First passage percolation and a model for competing spatial growth

Olle Häggström* Robin Pemantle†

Chalmers University of Technology
University of Wisconsin

Abstract

An interacting particle system modelling competing growth on the $\mathbf{Z}^2$ lattice is defined as follows. Each $x \in \mathbf{Z}^2$ is in one of the states $\{0, 1, 2\}$. 1's and 2's remain in their states forever, while a 0 flips to a 1 (resp. a 2) at a rate equal to the number of its neighbours which are in state 1 (resp. 2). This is a generalization of the well known Richardson model. 1's and 2's may be thought of as two types of infection, and 0's as uninfected sites. We prove that if we start with a single site in state 1 and a single site in state 2, then there is positive probability for the event both types of infection reach infinitely many sites. This result implies that the spanning tree of time-minimizing paths from the origin in first passage percolation with exponential passage times has at least two topological ends with positive probability.

Keywords: First passage percolation, Richardson's model, tree of infection, competing growth.

AMS Subject Classification: Primary 60K35, secondary 82B43.

*Research supported by grants from the Swedish Natural Science Council and the Royal Swedish Academy of Sciences.

†Research supported in part by a grant from the Alfred P. Sloan Foundation, by a Presidential Faculty Fellowship and by NSF grant # DMS9300191

1 Introduction and statements of results

We consider two-dimensional first passage percolation, where each pair of nearest neighbours of $\mathbf{Z}^2$ have an edge connecting them and each edge is equipped with a nonnegative random variable which is interpreted as the time it takes to traverse the edge (see [1] and [3] for reviews). We specialize to the case where these passage times are i.i.d. exponentials; this is the so called Richardson model [8]. By time scaling, we may assume without loss of genertality that the exponential distribution has mean one. For $x, y \in \mathbf{Z}^2$, let $T(x, y)$ be the time taken to reach y from x, i.e. $T(x, y)$ is the infimum over all paths from x to y of the sum of the passage times along the path. Write $\mathbf{0}$ for the origin and let, for $t \geq 0$,

$$B(t) = \{x \in \mathbf{Z}^2 : T(\mathbf{0}, x) \leq t\}$$

so that $B(t)$ is the set of sites reached from the origin by time t. It follows from the memoryless property of the exponential distribution that $B(t)$ is a Markov process, and the following interacting particle system formulation is natural. Each site x takes the value $\eta_x(t) \in \{0, 1\}$ at time t. Let

$$\eta_x(t) = \begin{cases} 1 & \text{if } x \in B(t) \\ 0 & \text{otherwise.} \end{cases}$$

and write $\eta(t) = \{\eta_x(t)\}_{x \in \mathbf{Z}^2}$ for the whole configuration of 0's and 1's at time t. We may think of sites in state 0 as healthy and those in state 1 as infected. Each site in state 1 remains in this state forever and tries to infect each of its four nearest neighbours at rate one, so that a site in state 0 flips to state 1 at rate equal to the number of nearest neighbours with state 1. At time 0, only the origin is infected.

One of the most important results in first passage percolation is the existence of the so called time constant μ such that

$$\lim_{n \to \infty} \frac{T(\mathbf{0}, (n, 0))}{n} = \mu \quad \text{a.s.} \tag{1}$$

which is a consequence of Kingman's subadditive ergodic theorem. This result in fact holds under much more general conditions than those considered in this paper. The precise value of μ is not known, although some bounds are available. We will need a lower bound on μ, and the best such bound we are aware of is

$$\mu > 0.298 \tag{2}$$

as shown in [2]. Similar time constants exist for all directions. Moreover, there is the following asymptotic shape result which, somewhat loosely speaking, states that analogues of (1) hold for all directions simultaneously: Let

$$\bar{B}(t) = \{x \in \mathbf{R}^2 : \exists y \in [-\tfrac{1}{2}, \tfrac{1}{2}]^2, z \in B(t) \text{ such that } y + z = x\},$$

i.e. $\bar{B}(t)$ is a "fattened" version of $B(t)$. Then there exists a nonrandom compact convex set B_0 such that for all $\varepsilon > 0$

$$(1-\varepsilon)B_0 \subseteq \frac{\bar{B}(t)}{t} \subseteq (1+\varepsilon)B_0 \quad \text{eventually a.s.,} \tag{3}$$

see e.g. [1].

We will be interested in a different aspect of the evolution of infection, namely the **tree of infection**, to be denoted by Γ. Let $\Gamma(t)$ be the graph with vertex set $B(t)$ and edge set obtained as follows. For each $x \in B(t) \setminus \{\mathbf{0}\}$, let e_x be the edge connecting x to the vertex y from which x got infected, and let $\{e_x : x \in B(t)\setminus\{\mathbf{0}\}\}$ be the edge set of $\Gamma(t)$. Each vertex of $\Gamma(t)$ gets a unique path to $\mathbf{0}$, so $\Gamma(t)$ is indeed a tree. Moreover, both the vertex set and the edge set of $\Gamma(t)$ are increasing in t, so the limiting object $\Gamma = \lim_{n\to\infty} \Gamma(t)$ also exists. The tree structure of $\Gamma(t)$ is inherited by Γ. Equivalently, we may define Γ as the graph with vertex set $\mathbf{Z}^2$ and edge set given by

$$\bigcup_{x \in \mathbf{Z}^2 \setminus \{\mathbf{0}\}} \{e : e \text{ is an edge of the fastest path from } \mathbf{0} \text{ to } x\}.$$

Our main interest is in the number of topological ends of Γ, i.e. how many infinite self-avoiding paths starting at $\mathbf{0}$ does Γ contain? Let $K(\Gamma)$ denote the number of such paths. By a standard compactness argument, $K(\Gamma) \geq 1$. Newman [6] has shown that $K(\Gamma) = \infty$ a.s. provided a certain hypothesis concerning uniformly bounded curvature of B_0. (See also [5] for related results.) The uniformly bounded curvature hypothesis is highly plausible, but has so far not been not proved. We shall prove the following result which is a small step towards the conjecture that $K(\Gamma) = \infty$ a.s.

Theorem 1.1: *The number of topological ends $K(\Gamma)$ of the tree of infection satisfies*

$$P(K(\Gamma) \geq 2) \geq \tfrac{4\mu - 1}{3} > 0.$$

In order to prove this result, we shall first study a simple and natural model for competing spatial growth. We will now go on to describe this model, which is a variant of the Richardson model and which we propose to call the **two-type Richardson model**.

Consider an interacting particle system on $\mathbf{Z}^2$ with state space $\{0, 1, 2\}$, where 0's may be thought of as healthy sites, and 1's and 2's as two different types of infection. The evolution is as follows. A site in state 1 stays in this state forever, and the same thing holds for a site in state 2. Both types of infected sites try to infect each of their nearest neighbours at rate one, so that a site in state 0 flips to state 1 at rate equal to the number of nearest neighbours in state 1, and to state

2 at rate equal to the number of nearest neighbours in state 2. We will write $\xi(t)$ for the configuration at time t; $\xi(t)$ will be an element of $\{0,1,2\}^{\mathbf{Z}^2}$. Note that if we disregard the type of infection (i.e. if we watch this system evolve wearing a pair of glasses which prevents us from distinguishing between 1's and 2's), then the system behaves exactly as the ordinary (one-type) Richardson model. This is an immediate consequence of the fact that the total flip rate of a site in state 0 equals its total number of infected nearest neighbours.

We start at time 0 with an infection of type 1 at $\mathbf{0}$ and an infection of type 2 at another site x, all other sites being healthy. The model can then be described in first passage percolation terms: A site y is infected at time $T(\{\mathbf{0}, x\}, y)$, which we define as the infimum, over all paths starting at $\mathbf{0}$ or x and ending at y, of the sum of the passage times along the path. Since the distribution of the passage times of the edges is continuous, it is not hard to see that the infimum is in fact a.s. a minimum which is attained for a unique path. If this fastest path starts at $\mathbf{0}$, then y gets infection of type 1, otherwise it gets type 2.

We may think of the two-type Richardson model as a crude model for two growing bacteria colonies (or two political empires) competing for space. It may happen that at some early stage, one of the types of infection completely surrounds the other type which then is prevented from growing indefinitely. Write A for the event that this does not happen, in which case both types of infection will grow indefinitely. The first question one would like to answer about the two-type Richardson model is whether or not $P(A) > 0$ (it is obvious that $P(A) < 1$). The answer to this question is in fact independent of x, as stated in the following proposition, where we write $P_{\mathbf{0},x}(A)$ for the probability of A with the described starting configuration. The proof will be given in Section 2.

Proposition 1.2: *For any $x_1, x_2 \in \mathbf{Z}^2 \setminus \{\mathbf{0}\}$,*

$$P_{\mathbf{0},x_1}(A) > 0 \quad \textit{implies} \quad P_{\mathbf{0},x_2}(A) > 0.$$

Hence we may restrict attention to $x = \mathbf{1} := (1,0)$, i.e. to the two-type Richardson model starting with a 1 at the origin, a 2 right next to the origin, and all other sites healthy. Our main result on the two-type Richardson model now says

Theorem 1.3:

$$P_{\mathbf{0},\mathbf{1}}(A) \geq \tfrac{4\mu - 1}{3} > 0,$$

so that with positive probability both bacteria colonies keep growing forever. Inserting (2) yields $P_{\mathbf{0},\mathbf{1}}(A) > 0.064$.

Let us now go back to the one-type Richardson model and the question of the number $K(\Gamma)$ of topological ends of the tree of infection. Start with a single infected site at $\mathbf{0}$ and suppose that the first site that $\mathbf{0}$ infects is $\mathbf{1}$. It is then an immediate consequence of Theorem 1.3 and the identification between the one-type Richardson model and the two-type Richardson model with types disregarded, that there is positive probability for the event that Γ has two different self-avoiding paths to infinity: one which goes through $\mathbf{1}$ and one which does not. Hence **Theorem 1.3 implies Theorem 1.1**.

Similarly, if it had turned out that infinite growth of both bacteria colonies had probability zero, then we would have been able to conclude that $K(\Gamma) = 1$ a.s. This remark is of course an empty statement, but we point it out anyway because it is feasible that a similar implication might be useful in some of the possible extensions of the model considered here; see below.

In order to prove Theorem 1.3, we will need the following proposition, which will be proved in Section 2. We think it is of some interest in its own, and it also seems related to questions concerning the roughness of the boundary of $B(t)$, studied e.g. in [4] and [7].

Proposition 1.4: *For any $\varepsilon > 0$, there exist infinitely many $x = (x_1, x_2) \in \mathbf{Z}^2$ in the right half-plane, such that*

$$P[T(\mathbf{0}, (x_1, x_2)) > T(\mathbf{0}, (x_1 - 1, x_2))] > \tfrac{2\mu+1}{3} - \varepsilon, \tag{4}$$

which is greater than 0.5 when ε is small. In fact, the stronger result holds that for some $l \in \{0, 1, \ldots\}$

$$\limsup_{n\to\infty} P[T(\mathbf{0}, (n, l)) > T(\mathbf{0}, (n - 1, l))] > \tfrac{2\mu+1}{3} - \varepsilon. \tag{5}$$

Inserting (2) gives that the right hand sides in (4) and (5) can be made greater that 0.532. Intuitively, Proposition 1.4 says that there are sites in $\mathbf{Z}^2$ arbitrarily far away from the origin which "strongly feel" from which direction the infection is coming. It seems reasonable to believe that the lim sup in (5) in fact is a limit, and independent of l.

Apart from proving the conjecture about Γ a.s. having infinitely many topological ends, there are various other ways in which one might want to extend and improve the results of this paper. An obvious question is what happens when $\mathbf{Z}^2$ is replaced by $\mathbf{Z}^d$ for $d \geq 3$. Another direction is to allow passage time distributions other than the exponential. The Markovian behaviour of $B(t)$ is then lost whence the interacting particle system formulation becomes less natural, but the questions about Γ remain just as natural as for the exponential case. An inspection of the proof of Proposition 1.4 shows that the result $P(K(\Gamma) \geq 2) > 0$ extends to the case where the passage times of the edges have a continuous distribution with a hazard rate which is bounded between two constants $\alpha < \beta$ satisfying $\frac{\alpha}{\beta} > \frac{1-3\mu}{\mu}$, (here μ still denotes the time constant when the passage time distribution is a mean one exponential). One can also ask what happens if we extend the two-type Richardson model to having three or more different types, in the obvious way. For this extension, showing that k different types simultaneously can grow indefinitely from a finite starting configuration is equivalent to showing that $P(K(\Gamma) \geq k) > 0$. Yet another direction of generalization would be to allow the two types to have different infection rates λ_1 and λ_2, and for this extension we conjecture that $P(A) = 0$ whenever $\lambda_1 \neq \lambda_2$.

Since Theorem 1.3 implies Theorem 1.1, it only remains to prove Propositions 1.2 and 1.4, and Theorem 1.3. The remainder of the paper is devoted to this task. The hard work is in the proof of Proposition 1.4.

2 Proofs

Proof of Proposition 1.2: Suppose $P_{\mathbf{0},x_1}(A) > 0$, and let S_r be the circle of radius

$$r = \max(|x_1|, |x_2|) + 2$$

(here $|\cdot|$ is the Euclidean norm) centered at $\mathbf{0}$. By conditioning on the first n infections (for some n which we need not define explicitly here) we can find $t > 0$ and a configuration $\xi \in \{0,1,2\}^{\mathbf{Z}^2}$ such that ξ restricted to $\mathbf{Z}^2 \cap S_r$ contains only 1's and 2's, and such that $P_{\mathbf{0},x_1}(\xi(t) = \xi, A) > 0$. Note that $\{x \in \mathbf{Z}^2 : \xi_x = 1\}$ and $\{x \in \mathbf{Z}^2 : \xi_x = 2\}$ both must be connected sets (in the graph-theoretic sense with edges between all nearest neighbours). Let

$$B_\xi = \{x \in \mathbf{Z}^2 : \xi_x \in \{1, 2\}\}$$

be the set the set of infected sites in ξ; by choice of r also B_ξ is connected. Define

$$\partial B_\xi^* = \{x \in \mathbf{Z}^2 : \xi_x \in \{1,2\} \text{ and } \exists y \in \mathbf{Z}^2 \text{ such that } \xi_y = 0 \text{ and } |x - y| = 1\},$$

i.e. ∂B_ξ is the set of infected sites with at least one healthy neighbour. It is easy to check, using the strong Markov property, that $P_{\mathbf{0},x_1}(A|\xi(t) = \xi)$ does not depend on the values of ξ on $B_\xi \setminus \partial B_\xi$. By choice of r, we may now construct a configuration $\xi' \in \{0,1,2\}^{\mathbf{Z}^2}$ such that

(i) $B_{\xi'} = B_\xi$ (whence in particular $\partial B_{\xi'} = \partial B_\xi$),

(ii) $\xi' = \xi$ on ∂B_ξ,

(iii) $\xi'_{\mathbf{0}} = 1$ and $\xi'_{x_2} = 2$,

(iv) $\{x \in \mathbf{Z}^2 : \xi'_x = 1\}$ and $\{x \in \mathbf{Z}^2 : \xi'_x = 2\}$ are both connected.

B'_ξ is finite, whence by (iii) and (iv) we have $P_{\mathbf{0},x_2}(\xi(t) = \xi') > 0$ because only finitely many infections are involved. Hence,

$$\begin{aligned} P_{\mathbf{0},x_2}(A) &\geq P_{\mathbf{0},x_2}(\xi(t) = \xi', A) \\ &= P_{\mathbf{0},x_2}(A|\xi(t) = \xi')P_{\mathbf{0},x_2}(\xi(t) = \xi') \\ &= P_{\mathbf{0},x_1}(A|\xi(t) = \xi)P_{\mathbf{0},x_2}(\xi(t) = \xi') \\ &> 0 \end{aligned}$$

as desired. □

Proof of Proposition 1.4: Our aim is to show that (5) holds for fixed $\varepsilon > 0$ and some l. We will consider the evolution of $B(t)$ inside the half-strip $S_k = \{0, 1, \ldots\} \times \{0, 1, \ldots, k\}$ for some large k (taking $k > \frac{2}{3\varepsilon}$ will suffice). Writing $N_k(t)$ for the number of infected sites in S_k at time t, we have, as an easy consequence of (1), that

$$\lim_{t\to\infty} \frac{N_k(t)}{t} = \tfrac{k+1}{\mu} \qquad \text{a.s.} \tag{6}$$

For $x \in \mathbf{Z}^2$, write $\lambda_x(t)$ for the flip rate of x at time t, and write $\lambda_{S_k}(t)$ for $\sum_{x\in S_k} \lambda_x(t)$. Furthermore, for $i = 0, 1, \ldots$, write τ_k^i for $\inf\{t \geq 0 : N_k(t) \geq i\}$, and for $i = 1, 2, \ldots$ define

$$\sigma_k^i = \int_{-\tau_k^{i-1}}^{\tau_k^i} \lambda_{S_k}(u)du$$

so that in words, σ_k^i is the accumulated infection rate between the $(i-1)$th and the ith infection in S_k. By the strong Markov property, we have that $\sigma_k^1, \sigma_k^2, \ldots$ are i.i.d. exponentials with mean 1. By the strong law of large numbers, we thus have that

$$\lim_{i\to\infty} \tfrac{1}{i} \int_0^{\tau_k^i} \lambda_{S_k}(u)du = \lim_{i\to\infty} \tfrac{1}{i} \sum_{j=1}^{i} \sigma_k^j = 1 \qquad \text{a.s.}$$

whence

$$\lim_{t\to\infty} \frac{\int_0^t \lambda_{S_k}(u)du}{N_k(t)} = 1 \qquad \text{a.s.}$$

In combination with (6), this implies that

$$\lim_{t\to\infty} \tfrac{1}{t} \int_0^t \lambda_{S_k}(u)du = \tfrac{k+1}{\mu} \qquad \text{a.s.} \tag{7}$$

Now write $N_k^G(t)$ (G as in good) for the number of infected sites in S_k at time t which became infected after their nearest neighbour to the left, and write $N_k^B(t)$ (B for bad) for the corresponding number of sites which became infected after their nearest neighbour to the right. The asymptotic shape result (3) implies for any $\varepsilon' > 0$ that a.s. for large enough t the set of infected sites in S_k contains

$\{0,1,\ldots,\frac{(1-\varepsilon')t}{\mu}\}\times\{0,1,\ldots,k\}$ and is contained in $\{0,1,\ldots,\frac{(1+\varepsilon')t}{\mu}\}\times\{0,1,\ldots,k\}$. For each pair $\{x,x+(1,0)\}$ of nearest neighbours in $\{0,1,\ldots,\frac{(1-\varepsilon')t}{\mu}\}\times\{0,1,\ldots,k\}$, we then have either

(a) x contributes 1 to the sum $N_k^B(t)$, whereas $x+(1,0)$ does not contribute to $N_k^G(t)$, or

(b) $x+(1,0)$ contributes 1 to $N_k^G(t)$, whereas x does not contribute to $N_k^B(t)$.

By letting $\varepsilon'\to 0$, we get that

$$\lim_{t\to\infty}\frac{N_k^G(t)+N_k^B(t)}{t}=\tfrac{k+1}{\mu}\quad\text{a.s.}$$

Write $\lambda_{S_k}^G(t)$ for $\sum\lambda_x(t)$ where this time the sum is taken over uninfected sites in S_k whose nearest neighbour to the left is already infected at time t, and define $\lambda_{S_k}^B(t)$ analogously. We have as in (7) that

$$\lim_{t\to\infty}\frac{\int_0^t\lambda_{S_k}^G(u)du}{N_k^G(t)}=1\quad\text{a.s.}$$

and

$$\lim_{t\to\infty}\frac{\int_0^t\lambda_{S_k}^G(u)du+\int_0^t\lambda_{S_k}^B(u)du}{\int_0^t\lambda_{S_k}(u)du}=1\quad\text{a.s.}\tag{8}$$

In order to show that (5) holds for some $l\in\{0,\ldots,k\}$, it suffices to prove that

$$\liminf_{t\to\infty}\frac{N_k^G(t)}{N_k(t)}>\tfrac{2\mu+1}{3}-\varepsilon\quad\text{a.s.}$$

To this end, it is sufficient to show that

$$\liminf_{t\to\infty}\frac{\int_0^t\lambda_{S_k}^G(u)du}{\int_0^t\lambda_{S_k}(u)du}>\tfrac{2\mu+1}{3}-\varepsilon\quad\text{a.s.}\tag{9}$$

so this is what we will proceed to prove.

We now look at a configuration $\tilde\eta_{S_k}\in\{0,1\}^{S_k}$, which will serve as an example of what $\eta_{S_k}(t)$ may look like at some reasonably large time point t. Since $\eta_{(0,i)}(t)=1$ for $i=0,\ldots,k$ and all sufficiently large t a.s., we assume that $\tilde\eta_{(0,i)}=1$ for $i=0,\ldots,k$. Also $\eta_x(t)=0$ for fixed t and all but finitely many $x\in\mathbf{Z}^2$, so we furthermore assume that $\tilde\eta_x=0$ for all but finitely many $x\in S_k$. Now pick $i\in\{0,1,\ldots,k-1\}$ and write $\tilde\eta_{S_k,i}$ for $\tilde\eta_{S_k}$ restricted to $\{0,1,\ldots\}\times\{i,i+1\}$, i.e. $\tilde\eta_{S_k,i}$ is the configuration on two infinite adjacent horizontal rows. Given $\tilde\eta_{S_k,i}$, we may partition $\{0,1,\ldots\}$ into 1-blocks, 0-blocks, down-blocks, up-blocks and excursion-blocks as in Figure 1.

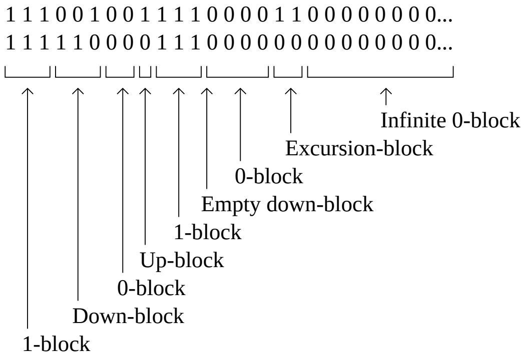

Figure 1: A typical blockpartition.

The blocks are determined (uniquely, given $\tilde{\eta}_{S_k,i}$) as follows. A 1-block is a maximal sequence of $\left[\begin{smallmatrix}1\\1\end{smallmatrix}\right]$'s and a 0-block is a maximal sequence of $\left[\begin{smallmatrix}0\\0\end{smallmatrix}\right]$'s, while down-blocks, up-blocks and excursion-blocks are maximal sequences consisting of $\left[\begin{smallmatrix}0\\1\end{smallmatrix}\right]$'s and $\left[\begin{smallmatrix}1\\0\end{smallmatrix}\right]$'s. All blocks are finite except for the final 0-block. A 1-block is always followed either by a down-block or by an excursion-block. A finite 0-block is always followed by an up-block or an excursion-block. A down-block is always followed by a 0-block while an up-block always is followed by a 1-block. An excursion-block, finally, is always surrounded either by two 1-blocks or by two 0-blocks. Down-blocks and up-blocks (but no other blocks) may be empty; an empty down-block is inserted whenever a $\left[\begin{smallmatrix}1\\1\end{smallmatrix}\right]$ is followed by a $\left[\begin{smallmatrix}0\\0\end{smallmatrix}\right]$, and, similarly, an empty up-block is inserted whenever a $\left[\begin{smallmatrix}0\\0\end{smallmatrix}\right]$ is followed by a $\left[\begin{smallmatrix}1\\1\end{smallmatrix}\right]$.
Call 1-blocks and 0-blocks **pure**, and call other blocks **mixed**. Call a pair of nearest neighbours **hot** if one of the sites has value 1 and the other has value 0. Each hot pair will be associated with a mixed block as follows. If a $\left[\begin{smallmatrix}1\\1\end{smallmatrix}\right]$ is followed by a $\left[\begin{smallmatrix}0\\0\end{smallmatrix}\right]$, then the two hot pairs are associated with the corresponding empty down-block, and similarly when a $\left[\begin{smallmatrix}0\\0\end{smallmatrix}\right]$ is followed by a $\left[\begin{smallmatrix}1\\1\end{smallmatrix}\right]$. Any other hot pair intersects exactly one mixed block and is then associated with this block.

We now consider rates of infection between sites in $\{0, 1, \ldots\} \times \{i, i+1\}$ when $\eta_{S_k}(t) = \tilde{\eta}_{S_k}$. Write $\tilde{\lambda}(M)$ for the weighted total infection rate for the hot pairs associated with the mixed block M, with weight $\frac{1}{2}$ for horizontal infections and weight 1 for vertical infections. The reason for these weights is that each horizontal infection is accounted for in $\tilde{\eta}_{S_k,i}$ for two different values of i (this is not true for rows 0 and k, but that will be corrected for later). Write $\tilde{\lambda}^G(M)$ (resp. $\tilde{\lambda}^B(M)$) for the corresponding sum where only good (resp. bad) infections are counted.

The following is easily checked to hold for $\tilde{\lambda}(M)$, $\tilde{\lambda}^G(M)$ and $\tilde{\lambda}^B(M)$.

Type of block	$\tilde{\lambda}(M)$	$\tilde{\lambda}^G(M) - \tilde{\lambda}^B(M)$
empty down-block	1	1
empty up-block	1	-1
nonempty down-block	≥ 2	2
nonempty up-block	≥ 2	-2
excursion-block	≥ 2	0

Write $\tilde{\lambda}_{S_k,i}$ (resp. $\tilde{\lambda}^G_{S_k,i}$ and $\tilde{\lambda}^B_{S_k,i}$) for $\lambda(\tilde{M})$ (resp. $\tilde{\lambda}^G(M)$ and $\tilde{\lambda}^B(M)$) summed over all mixed blocks in $\tilde{\eta}_{S_k,i}$. Note that since $\tilde{\eta}_{S_k,i}(t)$ starts with a 1-block and ends with a 0-block, we have that the number of down-blocks exceeds the number of up-blocks by exactly 1. Using this observation, and the above table, we may check (e.g. via induction over the number of down-blocks) that

$$\tilde{\lambda}_{S_k,i} \geq 4 - 3(\tilde{\lambda}^G_{S_k,i} - \tilde{\lambda}^B_{S_k,i}) \tag{10}$$

with equality if and only if the only mixed blocks in $\tilde{\lambda}_{S_k,i}$ are empty down-blocks and nonempty up-blocks with $\tilde{\lambda}(M) = 2$ for each up-block M. Applying (10) to $\eta_{S_k,i}(t)$ and summing over i, we get

$$\sum_{i=0}^{k-1} \lambda_{S_k,i}(t) \geq 4k - 3\sum_{i=0}^{k-1}(\lambda^G_{S_k,i}(t) - \lambda^B_{S_k,i}(t))$$

for all sufficiently large t a.s., whence

$$\liminf_{t\to\infty} \sum_{i=0}^{k-1} \frac{1}{t}\int_0^t (\lambda_{S_k,i}(u) + 3\lambda^G_{S_k,i}(u) - 3\lambda^B_{S_k,i}(u))du \geq 4k. \tag{11}$$

We next need to control the discrepancy between $\sum_{i=0}^{k-1} \lambda_{S_k,i}(u)$ and $\lambda_{S_k}(u)$, i.e. the rate of infection from rows -1 and $k+1$ to rows 0 and k, plus $\frac{1}{2}$ times the rate of infection within rows 0 and k. Since the number of infections up to time t in rows 0 and k is asymptotically $2\mu^{-1}t$, we have by the same arguments as those used to show (7) that

$$\liminf_{t\to\infty} \frac{1}{t}\int_0^t \left(\lambda_{S_k}(u) - \sum_{i=0}^{k-1}\lambda_{S_k,i}(u)\right) du \leq 2\mu^{-1}$$

and for the same reason

$$\liminf_{t\to\infty} \frac{1}{t}\int_0^t \left(\lambda^B_{S_k}(u) - \sum_{i=0}^{k-1}\lambda^B_{S_k,i}(u)\right) du \leq 2\mu^{-1}.$$

Hence, we may modify (11) to get

$$\liminf_{t\to\infty} \tfrac{1}{t} \int_0^t (\lambda_{S_k}(u) + 3\lambda^G_{S_k}(u) - 3\lambda^B_{S_k}(u))du \geq 4k - 6\mu^{-1}.$$

Combining this with (8) yields

$$\liminf_{t\to\infty} \tfrac{1}{t} \int_0^t (6\lambda^G_{S_k}(u) - 2\lambda_{S_k}(u))du \geq 4k - 6\mu^{-1}$$

i.e.

$$\begin{aligned}\liminf_{t\to\infty} \tfrac{1}{t} \int_0^t 6\lambda^G_{S_k}(u)du &\geq 4k - 6\mu^{-1} + 2\lim_{t\to\infty} \tfrac{1}{t}\int_0^t \lambda_{S_k}(u)du \\ &= 4k - 6\mu^{-1} + 2(k+1)\mu^{-1} \\ &= k(4+2\mu^{-1}) - 4\mu^{-1}\end{aligned}$$

which in conjunction with (7) implies

$$\liminf_{t\to\infty} \frac{\int_0^t \lambda^G_{S_k}(u)du}{\int_0^t \lambda_{S_k}(u)du} \geq \tfrac{k(4+2\mu^{-1})-4\mu^{-1}}{6k\mu^{-1}} = \tfrac{2\mu+1}{3} - \tfrac{2}{3k}.$$

Taking $k > \frac{2}{3\varepsilon}$, this implies (9), so the proof is complete. □

Proof of Theorem 1.3: Suppose for contradiction that $P_{\mathbf{0},\mathbf{1}}(A) < \frac{4\mu-1}{3} - 2\varepsilon$ for some $\varepsilon > 0$. Then, by symmetry, the probability that infection of type 2 eventually stops growing is greater than $\frac{1}{2}(1 - \frac{4\mu-1}{3} + 2\varepsilon) = \frac{2-2\mu}{3} + \varepsilon$. This implies

$$\limsup_{n\to\infty} P[T(\mathbf{0},(n,l)) > T(\mathbf{1},(n,l))] < 1 - \tfrac{2-2\mu}{3} - \varepsilon = \tfrac{2\mu+1}{3} - \varepsilon,$$

where l can be chosen as in Proposition 1.4. By reflecting the realization of passage times in the line $x_1 = \frac{n}{2}$, we see that the pair $(T(\mathbf{0},(n,l)), T(\mathbf{1},(n,l)))$ has the same joint distribution as $(T(\mathbf{0},(n,l)), T(\mathbf{0},(n-1,l)))$, so that

$$\limsup_{n\to\infty} P[T(\mathbf{0},(n,l)) > T(\mathbf{0},(n-1,l))] < \tfrac{2\mu+1}{3} - \varepsilon$$

contradicting (5). □

Acknowledgement. We thank Jeff Steif and an anonymous referee for helpful comments on the manuscript.

References

[1] Durrett, R. (1988) *Lecture Notes on Particle Systems and Percolation*, Wadsworth & Brooks/Cole, Pacific Grove.

[2] Janson, S. (1981) An upper bound for the velocity of first-passage percolation, *J. Appl. Probab.* **18**, 256–262.

[3] Kesten, H. (1987) Percolation theory and first-passage percolation, *Ann. Probab.* **15**, 1231–1271.

[4] Kesten, H. (1993) On the speed of convergence in first-passage percolation, *Ann. Appl. Probab.* **3**, 296–338.

[5] Licea, C. and Newman, C.M. (1996) Geodesics in two-dimensional first-passage percolation, *Ann. Probab.* **24**, 399–410.

[6] Newman, C.M. (1995) A surface view of first-passage percolation, *Proceedings of the 1994 International Congress of Mathematicians* (ed. S.D. Chatterij), 1017–1023, Birkhäuser, Boston.

[7] Newman, C.M. and Piza, M. (1995) Divergence of shape fluctuations in two dimensions, *Ann. Probab.* **23**, 97–1005.

[8] Richardson, D. (1973) Random growth in a tesselation, *Proc. Cambridge Phil. Soc.* **74**, 515–528.