mathbf{P}(E_{x_1x_2 \dots x_{n-1}}). \tag{2.13}$$

Notice that the words y_n and x both end in the same letter b . Consequently, $\mathbf{P}(E_{y_n}|E_{z_{n-1}})$ and $\mathbf{P}(E_x|E_{x_1x_2 \dots x_{n-1}})$ only depend on $O^{z_{n-1}}$ and $O^{x_1x_2 \dots x_{n-1}}$, respectively. Recall that, in view of the homogeneity of the tree, any two vertices have the same neighbourhood. The assumption $x_{n-1} = c$ implies that the subtree $\mathcal{T}(z_{n-1})$ is isomorphic to the subtree $\mathcal{T}(x_1x_2 \dots x_{n-1})$. The only possibility for a difference between the distributions of $O^{z_{n-1}}$ and $O^{x_1x_2 \dots x_{n-1}}$ to arise is when z_{n-1} and $x_1x_2 \dots x_{n-1}$ get infected and reinfected via their parent z_{n-2} and $x_1x_2 \dots x_{n-2}$, respectively. Yet, the inequality in (2.13) implies that

$\sum_{k \geq 1} |O_k^{z_{n-1}}|$ stochastically dominates $\sum_{k \geq 1} |O_k^{x_1 x_2 \dots x_{n-1}}|$. From this observation, it follows that

$$\mathbf{P}(E_{y_n} | E_{z_{n-1}}) \geq \mathbf{P}(E_x | E_{x_1 x_2 \dots x_{n-1}}). \quad (2.14)$$

Combining (2.13) together with (2.14) leads to

$$\begin{aligned} \mathbf{P}(E_{y_n}) &= \mathbf{P}(E_{y_n} | E_{z_{n-1}}) \mathbf{P}(E_{z_{n-1}}) \\ &\geq \mathbf{P}(E_x | E_{x_1 x_2 \dots x_{n-1}}) \mathbf{P}(E_{x_1 x_2 \dots x_{n-1}}) = \mathbf{P}(E_x), \end{aligned}$$

which finishes the proof of (2.10) as well as the induction.

Since we have seen earlier that, for every $a \in \mathcal{A}$ and each vertex $x = aa \dots a \in \mathcal{G}_n$, the limit

$$\lim_{|x|=n \rightarrow \infty} u_x(\lambda)^{1/n} = \beta_a = \beta_a(\lambda)$$

exists and that $u_x(\lambda) \leq \beta_a(\lambda)^n$ for all $n \geq 0$, we verified the claim for $n = 1$ and gain as an immediate consequence that

$$u_x(\lambda) \leq u_{y_n}(\lambda) \leq u_{z_{n-1}}(\lambda) \leq \beta_c(\lambda)^{n-1}$$

for all $n \geq 1$, which, in light of $\beta_c(\lambda) \leq \max_{a \in \mathcal{A}} \beta_a(\lambda)$, finishes the proof. \square

Remarks

(1) We wish to stress that Lemma 1 has valuable consequences because the upper bound on the decay rate of the u_x is strictly < 1 , uniformly in λ for $d \geq 2$, under some assumption on λ . More precisely, if there exists $\varepsilon > 0$ such that $\lambda_i, \lambda_j \geq \varepsilon$ for at least two distinct $i, j \in \mathcal{A}_+$, then it can be shown that there is a number $\rho = \rho(\varepsilon) < 1$ such that $\beta_i(\lambda) \leq \rho$ for each such λ and for every $i \in \mathcal{A}$.

(2) When can c be taken to be b in the proof above? Intuitively, it is quite reasonable for the infection to always choose b to continue along a path to move as fast as possible. It hence is of interest to know whether $u_{bb \dots b}(\lambda)$ maximizes $u_x(\lambda)$ for $bb \dots b, x \in \mathcal{G}_n$. Here, we leave this question open.

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