The pleasures and pains of studying the two-type Richardson model

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Abstract

This paper provides a survey of known results and open problems for the two-type Richardson model, which is a stochastic model for competition on $\mathbb{Z}^d$. In its simplest formulation, the Richardson model describes the evolution of a single infectious entity on $\mathbb{Z}^d$, but more recently the dynamics have been extended to comprise two competing growing entities. For this version of the model, the main question is whether there is a positive probability for both entities to simultaneously grow to occupy infinite parts of the lattice, the conjecture being that the answer is yes if and only if the entities have the same intensity. In this paper attention focuses on the two-type model, but the most important results for the one-type version are also described.

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1 Introduction

Consider an interacting particle system in which, at any time t, each site $x \in \mathbb{Z}^d$ is in either of two states, denoted by 0 and 1. A site in state 0 flips to a 1 at rate proportional to the number of nearest neighbors in state 1, while a site in state 1 remains a 1 forever. We may think of sites in state 1 as being occupied by some kind of infectious entity, and the model then describes the propagation of an infection where each infected site tries to infect each of its

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nearest neighbors on $\mathbb{Z}^d$ at some constant rate $\lambda > 0$. More precisely, if at time t a vertex x is infected and a neighboring vertex y is uninfected, then, conditional on the dynamics up to time t, the probability that x infects y during a short time window $(t, t+h)$ is $\lambda h + o(h)$. Here and in what follows, sites in state 0 and 1 are referred to as uninfected and infected respectively. This is the intuitive description of the model; a formal definition is given in Section 2.

The model is a special case of a class of models introduced by Richardson (1973), and is commonly referred to as the Richardson model. It has several cousins among processes from mathematical biology, see e.g. Eden (1961), Williams and Bjerknes (1972) and Bramson and Griffeath (1981). The model is also a special case of so called first-passage percolation, which was introduced in Hammersley and Welsh (1965) as a model for describing the passage of a fluid through a porous medium. In first-passage percolation, each edge of the $\mathbb{Z}^d$-lattice is equipped with a random variable representing the time it takes for the fluid to traverse the edge, and the Richardson model is obtained by letting these passage times be i.i.d. exponential.

Since an infected site stays infected forever, the set of infected sites in the Richardson model increases to cover all of $\mathbb{Z}^d$ as $t \to \infty$, and attention focuses on *how* this set grows. The main result is roughly that the infection grows linearly in time in each fixed direction and that, scaled by a factor $1/t$, the set of infected points converges to a non-random asymptotic shape as $t \to \infty$. To prove that the growth is linear in a fixed direction involves Kingman's subadditive ergodic theorem – in fact, the study of first-passage percolation was one of the main motivations for the development of subadditive ergodic theory. That the linear growth is preserved when all directions are considered simultaneously is stated in the celebrated shape theorem (Theorem 2.1 in Section 3) which originates from Richardson (1973).

Now consider the following extension of the Richardson model, known as the two-type Richardson model and introduced in Häggström and Pemantle (1998). Instead of two possible states for the sites there are three states, which we denote by 0, 1 and 2. The process then evolves in such a way that, for $i = 1, 2$, a site in state 0 flips to state i at rate λ_i times the number of nearest neighbors in state i and once in state 1 or 2, a site remains in that state forever. Interpreting states 1 and 2 as two different types of infection and state 0 as absence of infection, this gives rise to a model describing the simultaneous spread of two infections on $\mathbb{Z}^d$. To rigorously define the model requires a bit more work; see Section 3. In what follows we will always assume that $d \geq 2$; the model makes sense also for $d = 1$ but the questions considered here become trivial.

A number of similar extensions of (one-type) growth models to (two-type) competition models appear in the literature; see for instance Neuhauser (1992), Durrett and Neuhauser (1997), Kordzakhia and Lalley (2005) and Ferrari et al. (2006). These tend to require somewhat different techniques, and results tend

not to be easily translated from these other models to the two-type Richardson model (and vice versa). Closer to the latter are (non-Markovian) competition models based on first-passage percolation models with non-exponential passage time variables – Garet and Marchand (2005), Hoffman (2005:1), Hoffman (2005:2), Garet and Marchand (2006), Gouéré (2007), Pimentel (2007) – and a certain continuum model – Deijfen et al. (2004), Deijfen and Häggström (2004), Gouéré (2007). For ease of exposition, we shall not consider these variations even in cases where results generalize.

The behavior of the two-type Richardson model depends on the initial configuration of the infection and on the ratio between the intensities λ_1 and λ_2 of the infection types. Assume first, for simplicity, that the model is started at time 0 from two single sites, the origin being type 1 infected and the site $(1, 0, \ldots, 0)$ next to the origin being type 2 infected. Three different scenarios for the development of the infection are conceivable:

(a) The type 1 infection at some point completely surrounds type 2, thereby preventing type 2 from growing any further.

(b) Type 2 similarly strangles type 1.

(c) Both infections grow to occupy infinitely many sites.

It is not hard to see that, regardless of the intensities of the infections, outcomes (a) and (b) where one of the infection types at some point encloses the other have positive probability regardless of λ_1 and λ_2. This is because each of (a) and (b) can be guaranteed through some finite initial sequence of infections. In contrast, scenario (c) – referred to as infinite coexistence – can never be guaranteed from any finite sequence of infections, and is therefore harder to deal with: the main challenge is to decide whether, for given values of the parameters λ_1 and λ_2, this event (c) has positive probability or not. Intuitively, infinite coexistence represents some kind of power balance between the infections, and it seems reasonable to suspect that such a balance is possible if and only if the infections are equally powerful, that is, when $\lambda_1 = \lambda_2$. This is Conjecture 3.3 in Section 3, which goes back to Häggström and Pemantle (1998), and, although a lot of progress have been made, it is not yet fully proved. We describe the state of the art in Sections 4 and 5.

As mentioned above, apart from the intensities, the development of the infections in the two-type model also depends on the initial state of the model. However, if we are only interested in deciding whether the event of infinite coexistence has positive probability or not, it turns out that, as long as the initial configuration is bounded and one of the sets does not completely surround the other, the precise configuration does not matter, that is, whether infinite coexistence is possible or not is determined only by the relation between the intensities. This is proved in Deijfen and Häggström (2006:1); see Theorem 3.2 in Section 3 for a precise formulation. Of course one may also

consider unbounded initial configurations. Starting with both infection types occupying infinitely many sites means – apart from in very labored cases – that they will both infect infinitely many sites. A more interesting case is when one of the infection types starts from an infinite set and the other one from a finite set. We may then ask if outcomes where the finite type infects infinitely many sites have positive probability or not. This question is dealt with in Deijfen and Häggström (2007), and we describe the results in Section 6.

The dynamics of the two-type Richardson model is deceptively simple, and yet gives rise to intriguing phenomena on a global scale. In this lies a large part of the pleasure indicated in the title. Furthermore, proofs tend to involve elegant probabilistic techniques such as coupling, subadditivity and stochastic comparisons, adding more pleasure. The pain alluded to (which by the way is not so severe that it should dissuade readers from entering this field) comes from the stubborn resistance that some of the central problems have so far put up against attempts to solve them. A case in point is the "only if" direction of the aforementioned Conjecture 3.3, saying that infinite coexistence starting from a bounded initial configuration does not occur when $\lambda_1 \neq \lambda_2$.

2 The one-type model

As mentioned in the introduction, the one-type Richardson model is equivalent to first-passage percolation with i.i.d. exponential passage times. To make the construction of the model more precise, first define $E_{\mathbb{Z}^d}$ as the edge set for the $\mathbb{Z}^d$ lattice (i.e., each pair of vertices $x, y \in \mathbb{Z}^d$ at Euclidean distance 1 from each other have an edge $e \in E_{\mathbb{Z}^d}$ connecting them). Then attach i.i.d. non-negative random variables $\{\tau(e)\}_{e \in E_{\mathbb{Z}^d}}$ to the edges. We take each $\tau(e)$ to be exponentially distributed with parameter $\lambda > 0$, meaning that

$$P(\tau(e) > t) = \exp(-\lambda t)$$

for all $t \geq 0$. For $x, y \in \mathbb{Z}^d$, define

$$T(x, y) = \inf_{\Gamma} \sum_{e \in \Gamma} \tau(e) \tag{1}$$

where the infimum is over all paths Γ from x to y. The Richardson model with a given set $S_0 \subset \mathbb{Z}^d$ of initially infected sites is now defined by taking the set S_t of sites infected at time t to be

$$S_t = \{x \in \mathbb{Z}^d : T(y, x) \leq t \text{ for some } y \in S_0\}. \tag{2}$$

It turns out that the infimum in (1) is a.s. a minimum and attained by a unique path. That S_t grows in the way described in the introduction is a consequence of the memoryless property of the exponential distribution: for any $s, t > 0$ we have that $P(\tau(e) > s + t \,|\tau(e) > s) = \exp(-\lambda t)$.

Note that for any $x, y, z \in \mathbb{Z}^d$ we have $T(x,y) \leq T(x,z) + T(z,y)$. This subadditivity property opens up for the use of subadditive ergodic theory in analyzing the model. To formulate the basic result, let $T(x)$ be the time when the point $x \in \mathbb{Z}^d$ is infected when starting from a single infected site at the origin and write $\mathbf{n} = (n, 0, \ldots, 0)$. It then follows from the subadditive ergodic theorem – see e.g. Kingman (1968) – that there is a constant μ_λ such that $T(\mathbf{n})/n \to \mu_\lambda$ almost surely and in L_1 as $n \to \infty$. Furthermore, a simple time scaling argument implies that $\mu_\lambda = \lambda\mu_1$ and hence, writing $\mu_1 = \mu$, we have that

$$\lim_{n\to\infty} \frac{T(\mathbf{n})}{n} = \lambda\mu \quad \text{a.s. and in } L_1. \tag{3}$$

The constant μ indicates the inverse asymptotic speed of the growth along the axes in a unit rate process and is commonly referred to as the time constant. It turns out that $\mu > 0$, so that indeed the growth is linear in time. Similarly, an analog of (3) holds in any direction, that is, for any $x \in \mathbb{Z}^d$, there is a constant $\mu(x) > 0$ such that $T(nx)/n \to \lambda\mu(x)$. The infection hence grows linearly in time in each fixed direction and the asymptotic speed of the growth in a given direction is an almost sure constant.

We now turn to the shape theorem, which asserts roughly that the linear growth of the infection is preserved also when all directions are considered simultaneously. More precisely, when scaled down by a factor $1/t$ the set S_t converges to a non-random shape A. To formalize this, let $\tilde{S}_t \subset \mathbb{R}^d$ be a continuum version of S_t obtained by replacing each $x \in S_t$ by a unit cube centered at x.

Theorem 2.1 (Shape Theorem) *There is a compact convex set A such that, for any $\varepsilon > 0$, almost surely*

$$(1-\varepsilon)\lambda A \subset \frac{\tilde{S}_t}{t} \subset (1+\varepsilon)\lambda A$$

for large t.

In the above form, the shape theorem was proved in Kesten (1973) as an improvement on the original "in probability" version, which appears already in Richardson (1973). See also Cox and Durrett (1988) and Boivin (1991) for generalizations to first-passage percolation processes with more general passage times. Results concerning fluctuations around the asymptotic shape can be found, e.g., in Kesten (1993), Alexander (1993) and Newman and Piza (1995), and, for certain other passage time distributions, in Benjamini et al. (2003).

Working out exactly, or even approximately, what the asymptotic shape A is has turned out to be difficult. Obviously the asymptotic shape inherits all symmetries of the $\mathbb{Z}^d$ lattice – invarince under reflection and permutation

of coordiante hyperplanes – and it is known to be compact and convex, but, apart from this, not much is known about its qualitative features. These difficulties with characterizing the shape revolve around the fact that $\mathbb{Z}^d$ is not rotationally invariant, which causes the growth to behave differently in different directions. For instance, simulations on $\mathbb{Z}^2$ indicate that the asymptotic growth is slightly faster along the axes as compared to the diagonals. There is however no formal proof of this.

Before proceeding with the two-type model, we mention some work concerning properties of the time-minimizing paths in (1), also known as geodesics. Starting at time 0 with a single infection at the origin $\mathbf{0}$, we denote by $\Gamma(x)$ the (unique) path Γ for which the infimum $T(\mathbf{0}, x)$ in (1) is attained. Define $\Psi = \cup_{x \in \mathbb{Z}^d} \Gamma(x)$, making Ψ a graph specifying which paths the infection actually takes. It is not hard to see that Ψ is a tree spanning all of $\mathbb{Z}^d$ and hence there must be at least one semi-infinite self-avoiding path from the origin (called an end) in Ψ. The issue of whether Ψ has more than one end was noted by Häggström and Pemantle (1998) to be closely related to the issue of infinite coexistence in the two-type Richardson model with $\lambda_1 = \lambda_2$: such infinite coexistence happens with positive probability starting from a finite initial configuration if and only if Ψ has at least two ends with positive probability.

We say that an infinite path $x_1, x_2, \ldots$ has asymptotic direction $\hat{x}$ if $x_k/|x_k| \to \hat{x}$ as $k \to \infty$. In $d = 2$, it has been conjectured that every end in Ψ has an asymptotic direction and that, for every $x \in \mathbb{R}^2$, there is at least one end (but never more than two) in Ψ with asymptotic direction $\hat{x}$. In particular, this would mean that Ψ has uncountably many ends. For results supporting this conjecture, see Newman (1995) and Newman and Licea (1996). In the former of these papers, the conjecture is shown to be true provided an unproven but highly plausible assumption on the asymptotic shape A, saying roughly that the boundary is sufficiently smooth. See also Lalley (2003) for related work.

Results not involving unproven assumptions are comparatively weak: The coexistence result of Häggström and Pemantle (1998) shows for $d = 2$ that Ψ has at least two ends with positive probability. This was later improved to Ψ having almost surely at least $2d$ ends, by Hoffman (2005:2) for $d = 2$ and by Gouéré (2007) for higher dimensions.

3 Introducing two types

The definition of the two-type Richardson model turns out to be simplest in the symmetric case $\lambda_1 = \lambda_2$, where the same passage time variables $\{\tau(e)\}_{e \in E_{\mathbb{Z}^d}}$ as in the one-type model can be used, with $\lambda = \lambda_1 = \lambda_2$. Suppose we start with an initial configuration (S_0^1, S_0^2) of infected sites, where $S_0^1 \subset \mathbb{Z}^d$ are those initially containing type 1 infection, and $S_0^2 \subset \mathbb{Z}^d$ are those initially containing type 2 infection. We wish to define the sets S_t^1 and S_t^2 of type 1 and type 2 infected sites for all $t > 0$. To this end, set $S_0 = S_0^1 \cup S_0^2$, and take the

set $S_t = S_t^1 \cup S_t^2$ of infected sites at time t to be given by precisely the same formula (2) as in the one-type model; a vertex $x \in S_t$ is then assigned infection 1 or 2 depending on whether the $y \in S_0$ for which

$$\inf\{T(y,x) : y \in S_0\}$$

is attained is in S_0^1 or S_0^2.

As in the one-type model, it is a straightforward exercise involving the memoryless property of the exponential distribution to verify that $(S_t^1, S_t^2)_{t\geq 0}$ behaves in terms of infection intensities as described in the introduction.

This construction demonstrates an intimate link between the one-type and the symmetric two-type Richardson model: if we watch the two-type model wearing a pair of of glasses preventing us from distinguishing the two types of infection, what we see behaves exactly as the one-type model. The link between infinite coexistence in the two-type model and the number of ends in the tree of infection Ψ of the one-type model claimed in the previous section is also a consequence of the construction.

In the asymmetric case $\lambda_1 \neq \lambda_2$, the two-type model is somewhat less trivial to define due to the fact that the time it takes for infection to spread along a path depends on the type of infection. There are various ways to deal with this, one being to assign, independently to each $e \in E_{\mathbb{Z}^d}$, two independent random variables $\tau_1(e)$ and $\tau_2(e)$, exponentially distributed with respective parameters λ_1 and λ_2, representing the time it takes for infections 1 resp. 2 to traverse e. Starting from an intial configuration (S_0^1, S_0^2), we may picture the infections as spreading along the edges, taking time $\tau_1(e)$ or $\tau_2(e)$ to cross e depending on the type of infection, with the extra condition that once a vertex becomes hit by one type of infection it becomes inaccessible for the other type. This is intuitively clear, but readers with a taste for detail may require a more rigorous definition, which however we refrain from here; see Häggström and Pemantle (2000) and Deijfen and Häggström (2006:1).

We now move on to describing conjectures and results. Write G_i for the event that type i infects infinitely many sites on $\mathbb{Z}^d$ and define $G = G_1 \cap G_2$. The question at issue is:

$$\text{Does } G \text{ have positive probability?} \tag{4}$$

A priori, the answer to this question may depend both on the initial configuration – that is, on the choice of the sets S_0^1 and S_0^2 – and on the ratio between the infection intensities λ_1 and λ_2. However, it turns out that, if we are not interested in the actual value of the probability of G, but only in whether it is positive or not, then the initial configuration is basically irrelevant, as long as neither of the initial sets completely surrounds the other. This motivates the following definition.

Definition 3.1 *Let ξ_1 and ξ_2 be two disjoint finite subsets of $\mathbb{Z}^d$. We say that one of the sets (ξ_i)* strangles *the other (ξ_j) if there exists no infinite self-avoiding path in $\mathbb{Z}^d$ that starts at a vertex in ξ_j and that does not intersect ξ_i. The pair (ξ_1, ξ_2) is said to be* fertile *if neither of the sets strangles the other.*

Now write $P^{\lambda_1,\lambda_2}_{\xi_1,\xi_2}$ for the distribution of a two-type process started from $S^1_0 = \xi_1$ and $S^2_0 = \xi_2$. We then have the following result.

Theorem 3.2 *Let (ξ_1, ξ_2) and (ξ'_1, ξ'_2) be two fertile pairs of disjoint finite subsets of $\mathbb{Z}^d$, where $d \geq 2$. For all choices of (λ_1, λ_2), we have*

$$P^{\lambda_1,\lambda_2}_{\xi_1,\xi_2}(A) > 0 \Leftrightarrow P^{\lambda_1,\lambda_2}_{\xi'_1,\xi'_2}(A) > 0.$$

For connected initial sets ξ_1 and ξ_2 and $d = 2$, this result is proved in Häggström and Pemantle (1998). The idea of the proof in that case is that, by controlling the passage times of only finitely many edges, two processes started from (ξ_1, ξ_2) and (ξ'_1, ξ'_2) respectively can be made to evolve to the same total infected set after some finite time, with the same configuration of the infection types on the boundary. Coupling the processes from this time on and observing that the development of the infections depends only on the boundary configuration yields the result. This argument however breaks down when the initial sets are not connected (since it is then not sure that the same boundary configuration can be obtained in the two processes) and it is unclear whether it applies for $d \geq 3$. Theorem 3.2 is proved in full generality in Deijfen and Häggström (2006:1), using a more involved coupling construction.

It follows from Theorem 3.2 that the answer to (4) depends only on the value of the intensities λ_1 and λ_2. Hence it is sufficient to consider a process started from $S^1_0 = \mathbf{0}$ and $S^2_0 = \mathbf{1}$ (recall that $\mathbf{n} = (n, 0, \ldots, 0)$), and in this case we drop subscripts and write P^{λ_1,λ_2} for $P^{\lambda_1,\lambda_2}_{\mathbf{0},\mathbf{1}}$. Also, by time-scaling, we may assume that $\lambda_1 = 1$. The following conjecture, where we write $\lambda_2 = \lambda$, goes back to Häggström and Pemantle (1998).

Conjecture 3.3 *In any dimension $d \geq 2$, we have that $P^{1,\lambda}(G) > 0$ if and only if $\lambda = 1$.*

The conjecture is no doubt true, although proving it has turned out to be a difficult task. In fact, the "only if" direction is not yet fully established. In the following two sections we describe the existing results for $\lambda = 1$ and $\lambda \neq 1$ respectively.

4 The case $\lambda = 1$

When $\lambda = 1$, we are dealing with two equally powerful infections and Conjecture 3.3 predicts a positive probability for infinite coexistence. This part of the conjecture has been proved:

Theorem 4.1 *If $\lambda = 1$, we have, for any $d \geq 2$, that $P^{1,\lambda}(G) > 0$.*

This was first proved in the special case $d = 2$ by Häggström and Pemantle (1998). That proof has a very ad hoc flavor, and heavily exploits not only the two-dimensionality but also other specific properties of the square lattice, including a lower bound on the time constant μ in (3) that just happens to be good enough. When eventually the result was generalized to higher dimensions, which was done simultaneously and independently by Garet and Marchand (2005) and Hoffman (2005:1), much more appealing proofs were obtained. Yet another distinct proof of Theorem 4.1 was given by Deijfen and Häggström (2007). All four proofs are different, though if you inspect them for a smallest common denominator you find that they all make critical use of the fact that the time constant μ is strictly positive. We will give the Garet–Marchand proof below. In Hoffman's proof ergodic theory is applied to the tree of infection Ψ and a so-called Busemann function which is shown to exhibit contradictory behavior under the assumption that infinite coexistence has probability zero. The Deijfen–Häggström proof proceeds via the two-type Richardson model with certain infinite initial configurations (cf. Section 6).

Proof of Theorem 4.1: The following argument is due to Garet and Marchand (2005), though our presentation follows more closely the proof of an analogous result in a continuum setting in Deijfen and Häggström (2004) – a paper that, despite the publication dates, was preceded by and also heavily influenced by Garet and Marchand (2005).

Fix a small $\varepsilon > 0$. By Theorem 3.2, we are free to choose any finite starting configuration we want, and here it turns out convenient to begin with a single type 1 infection at the origin $\mathbf{0}$, and a single type 2 infection at a vertex $\mathbf{n} = (n, 0, \ldots, 0)$, where n is large enough so that

(i) $E[T(\mathbf{0}, \mathbf{n})] \leq (1 + \varepsilon)n\mu$, and

(ii) $P(T(\mathbf{0}, \mathbf{n}) < (1 - \varepsilon)n\mu) < \varepsilon$;

note that both (i) and (ii) hold for n large enough due to the asymptotic speed result (3). The reader may easily check, for later reference, that (i) and (ii) together with the nonnegativity of $T(\mathbf{0}, \mathbf{n})$ imply for any event B with $P(B) = \alpha$ that

$$E[T(\mathbf{0}, \mathbf{n}) \,|\, \neg B] \;\leq\; \left(1 + \frac{3\varepsilon}{1 - \alpha}\right) n\mu \,. \tag{5}$$

Next comes an important telescoping idea: for any positive integer k we have

$$\begin{aligned} E[T(\mathbf{0}, k\mathbf{n})] \;\;=\;\; & E[T(\mathbf{0}, \mathbf{n})] + E[T(\mathbf{0}, 2\mathbf{n}) - T(\mathbf{0}, \mathbf{n})] + E[T(\mathbf{0}, 3\mathbf{n}) - T(\mathbf{0}, 2\mathbf{n})] \\ & + \ldots + E[T(\mathbf{0}, k\mathbf{n}) - T(\mathbf{0}, (k-1)\mathbf{n})] \,. \end{aligned}$$

Since $\lim_{k\to\infty} k^{-1} E[T(\mathbf{0}, k\mathbf{n})] = n\mu$, there must exist arbitrarily large k such that

$$E[T(\mathbf{0}, (k+1)\mathbf{n}) - T(\mathbf{0}, k\mathbf{n})] \geq (1 - \varepsilon)n\mu \,.$$

By taking $\mathbf{m} = k\mathbf{n}$, and by translation and reflection invariance, we may deduce that

$$E[T(\mathbf{n}, -\mathbf{m}) - T(\mathbf{0}, -\mathbf{m})] \geq (1 - \varepsilon)n\mu \tag{6}$$

for some arbitrarily large m. We will pick such an m; how large will soon be specified.

The goal is to show that $P(G) > 0$, so we may assume for contradiction that $P(G) = 0$. By symmetry of the initial configuration, we then have that $P(G_1) = P(G_2) = \frac{1}{2}$. This implies that

$$\lim_{m\to\infty} P(-\mathbf{m} \text{ gets infected by type 2}) = \lim_{m\to\infty} P(T(\mathbf{n}, -\mathbf{m}) < T(\mathbf{0}, -\mathbf{m})) = \frac{1}{2}$$

so let us pick m in such a way that

$$P(T(\mathbf{n}, -\mathbf{m}) < T(\mathbf{0}, -\mathbf{m})) \geq \frac{1}{4} \tag{7}$$

while also (6) holds. Write B for the event in (7). The expectation $E[T(\mathbf{n}, -\mathbf{m}) - T(\mathbf{0}, -\mathbf{m})]$ may be decomposed as

$$\begin{aligned}
E[T(\mathbf{n}, -\mathbf{m}) - T(\mathbf{0}, -\mathbf{m})] &= E[T(\mathbf{n}, -\mathbf{m}) - T(\mathbf{0}, -\mathbf{m}) \,|B]P(B) \\
&\quad + E[T(\mathbf{n}, -\mathbf{m}) - T(\mathbf{0}, -\mathbf{m}) \,|\neg B]P(\neg B) \\
&\leq E[T(\mathbf{n}, -\mathbf{m}) - T(\mathbf{0}, -\mathbf{m}) \,|\neg B]P(\neg B) \\
&\leq \frac{3}{4} E[T(\mathbf{n}, -\mathbf{m}) - T(\mathbf{0}, -\mathbf{m}) \,|\neg B] \\
&\leq \frac{3}{4} E[T(\mathbf{n}, \mathbf{0}) | \neg B] \\
&\leq \frac{3}{4}(1 + 4\varepsilon)n\mu
\end{aligned}$$

where the second-to-last inequality is due to the triangle inequality $T(\mathbf{n}, -\mathbf{m}) \leq T(\mathbf{n}, \mathbf{0}) + T(\mathbf{0}, -\mathbf{m})$, and the last one uses (5). For small ε, this contradicts (6), so the proof is complete. □

5 The case $\lambda \neq 1$

Let us move on to the case when $\lambda \neq 1$, that is, when the type 2 infection has a different intensity than type 1. It then seems unlikely that the kind of equilibrium which is necessary for infinite coexistence to occur would persist in the long run. However, this part of Conjecture 3.3 is not proved. The best result to date is the following theorem from Häggström and Pemantle (2000).

Theorem 5.1 *For any $d \geq 2$, we have $P^{1,\lambda}(G) = 0$ for all but at most countably many values of λ.*

We leave it to the reader to decide whether this is a very strong or a very weak result: it is very strong in the sense of showing that infinite coexistence has probability 0 for (Lebesgue)-almost all λ, but very weak in the sense that infinite coexistence is not ruled out for any given λ.

The result may seem a bit peculiar at first sight and we will spend some time explaining where it comes from and where the difficulties arise when one tries to strengthen it. Indeed, as formulated in Conjecture 3.3, the belief is that the set $\{\lambda : P^{1,\lambda}(G) > 0\}$ in fact consists of the single point $\lambda = 1$, but Theorem 5.1 only asserts that the set is countable.

First note that, by time-scaling and symmetry, we have $P^{1,\lambda}(G) = P^{1,1/\lambda}(G)$ and hence it is enough to consider $\lambda \leq 1$. An essential ingredient in the proof of Theorem 5.1 is a coupling of the two-type processes $\{P^{1,\lambda}\}_{\lambda\in(0,1]}$ obtained by associating two independent exponential mean 1 variables $\tau_1(e)$ and $\tau_2'(e)$ to each edge $e \in \mathbb{Z}^d$ and then letting the type 2 passage time at parameter value λ be given by $\tau_2(e) = \lambda^{-1}\tau_2'(e)$ and the type 1 time (for any λ) by $\tau_1(e)$. Write Q for the probability measure underlying this coupling and let G^λ be the event that infinite coexistence occurs at parameter value λ. Theorem 5.1 is obtained by showing that

$$\begin{array}{l}\text{with } Q\text{-probability 1 the event } G^\lambda \text{ occurs}\\ \text{for at most one value of } \lambda \in (0,1].\end{array} \tag{8}$$

Hence, $Q(G^\lambda)$ can be positive for at most countably many λ, and Theorem 5.1 then follows by noting that $P^{1,\lambda}(G) = Q(G^\lambda)$.

But why is (8) true? Let G_i^λ be the event that the type i infection grows unboundedly at parameter value λ. Then the coupling defining Q can be shown to be monotone in the sense that G_1^λ is decreasing in λ – that is, if G_1^λ occurs then $G_1^{\lambda'}$ occurs for all $\lambda' < \lambda$ as well – and G_2^λ is increasing in λ. This kind of monotonicity of the coupling is crucial for proving (8), as is the following result, which asserts that, on the event that the type 2 infection survives, the total infected set in a two-type process with distribution $P^{1,\lambda}$, where $\lambda < 1$, grows to a first approximation like a one-type process with intensity λ. More precisely, the speed of the growth in the two-type process is determined by the weaker type 2 infection type. We take $\tilde{S}_t^i$ to denote the union of all unit cubes centered at points in S_t^i and A is the limiting shape for a one-type process with rate 1.

Theorem 5.2 *Consider a two-type process with distribution $P^{1,\lambda}$ for some $\lambda \leq 1$. On the event G_2 we have, for any $\varepsilon > 0$, that almost surely*

$$(1-\varepsilon)\lambda A \subset \frac{\tilde{S}_t^1 \cup \tilde{S}_t^2}{t} \subset (1+\varepsilon)\lambda A$$

for large t.

Theorem 5.1 follows readily from this result and the monotonicity properties of the coupling Q. Indeed, fix $\varepsilon > 0$ and suppose G^λ occurs. Then Theorem 5.2 guarantees that on level λ the type 1 infection is eventually contained in $(1+\varepsilon)\lambda tA$, a conclusion that extends to all $\lambda' > \lambda$, because increasing the type 2 infection rate does not help type 1. On the other hand, for any $\lambda' > \lambda$ we get on level λ' that the union of the two infections will – again by Theorem 5.2 – eventually contain $(1-\varepsilon)\lambda' tA$, so by taking ε sufficiently small we see that the type 1 infection is strangled on level λ', implying (8), and Theorem 5.1 follows.

We will not prove Theorem 5.2, but mention that the hard work in proving it lies in establishing a certain key result (Proposition 2.2 in Häggström and Pemantle (2000)) that asserts that if the strong infection type reaches outside $(1+\varepsilon)\lambda tA$ infinitely often, then the weak type is doomed. The proof of this uses geometrical arguments, the most important ingredient being a certain spiral construction, emanating from the part of the strong of infection reaching beyond $(1+\varepsilon)\lambda tA$, and designed to allow the strong type to completely surround the weak type before the weak type catches up from inside.

How would one go about to strengthen Theorem 5.1 and rule out infinite coexistence for all $\lambda \neq 1$? One possibility would be to try to derive a contradiction with Theorem 5.2 from the assumption that the strong infection type grows unboundedly. For instance, intuitively it seems likely that the strong type occupying a positive fraction of the boundary of the infected set would cause the speed of the growth to exceed the speed prescribed by the weak infection type. This type of argument is indeed used in Garet and Marchand (2007) to show, for $d = 2$, that on the event of infinite coexistence the fraction of infected sites occupied by the strong infection will tend to 0 as $t \to \infty$. This feels like a strong indication that infinite coexistence does not happen.

Another approach to strengthening Theorem 5.1 in order to obtain the only-if direction of Conjecture 3.3 is based on the observation that, since coexistence represents a power balance between the infections, it is reasonable to expect that $P^{1,\lambda}(G)$ decreases as λ moves away from 1. We may formulate that intuition as a conjecture:

Conjecture 5.3 *For the two-type Richardson model on $\mathbb{Z}^d$ with $d \geq 2$, we have, for $\lambda < \lambda' \in (0,1]$, that $P^{1,\lambda}(G) \leq P^{1,\lambda'}(G)$.*

A confirmation of this conjecture would, in combination with Theorem 5.1, clearly establish the only-if direction of Conjecture 3.3: If $P^{1,\lambda}(G) > 0$ for some $\lambda < 1$, then, according to Conjecture 5.3, we would have $P^{1,\lambda'}(G) > 0$ for all $\lambda' \in (\lambda, 1]$ as well. But the interval $(\lambda, 1]$ is uncountable, yielding a contradiction to Theorem 5.1.

Although Conjecture 5.3 might seem close to obvious, it has turned out to be very difficult to prove. A natural first attempt would be to use coupling. Consider for instance the coupling Q described above. As pointed out, the

events G_1^λ and G_2^λ that the individual infections grow unboundedly at parameter value λ are then monotone in λ, but one of them is increasing and the other is decreasing, so monotonicity of their intersection G^λ does not follow. Hence more sophisticated arguments are needed.

Observing how our colleagues react during seminars and corridor chat, we have noted that it is very tempting to go about trying to prove Conjecture 5.3 by abstract and "easy" arguments, here meaning arguments that do not involve any specifics about the geometry or graph structure of $\mathbb{Z}^d$. To warn against such attempts, Deijfen and Häggström (2006:2) constructed graphs on which the two-type Richardson model fails to exhibit the monotonicity behavior predicted in Conjecture 5.3. Let us briefly explain the results.

The dynamics of the two-type Richardson model can of course be defined on graphs other than the $\mathbb{Z}^d$ lattice. For a graph $\mathcal{G}$, write $\mathrm{Coex}(\mathcal{G})$ for the set of all $\lambda \geq 1$ such that there exists a finite initial configuration (ξ_1, ξ_2) for which the two-type Richardson model with infection intensities 1 and λ started from (ξ_1, ξ_2) yields infinite coexistence with positive probability. Note that, by time-scaling and interchange of the infections, coexistence is possible at parameter value λ if and only if it is possible at λ^{-1}, so no information is lost by restricting to $\lambda \geq 1$. In Deijfen and Häggström (2006:2) examples of graphs $\mathcal{G}$ are given that demonstrate that, among others, the following kinds of coexistence sets $\mathrm{Coex}(\mathcal{G})$ are possible:

(i) $\mathrm{Coex}(\mathcal{G})$ may be an interval (a, b) with $1 < a < b$.

(ii) For any positive integer k the set $\mathrm{Coex}(\mathcal{G})$ may consist of exactly k points.

(iii) $\mathrm{Coex}(\mathcal{G})$ may be countably infinite.

All these phenomena show that the monotonicity suggested in Conjecture 5.3 fails for general graphs. However, a reasonable guess is that Conjecture 5.3 is true on transitive graphs. Indeed, all counterexamples provided by Deijfen and Häggström are highly non-symmetric (one might even say ugly) with certain parts of the graph being designed specifically with propagation of type 1 in mind, while other parts are meant for type 2. We omit the details.

6 Unbounded initial configurations

Let us now go back to the $\mathbb{Z}^d$ setting and describe some results from our most recent paper, Deijfen and Häggström (2007), concerning the two-type model with unbounded initial configurations. Roughly, the model will be started from configurations where one of the infections occupies a single site in an infinite "sea" of the other type. The dynamics is as before and also the question at issue is the same: can both infection types simultaneously infect infinitely many sites? With both types initially occupying infinitely many

sites the answer is (apart from in particularly silly cases) obviously yes, so we will focus on configurations where type 1 starts with infinitely many sites and type 2 with finitely many – for simplicity only one. The question then becomes whether type 2 is able to survive.

To describe the configurations in more detail, write $(x_1, \ldots, x_d)$ for the coordinates of a point $x \in \mathbb{Z}^d$, and define $\mathcal{H} = \{x : x_1 = 0\}$ and $\mathcal{L} = \{x : x_1 \leq 0 \text{ and } x_i = 0 \text{ for } i = 2, \ldots, d\}$. We will consider the following starting configurations.

$$\begin{array}{rl} I(\mathcal{H}): & \text{all points in } \mathcal{H}\backslash\{\mathbf{0}\} \text{ are type 1 infected and} \\ & \mathbf{0} \text{ is type 2 infected, and} \\ I(\mathcal{L}): & \text{all points in } \mathcal{L}\backslash\{\mathbf{0}\} \text{ are type 1 infected and} \\ & \mathbf{0} \text{ is type 2 infected.} \end{array} \tag{9}$$

Interestingly, it turns out that the set of parameter values for which type 2 is able to grow indefinitely is slightly different for these two configuration. First note that, as before, we may restrict to the case $\lambda_1 = 1$. Write $P^{1,\lambda}_{\mathcal{H},\mathbf{0}}$ and $P^{1,\lambda}_{\mathcal{L},\mathbf{0}}$ for the distribution of the process started from $I(\mathcal{H})$ and $I(\mathcal{L})$ respectively and with type 2 intensity λ. The following result, where G_2 denotes the event that type 2 grows unboundedly, is proved in Deijfen and Häggström (2007).

Theorem 6.1 *For the two-type Richardson model in $d \geq 2$ dimensions, we have*

(a) $P^{1,\lambda}_{\mathcal{H},\mathbf{0}}(G_2) > 0$ *if and only if* $\lambda > 1$;

(b) $P^{1,\lambda}_{\mathcal{L},\mathbf{0}}(G_2) > 0$ *if and only if* $\lambda \geq 1$.

In words, a strictly stronger type 2 infection will be able to survive in both configurations, but, when the infections have the same intensity, type 2 can survive only in the configuration $I(\mathcal{L})$.

The proof of the if-direction of Theorem 6.1 (a) is based on a lemma stating roughly that the speed of a hampered one-type process, living only inside a tube which is bounded in all directions except one, is close to the speed of an unhampered process when the tube is large. For a two-type process started from $I(\mathcal{H})$, this lemma can be used to show that, if the strong type 2 infection at the origin is successful in the beginning of the time course, it will take off along the x_1-axis and grow faster than the surrounding type 1 infection inside a tube around the x_1-axis, thereby escaping eradication. The same scenario – that the type 2 infection rushes away along the x_1-axis – can, by different means, be proved to have positive probability in a process with $\lambda = 1$ started from $I(\mathcal{L})$. Infinite growth for type 2 when $\lambda < 1$ is ruled out by the key proposition from Häggström and Pemantle (2000) mentioned in Section 3. Proving that type 2 cannot survive in a process with $\lambda = 1$ started from $I(\mathcal{H})$ is the most tricky part. The idea is basically to divide $\mathbb{Z}^d$ in different

levels, the l-th level being all sites with x_1-coordinate l, and then show that the expected number of type 2 infected sites at level l is constant and equal to 1. It then follows from a certain comparison with a one-type process on each level combined with an application of Levy's 0-1 law that the number of type 2 infected sites at the l-th level converges almost surely to 0 as $l \to \infty$.

Finally we mention a question formulated by Itai Benjamini as well as by an anonymous referee of Deijfen and Häggström (2007). We have seen that, when $\lambda = 1$, the type 2 infection at the origin can grow unboundedly from $I(\mathcal{L})$ but not from $I(\mathcal{H})$. It is then natural to ask what happens if we interpolate between these two configurations. More precisely, instead of letting type 1 occupy only the negative x_1-axis (as in $I(\mathcal{L})$), we let it occupy a cone of constant slope around the same axis. The question then is what the critical slope is for this cone such that there is a positive probability for type 2 to grow unboundedly. That type 2 cannot survive when the cone occupies the whole left half-space follows from Theorem 6.1, as this situation is equivalent to starting the process from $I(\mathcal{H})$. It seems likely, as suggested by Itai Benjamini, that this is actually also the critical case, that is, infinite growth for type 2 most likely have positive probability for any smaller type 1 cone. This however remains to be proved.

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