

JOCHEN BLATH, FELIX HERMANN & MICHEL REITMEIER **The Contact Process with switching** Volume 12 (2023), p. 135-154. https://doi.org/10.5802/msia.35

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MathematicS In Action est membre du Centre Mersenne pour l'édition scientifique ouverte http://www.centre-mersenne.org/ e-ISSN : 2102-5754

The Contact Process with switching

Jochen Blath * Felix Hermann ** Michel Reitmeier ***

* Goethe-Universität Frankfurt am Main, Robert-Mayer Str. 10, 60629 Frankfurt, Germany *E-mail address*: blath@math.uni-frankfurt.de

** Goethe-Universität Frankfurt am Main, Robert-Mayer Str. 10, 60629 Frankfurt, Germany *E-mail address*: hermann@math.uni-frankfurt.de

*** Goethe-Universität Frankfurt am Main, Robert-Mayer Str. 10, 60629
 Frankfurt, Germany E-mail address: reitmeier@math.uni-frankfurt.de.

Abstract

In this paper, we introduce a type switching mechanism for the Contact Process. That is, we allow the individual particles/sites to switch between two (or more) types independently of one another, and the different types may exhibit specific infection and recovery dynamics. Such type switches can e.g. be motivated from biology, where "phenotypic switching" is common among micro-organisms. Our framework includes as special cases systems with switches between "active" and "dormant" states (the Contact Process with dormancy, CPD), and the Contact Process in a randomly evolving environment (CPREE) introduced by Broman (2007). The "standard" multi-type Contact Process (without type-switching) can also be recovered as a limiting case.

After constructing the process from a graphical representation, we first establish basic properties that are mostly analogous to the classical Contact Process. We then provide couplings between several variants of the system, obtaining sufficient conditions for the existence of a phase transition. Further, we investigate the effect of the switching parameters on the critical value of the system by providing rigorous bounds obtained from the coupling arguments as well as numerical and heuristic results. Finally, we investigate scaling limits for the process as the switching parameters tend to 0 (slow switching regime) resp. ∞ (fast switching regime). We conclude with a brief discussion of further model variants and questions for future research.

1. The Contact Process with Switching

1.1. Model description

We introduce the Contact Process with switching (CPS) as a continuous-time Markov process on the grid $S := \mathbb{Z}^d$ as follows. At each time $t \ge 0$, the state of the process is a function $\xi_t : \mathbb{Z}^d \to F$, where

$$F := \{(0, a), (0, d), (1, a), (1, d)\}$$

describes the possible states of the individuals at each grid point. (We call this the map-based representation.) The first component of an element from F indicates whether the corresponding site (or "particle") is infected ("1") or healthy ("0"), and the second component refers to the type of the particle (say, "a" or "d"). The dynamics is as follows: Independently of all other particles an infected particle of type a (resp. d) recovers with rate δ_a (resp. δ_d), and any infected particle of type $\tau_1 \in \{a, d\}$ infects a healthy neighbour of type $\tau_2 \in \{a, d\}$ at rate $\lambda_{\tau_1 \tau_2} \geq 0$. Finally, healthy type a particles switch into type d at rate σ_0 , and from d to a at rate ρ_0 (with similar rates σ_1 and ρ_1 for the infected individuals). See Figure 1.1 for a visualization of these

Keywords: Contact Process, Coupling, Dormancy, Random environment, Switching. 2020 Mathematics Subject Classification: 60K35, 92D25, 92D30.

transition rates at a given site x, where we define

$$n_{1,a}(x,\xi) := |\{z \in \mathcal{N} : \xi(x+z) = (1,a)\}|$$

as the number of infected type a individuals in the (finite) neighbourhood of x given by $\mathcal{N} \subset \mathbb{Z}^d$, and correspondingly,

$$n_{1,d}(x,\xi) := |\{z \in \mathcal{N} : \xi(x+z) = (1,d)\}|$$

for the number of infected neighbours of x of type d.

For simplicity, we restrict ourselves here to finite neighbourhoods \mathcal{N} , so that the CPS $\{\xi_t\}$ can readily be constructed e.g. from a graphical representation (see Section 2.1 for details). Further, we assume $\sigma_0 = \sigma_1 = \sigma$ and $\rho_0 = \rho_1 = \rho$ unless stated otherwise.



FIGURE 1.1. Flip rates of the Contact Process with switching (CPS).

Finally, note that one could also work on more general graphs S than just \mathbb{Z}^d , e.g. infinite regular trees. However, for notational convenience we restrict our results to integer lattices.

The above abstract type switching mechanism can be motivated for example by *phenotypic* switches, which in a microbial context may result from stochastic or responsive changes of cell properties. Phenotypic switching mechanisms come in many different forms and are ubiquitous in bacterial communities, where they foster persistence and coexistence, cf. e.g. [2, 18, 19]. They are also exhibited by cancer cells, e.g. in response to immunotherapy [1, 10], where they can contribute to treatment failure. In ecology, in the presence of fluctuating environments, such switching mechanisms can act as efficient bet-hedging strategies, contributing to the long term survival of populations. Mathematically, such strategies have recently been investigated e.g. in branching process based models [4, 9]. In these papers, optimal switching mechanisms, depending on the distribution of the random environment, have been established.

The special case of microbial *dormancy* [17] may be seen as drastic form of such a type switch, where micro-organisms enter reversible states with low or vanishing metabolic activity. In such a dormant stage, the individual is shielded form adverse environmental conditions, but potentially also from anti-microbial treatment [11], or from intra- and inter-species competition [5]. Recently, dormancy has also been investigated as an efficient host-strategy to curb virus epidemics, cf. e.g. [6, 14].

Yet, in classical interacting particle systems, type switches or dormancy seem to be rather new concepts. To gain an understanding, in idealized scenarios, of the benefits for survival of populations exhibiting type switching, and in particular dormancy, motivated the present study.

1.2. Special cases

Obviously, a *first special case* of the CPS model is given by the classical Contact Process, denoted by CP, with parameters λ and δ chosen as

$$\lambda_{aa} = \lambda_{ad} = \lambda_{dd} = \lambda_{da} = \lambda$$
 and $\delta_a = \delta_d = \delta$.

A second special case can be obtained from choosing

$$\lambda_{aa} = \lambda$$
, and $\lambda_{ad} = \lambda_{da} = \lambda_{dd} = 0$.

This process, in which the type d particles neither infect neighbours nor get infected, will be called the Contact Process with (host) dormancy (CPD). This special case served as motivation for the notation a (for "active state") and d (for "dormant state"). See Figure 1.2 (left) for a visualization of the corresponding flip rates.

Intuitively, the incorporation of dormancy should lead to an increase of the critical infection rate of the process (compared to the classical Contact Process), and this is indeed the case as we will see below. However, the strength of the observed effect will depend on the particular model parameters, and here further natural parameter choices arise. For example, if one interprets (microbial) host dormancy as a state of vanishing metabolic activity, then this suggests that the recovery rate δ_d should be close to zero (the host does not recover from an infection while dormant). In contrast, if one considers (human) dormancy from an epidemiological point of view as periods of reduced social contacts, then during such "dormant states", recovery should still be possible (while the ability to infect neighbours should be 0). Clearly, the second strategy seems more efficient for the hosts since it allows recovery during dormancy. This intuition will be confirmed later on (cf. Remark 3.5).

Naturally, one could also consider dormancy on the level of infectors instead of hosts. For example, when the activity of the infector can be linked to a specific state of the host, such as the reactivation of Herpes viruses which can be triggered by periods of stress of the host (cf. [16]), then the CPS model could still be applicable. However, if the dormancy state of an infector switches independently of the state of the host, this will lead to yet another model class outside the scope of the present paper. A possible corresponding "CPID" model will briefly be discussed in Section 4.3.

A third special case of our model is given by the Contact Process in a randomly evolving environment (CPREE) investigated by Broman [8]. Here, only the recovery rates depend on the type of the particle in question. Denoting by $n_{(1)}(x,\xi)$ the number of infected individuals in the \mathcal{N} -neighbourhood of x in ξ , its flip rates are given by Figure 1.2 (right).

In this model, the states a and d can be interpreted as the two states of a random environment which evolves independently for each individual whose ability for recovery is governed by the state of the environment, while the susceptibility for infection remains unaffected.

2. Basic properties of the Contact Process with switching

2.1. Graphical construction and self-duality

We introduce a graphical construction of the CPS, which is the key to establish several couplings within our model and the CP. Based on this construction, we also find that the CPS has a dual process within the same class of processes and even obtain an exact *self-duality* in the case $\lambda_{ad} = \lambda_{da}$.

The construction is a simple extension of the classical graphical representation for the basic CP given by Harris [13] and relies on families of independent Poisson processes driving infection and recovery events, combined with an idea from the construction of the CPREE [25] to determine the activity states of the particles.



FIGURE 1.2. Left: Flip rates of the Contact Process with dormancy (CPD). Here, δ_d can be either positive or zero. Right: The Contact Process in a randomly evolving environment (CPREE).

Recalling the *map*-based representation $\{\xi_t\}$ from the introduction, note that here it will be convenient to work with the equivalent *set*-based representation involving

$$\mathcal{A}_t := \{ x \in \mathbb{Z}^d \mid \xi_t(x)_2 = a \},\$$

the set of all active sites at time t, and

$$\mathcal{X}_t := \{ x \in \mathbb{Z}^d \mid \xi_t(x)_1 = 1 \},\$$

the set of all infected sites at time t, where $\xi_t(x)_i$ denotes the *i*th component of $\xi_t(x)$.

To carry out the construction, let the following families of independent Poisson Point Processes (PPPs) be defined on a common probability space $(\Omega, \mathcal{F}, \mathbb{P})$:

- The (potential) recovery events, given by PPPs $U^{a,x} = \{U_n^{a,x}, n \ge 1\}, U^{d,x} = \{U_n^{d,x}, n \ge 1\}$, for every $x \in \mathbb{Z}^d$ with rates δ_a and δ_d , respectively.
- The (potential) type switching events, given by the set of PPPs $V^{x,a\to d}$ and $V^{x,d\to a}$ for every $x \in \mathbb{Z}^d$ with rates σ and ρ , respectively.
- The potential *infection events*, given by PPPs $T^{x \to y,aa}$, $T^{x \to y,ad}$, $T^{x \to y,da}$ and $T^{x \to y,dd}$ for every pair of neighbours $(x, y) \in (\mathbb{Z}^d)^2$ with rates λ_{aa} , λ_{ad} , λ_{da} and λ_{dd} , respectively.

For convenience we denote these collections of PPPs by \mathcal{U}, \mathcal{V} and \mathcal{T} , respectively.

On the space time grid $\mathbb{Z}^d \times [0, \infty)$ we draw δ_a 's, δ_d 's, σ 's or ρ 's for every arrival time in $U^{a,x}$, $U^{d,x}$, $V^{x,a \to d}$ or $V^{x,d \to a}$, respectively. A δ_a (or δ_d) at (x, t) indicates that the particle x recovers from infection if it is infected and active (or dormant) at time t. The σ 's and ρ 's mark changes from active to dormant and vice versa. Moreover, we draw arrows of type $\tau \in \{aa, ad, da, dd\}$ from (x, t) to (y, t), whenever $t \in T^{x \to y, \tau}$. In particular, an arrow of type aa from (x, t) to (y, t) signifies that y gets infected by x at time t if both particles are active and x is infected. The graphical construction is illustrated in Figure 2.1.

Now, we can construct the process of active particles, $(\mathcal{A}_t)_{t\geq 0}$, by letting $\mathcal{V}_{x,t} = (V^{x,d\to a} \cup V^{x,a\to d}) \cap [0,t]$, the switching times of x until time t, and writing

$$\mathcal{A}_t := \Big\{ x \in \mathbb{Z}^d \, \Big| \, \mathcal{V}_{x,t} = \emptyset \land x \in \mathcal{A}_0 \quad \text{or} \quad \max(\mathcal{V}_{x,t}) \in V^{x,d \to a} \Big\},$$

such that a particle is active, iff either it was active in the beginning and no switching occurred or the last switching event before t is of type $d \to a$. Finally, this can be used to define the notion of *infection paths* similarly to [25, Definition 2.3]. Note that we desist from their notion of *active* paths to avoid confusion.

For $x, y \in \mathbb{Z}^d$ we say there exists an infection path from (x, t_0) to (y, t_1) if $t_0 < t_1$ and if there exists a time sequence $s_0 = t_0 < s_1 < s_2 < \cdots < s_{\ell+1} = t_1$ with $\ell \in \mathbb{N}$ and a sequence of particles $x_0 = x, x_1, x_2, \ldots, x_\ell = y$ such that:

- (i) For $i \in [\ell]$ there exists at time s_i an arrow from x_{i-1} to x_i and the activity states of x_{i-1} and x_i at time s_i match with the type of the arrow, i.e. if both particles are for example active at time s_i then the arrow has to be of type aa.
- (ii) For any δ_a at $(\tilde{x}, t) \in \{x_i\} \times (s_i, s_{i+1})$ it holds $\tilde{x} \notin \mathcal{A}_t$.
- (iii) For any δ_d at $(\tilde{x}, t) \in \{x_i\} \times (s_i, s_{i+1})$ it holds $\tilde{x} \in \mathcal{A}_t$.

Consequently, for t > 0 we define

 $\mathcal{X}_t := \{ y \in \mathbb{Z}^d : \text{for } x \in \mathcal{X}_0 \text{ exists an infection path from } (x, 0) \text{ to } (y, t) \},\$

the set of infected particles at time t. The pair $(\mathcal{X}_t, \mathcal{A}_t)_t$ now gives an explicit construction of the CPS via the *set*-based representation.

To see that the process is indeed well-defined, one may consider the so-called *interaction graph* of the CPS at time t, that is the graph on \mathbb{Z}^d in which two sites $x, y \in \mathbb{Z}^d$ are connected by an edge iff there exists a time $s \leq t$ at which there is either an arrow from x to y or from y to x. In the same way as in [3, (2.1) Theorem] we can then find a finite time $t_0 > 0$ such that the interaction graph at t_0 splits almost surely into clusters of finite size.

Translation invariance as well as the Feller property follow analogously to [3] (cf. (2.2) Corollary and (2.3) Corollary).

Remark 2.1.

- (1) To specify initial configurations of infected particles $I \subset \mathbb{Z}^d$ and of active particles $A \subset \mathbb{Z}^d$, i.e. choosing $\mathcal{X}_0 = I$ and $\mathcal{A}_0 = A$, we use the notation $\mathcal{X}_t^{I,A}$ and $\mathcal{A}_t^{I,A}$ as well as $\xi_t^{I,A}$, whereas by a probability measure in the superscript we specify initial distributions.
- (2) Strictly speaking, the above defined process is left- but not right-continuous (cf. [3, p. 127]). Nevertheless, the left- and right-continuous versions are almost surely equal at any fixed time t because the process has only countably many jumps. Hence, if we insist on càdlàg paths, we simply can redefine our process as the corresponding right-continuous version.

From this graphical construction, by the usual thinning and coupling arguments, the following basic properties can easily be established:

Theorem 2.2. Let $(\mathcal{X}_t, \mathcal{A}_t)_{t\geq 0}$ and $(\mathcal{X}_t, \mathcal{A}_t)_{t\geq 0}$ be two CPS with coupled activity states.

(1) If $\mathcal{X}_0 = \widetilde{\mathcal{X}}_0$ and the respective parameters satisfy $\widetilde{\lambda}_{\tau} \ge \lambda_{\tau} \ge 0$ for each $\tau \in \{aa, ad, da, dd\}$ and $0 \le \widetilde{\delta}_{\bullet} \le \delta_{\bullet}$ for every $\bullet \in \{a, d\}$, then there exists a coupling of (\mathcal{X}_t) and $(\widetilde{\mathcal{X}}_t)$ such that

$$\mathcal{X}_t \subseteq \mathcal{X}_t,$$
 for all $t \ge 0.$ (monotonicity)

(2) If
$$I_1, I_2 \subseteq \mathbb{Z}^d$$
, then
 $\mathcal{X}_t^{I_1, \mathcal{A}_0} \cup \mathcal{X}_t^{I_2, \mathcal{A}_0} = \mathcal{X}_t^{I_1 \cup I_2, \mathcal{A}_0}$, for all $t \ge 0$. (additivity)

(3) If $I \subseteq J \subseteq \mathbb{Z}^d$, then

 $\mathcal{X}_t^{I,\mathcal{A}_0} \subseteq \mathcal{X}_t^{J,\mathcal{A}_0}, \qquad \qquad for \ all \ t \ge 0. \quad (attractivity)$

As for the basic CP and the CPREE we obtain the dual process starting at a fixed time t and letting the process run backwards in time, denoting $\hat{\mathcal{X}}_s^t = \mathcal{X}_{t-s}$ and $\hat{\mathcal{A}}_s^t = \mathcal{A}_{t-s}$ for $0 \le s \le t$. By reverting all arrows in the graphical construction of the forward-time version, one verifies that $(\hat{\mathcal{X}}_s^t, \hat{\mathcal{A}}_s^t)_s$ follows almost the same dynamics as $(\mathcal{X}_s, \mathcal{A}_s)_s$, except for the roles of *ad*-arrows and *da*-arrows being switched. Hence, it holds:

Theorem 2.3 (Duality). The dual process of a CPS $(\mathcal{X}_t, \mathcal{A}_t)_t$ is also distributed as a CPS with the same parameters as $(\mathcal{X}_t, \mathcal{A}_t)_t$ except for λ_{ad} and λ_{da} being swapped. Consequently, if it holds that $\lambda_{ad} = \lambda_{da}$, then $(\mathcal{X}_t, \mathcal{A}_t)$ is self-dual.

In particular, all special cases mentioned in the introduction are self-dual. The construction of the dual process is also illustrated in Figure 2.1. Further, from the picture it follows immediately that the so called *duality relation* holds, namely

$$\{\mathcal{X}_t^{I,A} \cap J \neq \emptyset\} = \{\mathcal{X}_s^{I,A} \cap \widehat{\mathcal{X}}_s^{t,J,\mathcal{A}_t} \neq \emptyset\} = \{I \cap \widehat{\mathcal{X}}_t^{t,J,\mathcal{A}_t} \neq \emptyset\} \quad \text{for all } 0 \le s \le t.$$

FIGURE 2.1. CPS and the dual process. The CPS $\mathcal{X}_t^{I,A}$ started with $I = \{2,3\}$ and $A = \{2,4,5,6\}$ running upwards, and the dual $\hat{\mathcal{X}}_s^{t,J,A_t}$ started from time t in $J = \{4\}$ and $\mathcal{A}_t = \{2,3,6\}$ running downwards. The states (0,a), (0,d), (1,a) and (1,d) are depicted with grey solid, grey dotted, black solid and black dashed lines, respectively.

2.2. Stationary distributions and phase transition

In what follows, we will make use of both the map-based as well as the set-based representation for notational convenience.

Since type switches are independent of the infection and recovery dynamics, it is easy to verify that \mathcal{A}_t converges in distribution to its unique stationary distribution π_{α} , which lets any site in \mathbb{Z}^d be active with probability $\alpha := \frac{\rho}{\sigma + \rho}$ and dormant otherwise, independently of each other. Thus,

$$\mu^{triv} := \delta_{\{0\}^{\mathbb{Z}^d}} \otimes \pi_\alpha$$

is a *trivial* stationary distribution of (ξ_t) . We now investigate the existence of, and convergence into, further stationary distributions. We denote by " \Rightarrow " weak convergence with respect to the product topology generated by the open cylinder sets. Note that the cylinder functions lie dense in the set of continuous functions on $F^{\mathbb{Z}^d}$ and can be written as linear combinations of indicator functions (cf. [3, p. 103]). Hence, weak convergence is equivalent to convergence of finite dimensional distributions, i.e. $\xi_t \Rightarrow \xi_\infty$ as $t \to \infty$ if and only if for any $m \in \mathbb{N}, x_1, \ldots, x_m \in \mathbb{Z}^d$ and $i_1, \ldots, i_m \in F$ we have

$$\mathbb{P}(\xi_t(x_1) = i_1, \dots, \xi_t(x_m) = i_m) \xrightarrow{t \to \infty} \mathbb{P}(\xi_\infty(x_1) = i_1, \dots, \xi_\infty(x_m) = i_m).$$

Theorem 2.4. Let (ξ_t^1) be a CPS with initial distribution $\delta_{\{1\}\mathbb{Z}^d} \otimes \pi_\alpha$. Then, $\xi_t^1 \Rightarrow \xi_\infty^1$ as $t \to \infty$, where $\mathcal{L}(\xi_\infty^1)$ is a stationary distribution of (ξ_t) .

The proof of this theorem will be very similar to that of [3, (2.7) Theorem, p. 123] and will make use of the following lemma (cf. [3, (2.8) Lemma, p. 123]):

Lemma 2.5. Let $\xi_t^{\mathbb{1}}$ as specified above, then for any sets $H, A, D \subseteq \mathbb{Z}^d$ with $A \cap D = \emptyset$ the function

$$\Phi_t(H, A, D) := \mathbb{P}(H \subseteq (\mathcal{X}_t^{\mathbb{1}})^c | A \subseteq \mathcal{A}_t, D \subseteq (\mathcal{A}_t)^c)$$

is increasing in t.

Proof. This follows from a restarting argument combined with the attractivity established in Theorem 2.2(3): Let $H, A, D \subseteq \mathbb{Z}^d$ with $A \cap D = \emptyset$ and s, t > 0. Then,

$$\Phi_{t+s}(H, A, D) = \mathbb{P}(H \subseteq (\mathcal{X}_{t+s}^{\mathbb{1}})^c | A \subseteq \mathcal{A}_{t+s}, D \subseteq (\mathcal{A}_{t+s})^c)$$

$$= \mathbb{P}(H \subseteq (\mathcal{X}_t^{\mathcal{X}_s})^c | A \subseteq \mathcal{A}_t^{\mathcal{A}_s}, D \subseteq (\mathcal{A}_t^{\mathcal{A}_s})^c)$$

$$\geq \mathbb{P}(H \subseteq (\mathcal{X}_t^{\mathbb{1}})^c | A \subseteq \mathcal{A}_t^{\mathcal{A}_s}, D \subseteq (\mathcal{A}_t^{\mathcal{A}_s})^c)$$

$$= \Phi_t(H, A, D),$$

where we used stationarity of (\mathcal{A}_t) in the last step.

Proof of Theorem 2.4. We show convergence of the finite dimensional distributions (depending on finitely many sites). For this, let $m \in \mathbb{N}, x_1, \ldots, x_m \in \mathbb{Z}^d$ and $i_1, \ldots, i_m \in F$ be fixed. Now, denote by $H = \{x_k \mid 1 \leq k \leq m, i_{k,1} = 0\}$, the set of the x_k such that i_k is healthy, and define I, A, D accordingly. Then,

$$\mathbb{P}(\xi_t^{\mathbb{1}}(x_1) = i_1, \dots, \xi_t^{\mathbb{1}}(x_m) = i_m) \\ = \mathbb{P}(A \subseteq \mathcal{A}_t) \cdot \mathbb{P}(D \subseteq (\mathcal{A}_t)^c) \cdot \mathbb{P}(H \subseteq (\mathcal{X}_t^{\mathbb{1}})^c, I \subseteq \mathcal{X}_t^{\mathbb{1}} | A \subseteq \mathcal{A}_t, D \subseteq (\mathcal{A}_t)^c),$$

where the first two factors multiply to $\alpha^{|A|}(1-\alpha)^{|D|}$ and the third can be rewritten as a finite sum in terms of $\Phi_t(\bullet, A, D)$ using the inclusion-exclusion-formula:

$$\mathbb{P}(H \subseteq (\mathcal{X}_t^{\mathbb{1}})^c, I \subseteq \mathcal{X}_t^{\mathbb{1}} | A \subseteq \mathcal{A}_t, D \subseteq (\mathcal{A}_t)^c)$$

= $\Phi_t(H, A, D) - \mathbb{P}(H \subseteq (\mathcal{X}_t^{\mathbb{1}})^c, I \not\subseteq \mathcal{X}_t^{\mathbb{1}} | A \subseteq \mathcal{A}_t, D \subseteq (\mathcal{A}_t)^c)$
= $\Phi_t(H, A, D) - \mathbb{P}\left(\bigcup_{x \in I} \{H \cup \{x\} \subseteq (\mathcal{X}_t^{\mathbb{1}})^c\} \middle| A \subseteq \mathcal{A}_t, D \subseteq (\mathcal{A}_t)^c\right)$
= $\Phi_t(H, A, D) + \sum_{J \subseteq I, J \neq \emptyset} (-1)^{|J|} \Phi_t(H \cup J, A, D).$

Hence, Lemma 2.5 implies the desired convergence. The stationarity of the limiting distribution follows from the Feller property (cf. [3, (2.9) Lemma, p. 123]). \Box

We refer to μ^{triv} as the *lower* invariant distribution, and to $\mu^1 := \mathcal{L}(\xi_{\infty}^1)$ as the *upper* invariant distribution. Due to attractivity (Theorem 2.2), these provide the extreme cases of limiting distributions, starting without any or only with infected individuals respectively. Both distributions may or may not be distinct, which gives rise to the following notions of survival.

Definition 2.6 (Survival). A CPS (ξ_t) is said to survive, if $\mu^1 \neq \mu^{triv}$. Otherwise it goes extinct.

Remark 2.7 (Strong survival). By a duality argument (cf. [22, p. 36]) combined with stationarity of (\mathcal{A}_t) , it can be shown that the above notion of ("infinite") survival of the CPS is equivalent to finite survival of the dual, i.e.

$$\mathbb{P}(\widehat{\mathcal{X}}_t^{\{0\},\pi_\alpha} \neq \emptyset \text{ for all } t \ge 0) > 0.$$

For the CP on \mathbb{Z}^d it has been shown in [22, Theorem 2.25] that this is even equivalent to the generally more restrictive notion of *strong* survival, given as

$$\mathbb{P}(0 \in \mathcal{X}_t^{\{0\}, \pi_\alpha} \text{ infinitely often}) > 0,$$

which can be very well interpreted as "endemic" behaviour. This result, however, is entangled with the proof of *complete convergence*, i.e. the property that any stationary distribution arises as convex combination of the lower and the upper invariant distributions (cf. [22, Theorem 2.27]). We conjecture that both properties also hold for the CPS, but defer the proofs to future work.

Since the CPS is monotone in the parameters λ_{aa} , λ_{ad} , λ_{da} , λ_{dd} , δ_a , δ_d , we can consider a critical parameter λ^c_{\bullet} or δ^c_{\bullet} , for some \bullet , separating a survival from an extinction phase in the obvious way, while fixing all other parameters. The coupling results in Section 3.2 will give us some bounds on the critical parameters and ensure that a phase transition can occur in suitable cases, while there are also cases where a phase transition does not exist. See Remark 3.12 for more details.

3. Relation between CPS and CP

In this section we pursue two different approaches to compare the CPS with the classical CP. At first we discuss scaling limits in the switching rates. We show that for high σ and ρ the CPS is well approximated by a CP with "effective" rates λ^* and δ^* that can be computed explicitly from the model parameters, whereas for low σ and ρ the CPS is close to a CP in a static environment. Secondly, we give rigorous couplings between the CPS and suitable CPs in Section 3.2, establishing phase transitions. We will discuss various simulation results in Section 3.3.

3.1. Fast and slow switching limits

In this section, we will consider a rescaled CPS $(\xi_t^h)_{t\geq 0}$ whose switching rates σ, ρ are replaced by $h\sigma$ and $h\rho$, where we let h either go to ∞ or to 0, while the λ_{\bullet} remain fixed. (Mind the difference between ξ_t^1 here and ξ_t^1 of Theorem 2.4.) Note that for a CP (η_t) the product measure $\mathcal{L}(\eta_t) \otimes \pi_{\alpha}$ is for fixed t a distribution on

 $F^S = \{\{0,1\} \times \{a,d\}\}^{\mathbb{Z}^d}$

Theorem 3.1 (Fast switching). For $k \in \mathbb{N}$ let $(\xi_t^k)_{t\geq 0}$ be the rescaled CPS as above, with switching parameters σk , ρk , all other parameters fixed and initial infections and initially active sites given by $I, A \subseteq \mathbb{Z}^d$ respectively. Then, for each fixed $t \geq 0$,

$$\mathcal{L}(\xi_t^k) \Rightarrow \mathcal{L}(\eta_t) \otimes \pi_{\alpha}$$

as $k \to \infty$. Here, (η_t) is a CP with initial infections given by I, infection rate

$$\lambda^* := \alpha^2 \lambda_{aa} + \alpha (1 - \alpha) (\lambda_{ad} + \lambda_{da}) + (1 - \alpha)^2 \lambda_{dd}$$

and recovery rate

$$\delta^* := \alpha \delta_a + (1 - \alpha) \delta_d.$$

Proof. As before, we show convergence of the finite-dimensional distributions of (ξ_t^k) , that is, of the laws restricted to finite subsets of sites $S' \subset \mathbb{Z}^d$, $|S'| < \infty$.

Via the graphical construction we couple the random states $\xi_t^k, k \in \mathbb{N}$, in the following way. Let Γ be the graphical construction of $(\xi_s^0)_{0 \leq s \leq t}$ restricted to the sites $S_{\Gamma} \subseteq \mathbb{Z}^d$ possibly influencing S', i.e. denoting $x \rightsquigarrow_t y$ when there is a path (not necessarily an infection path) from (x, 0) to (y, t)

$$S_{\Gamma} := S' \cup \{ x \in \mathbb{Z}^{d} \mid \exists y \in S' : x \rightsquigarrow_{t} y \}.$$

Note that since the switching rates in this case are 0, there are no type switches present in Γ . By a time-reversal argument, S_{Γ} can be explored through the graphical construction: Starting at time t with infections at S', traversing backwards through arrows of any type and ignoring δ events, at time 0 we arrive at S_{Γ} (see Figure 3.1 (left) for an illustration). Hence, the cardinality of S_{Γ} is stochastically dominated by a Yule Process with birth-rate $\sum_{\bullet} \lambda_{\bullet}$. Thus, S_{Γ} is almost surely finite. Now, we iteratively construct (ξ_t^{k+1}) from (ξ_t^k) for $k \geq 0$ by adding a new pair of independent Poisson processes of rates σ and ρ to each line $s \in S_{\Gamma}$ providing additional switching events.

There are a.s. only finitely many arrows and δ -events in Γ , say at times $t_1, \ldots, t_{n_{\Gamma}}$. Then, the activity states $(\mathbb{1}_{\mathcal{A}_{t_i}}(s))_{i \in [n_{\Gamma}], s \in S_{\Gamma}}$ converge in distribution (conditional on Γ) as $k \to \infty$ to a family of independent Bernoulli- α random variables, also independent of the initial activities. This is illustrated in Figure 3.1 (right): Increasing the rates, on any line segment $(t_i, t_{i+1}]$ for any site s the law of $\mathbb{1}_{\mathcal{A}_{t_{i+1}}}(s)$ approximates the stationary distribution, which is Bernoulli- α .

Hence, outside an event of vanishing probability conditioned on Γ , the infection process flowing through the graphical construction of ξ_t^k on S_{Γ} behaves as an infection flowing through Γ , stopping at δ_a - and δ_d -events with probability α and $1 - \alpha$ respectively and passing through arrows of type aa and dd with probability α^2 and $(1 - \alpha)^2$ and through arrows of types ad and da with probability $\alpha(1 - \alpha)$. Conditioned on Γ , this now is equal in distribution to the desired contact process (η_t) with rates λ^* and δ^* .

In conclusion, the conditional law of $\xi_t^k \cap S'$ given Γ converges to the conditional law of $\eta_t \cap S'$. Finally, to eliminate the conditioning on Γ , we use dominated convergence.



FIGURE 3.1. Illustration of the proof of Theorem 3.1. Left: $S' = \{2\}$. Γ is depicted black, solid lines represent the exploration of S_{Γ} starting from S' at time t, going backwards. Right: Graphical construction of ξ_t^k obtained from Γ , for large k, by adding to each line σ -events (dots to the left) and ρ -events (dots to the right).

Simulations suggest that the approximation in this theorem already works rather well for relatively low values of k (cf. Figure 3.5).

Similarly we can show that for a suitable limit $h \to 0$, the family (ξ_t^h) converges to a CP in *static environment*, by which we mean that activity states are determined by \mathcal{A}_0 and remain constant over time.

Theorem 3.2 (Slow switching). Let $(\xi_t^{1/k})$ as above, with switching parameters σ/k , ρ/k , all other parameters fixed, and initial infections and initially active sites given by $I, A \subseteq \mathbb{Z}^d$ respectively. Then, for each fixed $t \ge 0$,

$$\mathcal{L}(\xi_t^{1/k}) \Rightarrow \mathcal{L}(\xi_t^0)$$

as $k \to \infty$. Here, (ξ_t^0) is a CPS with switching rates $\rho = \sigma = 0$, i.e. a CP in a static environment determined by A and initially infected individuals given by I.

Proof. This can be proved analogously to the previous theorem. Here, consider as Γ the corresponding graph given by (ξ_t^1) restricted to S', this time *including* switching events. Now by a thinning argument, i.e. by deleting any σ - or ρ -event in Γ with probability k/(k+1) independently of each other, one iteratively obtains a graphical construction of $\xi_t^{1/(k+1)}$ restricted to S' from that of $\xi_t^{1/k}$. Since for k large enough no such events will remain in this finite graphical construction, the result follows.

Note that the CP in static environment has been studied comprehensively over the last decades, cf. e.g. Bramson, Durrett and Schonmann [7] or Klein [15]. In these references the assumption is made that the intensities $\lambda(x, y)$ for the Poisson processes indicating the infections from x to y are independent and identically distributed for all neighbouring pairs of $x, y \in \mathbb{Z}^d$. However, considering the CPS-limit obtained above, this is possible only in the special case where the infection rates do not depend on the activity state at all, i.e. when the original CPS is a CPREE.

To the authors' knowledge, the scaling limits of Theorems 3.1 and 3.2 have not yet been shown for the CPREE. However, Broman conjectured that ξ_t^k behaves like the ordinary CP (η_t) for large k in [8, Remark after Proposition 1.9].

Remark 3.3. Although the iid condition of [7] and [15] is not satisfied for the static version of the CPD, one can still give conditions for survival and extinction: First of all note that the duality of Theorem 2.3 still holds and hence, survival is equivalent to finite survival (cf. Remark 2.7). Finite survival is heavily linked to the existence of an infinite percolation cluster containing $0_{\mathbb{Z}^d}$ in the sense that, if \mathcal{A}_0 is distributed according to π_{α} and α is smaller than the critical value p_c for site percolation on \mathbb{Z}^d , the infection goes extinct almost surely. However, in the extreme case of $\delta_d = 0$, whenever $\alpha < 1$ there is finite survival since in the event $0_{\mathbb{Z}^d} \notin \mathcal{A}_0$ the particle at $0_{\mathbb{Z}^d}$ will never recover.

3.2. Couplings between CPS and the classical Contact Process

In this section we employ and extend the methods and results of [8] to construct couplings between CPS and CP. Trivial versions of these couplings can be directly obtained by monotonicity, maximizing λ_{\bullet} and minimizing δ_{\bullet} for an upper bound and vice versa for a lower bound. One method to improve these trivial bounds can be based on [8, Theorem 1.4]:

There, a background process (B_t) is considered, that flips between the states 0 and 1, where flips to state 1 and 0 occur at rates γp and $\gamma(1-p)$, for fixed $\gamma > 0, p \in (0, 1)$, respectively. On top of that, for two independent PPPs P^0 and P^1 with rates \mathfrak{a}_0 and $\mathfrak{a}_1, \mathfrak{a}_0 \leq \mathfrak{a}_1$, a point process (X_t) is constructed by letting

$$X_t = \{ \tau \in P_t^j \mid j \in \{0, 1\}, B_\tau = j \},\$$

which corresponds to an inhomogeneous Poisson Process jumping at rate \mathfrak{a}_{B_t} at time t. [8, Theorem 1.4] then shows that there is a non-trivial maximal value $\bar{\mathfrak{a}} \in (\mathfrak{a}_0, \mathfrak{a}_1)$ such that a PPP L of rate $\bar{\mathfrak{a}}$ can be constructed that is dominated by (X_t) in the sense that $L_t \subseteq X_t$ for all t. Furthermore $\bar{\mathfrak{a}}$ is given as an explicit function of the parameters:

$$\bar{\mathfrak{a}}(\mathfrak{a}_0,\mathfrak{a}_1,\gamma,p) = \frac{1}{2} \Big(\mathfrak{a}_0 + \mathfrak{a}_1 + \gamma - \sqrt{(\mathfrak{a}_1 - \mathfrak{a}_0 - \gamma)^2 + 4\gamma(1-p)(\mathfrak{a}_1 - \mathfrak{a}_0)} \Big).$$
(3.1)

Unfortunately, a corresponding dominating Poisson Point Process U, with $U_t \supseteq P_t$ for all t, is shown to only exist in trivial cases, i.e. for rates of at least \mathfrak{a}_1 . Hence, in the following theorems one rate will always correspond to the trivial bounds (λ_{\max} and δ_{\max}), while the respective other rate ($\overline{\delta}$ and $\overline{\lambda}$) will arise from (3.1).

Theorem 3.4 (Dominating CP). Let (ξ_t) be a CPS where the initial distribution is of the form $\mu = \nu \otimes \pi_{\alpha}$ and define

$$\lambda_{\max} := \max \{\lambda_{aa}, \lambda_{ad}, \lambda_{da}, \lambda_{dd}\}, \qquad \tau := \mathbb{1}_{\{\delta_a \ge \delta_d\}} \sigma + \mathbb{1}_{\{\delta_a < \delta_d\}} \rho,$$
$$\bar{\delta} := \frac{1}{2} \Big(\delta_a + \delta_d + \rho + \sigma - \sqrt{(|\delta_a - \delta_d| - \rho - \sigma)^2 + 4\tau |\delta_a - \delta_d|} \Big) \ge 0.$$

Then (ξ_t) is stochastically dominated by a basic CP (η_t) with infection rate λ_{\max} and recovery rate $\overline{\delta}$, i.e. we can couple the processes via the graphical construction such that for any initial configuration of infected particles $I \subseteq \mathbb{Z}^d$ we have

$$\mathcal{X}_t^I \subseteq \mathcal{Z}_t^I \quad for \ all \quad t \ge 0,$$

where \mathcal{Z}_t^I denotes the set of infected particles of η_t^I .

Proof. By monotonicity (cf. Theorem 2.2) it is clear that (ξ_t) is dominated by another CPS $(\hat{\xi}_t)$ with the same δ -rates but $\hat{\lambda}_{\bullet} = \lambda_{\max}$ for all $\bullet \in \{aa, ad, da, dd\}$, which is in fact a CPREE. Now, the above theorem follows from [8, Theorem 1.6]. For completeness we provide a sketch of proof:

First assume that $\delta_a \geq \delta_d$ and hence $\tau = \sigma$. Otherwise, exchange the roles of a and d (and thus σ and ρ). For any $x \in \mathbb{Z}^d$ we can regard $B_t = \mathbb{1}_{\mathcal{A}_t}(x)$, $P^0 = U^{d,x}$ and $P^1 = U^{a,x}$. The process $X = (X_t)$ constructed as above then contains any δ -event along the line of x that aligns with the activity states of x and thus can actually affect a present infection. Now, [8, Theorem 1.4] guarantees the existence of a PPP $L^{(x)}$ of rate $\bar{\mathfrak{a}}(\delta_d, \delta_a, \rho + \sigma, \alpha) = \bar{\delta}$ dominated by X. (Note here that in the case $\delta_a < \delta_d$, the last entry has to be $1 - \alpha$ instead of α , since p in (3.1) denotes the fraction of flips into the state 1 of the higher rate \mathfrak{a}_1 .) Using the collection of the $L^{(x)}$, $x \in \mathbb{Z}^d$, as recovery-events to construct a CP gives rise to the desired process (η_t) .

The non-negativity of δ holds, since

$$\begin{aligned} (\delta_a + \delta_d + \rho + \sigma)^2 &= (2\min\{\delta_a, \delta_d\} + |\delta_a - \delta_d| + \rho + \sigma)^2 \\ &\geq (|\delta_a - \delta_d| + \rho + \sigma)^2 \\ &= (|\delta_a - \delta_d| - \rho - \sigma)^2 + 4(\rho + \sigma)|\delta_a - \delta_d|. \end{aligned}$$

Remark 3.5. As mentioned in the introduction, there are two natural interpretations for the CPD: (microbial) host dormancy with $\delta_d = 0$, and social distancing interpreted as (human) dormancy with $\delta_d = \delta_a$. Since for $\rho \to 0$ it holds $\overline{\delta} \to \min\{\delta_a, \delta_d\}$, one can see that Theorem 3.4 leads to significantly different bounds between these cases. Also, the fast switching approximation from Theorem 3.1 can differ strongly for these two strategies. Indeed, in both cases the human dormancy is favourable.

For a lower bound we now strive to apply Broman's coupling to the infection arrows originating from a single particle, instead of to the recovery events. To account for the stochastic dependencies between such arrows, we need to extend [8, Theorem 1.4]:

Lemma 3.6. For $k \in \mathbb{N}$ let $N^{1,0}, \ldots, N^{k,0}$ be independent Poisson Point Processes (PPPs) with rate λ_0 and $N^{1,1}, \ldots, N^{k,1}$ independent PPPs with rate $\lambda_1 \geq \lambda_0$ such that $N^{i,0}$ is also independent of $N^{j,1}$ for all $i, j \in [k]$ with $i \neq j$. Further, let (J_t) be a jump process in $\{0, 1\}$ jumping to 0 at rate σ and to 1 at rate ρ , satisfying $\mathbb{P}(J_0 = 1) = \alpha$. Then, there are k independent PPPs L^1, \ldots, L^k with rate

$$\bar{\lambda}(\lambda_0,\lambda_1,\sigma,\rho,k) := \frac{1}{2} \left(\lambda_1 + \lambda_0 + \frac{\rho}{k} + \frac{\sigma}{k} - \sqrt{(\lambda_1 - \lambda_0 - \frac{\rho}{k} - \frac{\sigma}{k})^2 + \frac{4\sigma}{k}(\lambda_1 - \lambda_0)} \right)$$

such that almost surely for any $i \in [k]$ it holds

$$L^i \subseteq \{ \tau \in N^{i,j} \mid J_\tau = j \}.$$

Proof. Consider $N^j := \bigcup_{i=1}^k N^{i,j}$, $j \in \{0,1\}$, which are PPPs of rate $k \cdot \lambda_j$ respectively. Applying [8, Theorem 1.4] delivers existence of a PPP L of rate

$$\gamma := \bar{\mathfrak{a}}(k\lambda_0, k\lambda_1, \rho + \sigma, \frac{\rho}{\rho + \sigma}) = k \cdot \bar{\lambda}(\lambda_0, \lambda_1, \sigma, \rho, k),$$

such that any $\tau \in L$ holds $\tau \in N^{J_{\tau}}$. (Note that for k = 1 this already concludes the proof.)

Now, noting that the random variables U_{τ} for $\tau \in N^0 \cup N^1$, given by $U_{\tau} = i \in [k]$ iff $\tau \in N^{i,j}$ for some j, are iid, uniformly distributed on [k] and independent of (J_t) , it is straightforward to subdivide L into L^1, \ldots, L^k such that the Lemma holds.

To illustrate the lemma above and the strategy of the following proof, we provide Figure 3.2.



FIGURE 3.2. This figure illustrates Lemma 3.6 for k = 2 in the context of the graphical construction. We interpret the states 0 and 1 of J_t as the activity of a site $x \in \mathbb{Z}^d$ at time t, indicated by the colours grey and black, respectively. Gray arrows belong to the PPPs $N^{i,0}$, black ones to $N^{i,1}$. They are depicted solid iff they fit the activity of x. Arrows to the left belong to $N^{1,j}$, those to the right to $N^{2,j}$. The lemma aims to estimate the solid arrows (black and grey) pointing to the left from below (in the sense of " \subseteq ") by a PPP L^1 and the ones pointing to the right by L^2 (as depicted on the right) such that L^1 and L^2 are independent.

Note that in the above lemma, the PPPs $N^{i,0}$ and $N^{i,1}$ for fixed *i* do not need to be independent, which leaves room for the above mentioned dependencies. With this, we can now provide a complementary result to Theorem 3.4, involving a dominated CP, by taking the trivial bound for the δ -rates and applying Lemma 3.6 to the λ -rates.

Theorem 3.7 (Dominated CP). Let (ξ_t) be a CPS whose initial distribution is of the form $\mu = \nu \otimes \pi_{\alpha}$. Further, we define $\lambda_a = \min\{\lambda_{aa}, \lambda_{ad}\}, \lambda_d = \min\{\lambda_{da}, \lambda_{dd}\},$

$$\begin{aligned} \tau &:= \mathbb{I}_{\{\lambda_a \ge \lambda_d\}} \sigma + \mathbb{I}_{\{\lambda_a < \lambda_d\}} \rho, \\ \bar{\lambda} &:= \frac{1}{2} \left(\lambda_a + \lambda_d + \frac{\rho + \sigma}{|\mathcal{N}|} - \sqrt{\left(|\lambda_a - \lambda_d| - \frac{\rho + \sigma}{|\mathcal{N}|} \right)^2 + \frac{4\tau}{|\mathcal{N}|} |\lambda_a - \lambda_d|} \right) \ge 0 \end{aligned}$$

Then (ξ_t) dominates a basic CP (η_t) with infection rate $\bar{\lambda}$ and recovery rate $\delta_{\max} = \max\{\delta_a, \delta_d\}$. Proof. First of all, w.l.o.g. assume that $\delta_a = \delta_d = \delta_{\max}$, $\lambda_{aa} = \lambda_{ad} = \lambda_a$ and $\lambda_{da} = \lambda_{dd} = \lambda_d$. Otherwise, consider a CPS $(\bar{\xi}_t)$ with these rates, which is dominated by (ξ_t) by monotonicity. Further, assume that $\lambda_a \geq \lambda_d$ or exchange the roles of a and d.

Let $x \in \mathbb{Z}^d$ and enumerate $\{y \in \mathbb{Z}^d \mid y \sim x\} = \{y_1, \ldots, y_{|\mathcal{N}|}\}$. Further, for $i \in [|\mathcal{N}|]$ let $A_i = \{t \ge 0 \mid y_i \in \mathcal{A}_t\}$, the active times of y_i , and define

$$N^{i,0} := (T^{x \to y, da} \cap A_i) \cup (T^{x \to y, dd} \cap A_i^c) \text{ as well as}$$
$$N^{i,1} := (T^{x \to y, aa} \cap A_i) \cup (T^{x \to y, ad} \cap A_i^c).$$

Now, both $(N^{i,0})$ and $(N^{i,1})$ are collections of PPPs with rates λ_d and λ_a respectively, such that, letting $J_t = \mathbb{1}_{\mathcal{A}_t}(x)$, Lemma 3.6, provides independent PPPs $(L^{x \to y})_{y \sim x}$ each with rate $\bar{\lambda}$, dominated by the infection arrows of the CPS in the desired sense. Repeating this for every $x \in \mathbb{Z}^d$, using all the resulting $L^{x \to y}$ to build a graphical construction, delivers the desired dominated CP with infection rate $\bar{\lambda}(\lambda_d, \lambda_a, \sigma, \rho, |\mathcal{N}|)$. Insertion concludes the proof. (Non-negativity of $\bar{\lambda}$ follows analogously to that of $\bar{\delta}$.)

From the previous two theorems we can now read off sufficient conditions for survival and extinction.

Corollary 3.8. Let (ξ_t) be a CPS with $\sigma, \rho > 0$.

- (1) If $\delta_a + \delta_d > 0$, (ξ_t) goes extinct if the λ_{\bullet} are small enough.
- (2) If $\min\{\lambda_{aa}, \lambda_{ad}\} + \min\{\lambda_{da}, \lambda_{dd}\} > 0$, (ξ_t) survives if the δ_{\bullet} are small enough. In particular, in this case a non-trivial upper invariant distribution exists.

Proof. From the last part of the proof of Theorem 3.4 one can see that $\delta > 0$ if either $\delta_a > 0$ or $\delta_d > 0$. Thus, if λ_{\max} is small enough, (η_t) goes extinct and hence does (ξ_t) . The second part follows analogously by consideration of Theorem 3.7.

These conditions imply existence of phase transitions for a broad subclass of CPS. However, due to the high number of parameters, it is complicated to formulate more specific conditions for the existence of phase transitions in a single parameter, e.g. λ_{aa}^c . Remark 3.12 illustrates an example of absence of such a phase transition.

Remark 3.9 (Sharpness and further improvements of the bounds). Trivially, both the dominating and dominated CP bounds become sharp when λ_{\bullet} and δ_{\bullet} are constant, i.e. when the CPS is a CP itself.

Further, for extreme values of σ and ρ the bounds can also be sharp, since for $\rho > |\delta_a - \delta_d|$ as $\sigma \to 0$ it holds that $\bar{\delta} \to \delta_{\max}$. This implies that the coupling is good when dormant states are lost and the CPREE becomes a CP itself. In particular, this shows that $\bar{\delta}$ is a much better bound than the trivial bound min{ δ_a, δ_d }. Similar arguments can be made for $\bar{\lambda}$.

Adapting the proof above slightly, considering *incoming* arrows instead of outgoing ones, one can also choose $\lambda'_a = \min\{\lambda_{aa}, \lambda_{da}\}$ and $\lambda'_d = \min\{\lambda_{ad}, \lambda_{dd}\}$, leading to a dominated CP with infection rate $\bar{\lambda}' = \bar{\lambda}(\lambda'_a, \lambda'_d, \sigma, \rho, |\mathcal{N}|)$. Then, exchanging $\bar{\lambda}$ for $\max\{\bar{\lambda}, \bar{\lambda}'\}$ can further improve the bounds obtained from Theorem 3.7 and relaxes the condition in Corollary 3.8(2) to $\lambda_a + \lambda_d + \lambda'_a + \lambda'_d > 0$.





FIGURE 3.3. Sketch of the minimal survival and extinction regions of a CPS due to the domination results in Theorems 3.4 and 3.7. We consider the special case where $\sigma = \rho = 1$ and $\lambda_{da} = \lambda_{dd} = \delta_d = 0$ are fixed and the parameters $\lambda = \lambda_{aa} = \lambda_{ad}$ and $\delta = \delta_a$ vary. The parameters $\bar{\lambda}$ and $\bar{\delta}$ depend on λ and δ respectively and are determined according to the equations in Theorems 3.4 and 3.7. The quantity δ_{CP}^c represents the critical recovery rate of the basic Contact Process as a linear function of λ .



FIGURE 3.4. Sketch of the minimal survival and extinction regions of a CPD. The parameters $\delta = \delta_a = \delta_d$ and $\lambda = \lambda_{aa}$ vary. We assume σ and ρ are fixed and chosen according to Remark 3.12 so that for some δ^* we have $\lambda^c(\sigma, \rho, \delta^*) = \infty$, i.e. survival is impossible for any $\delta \geq \delta^*$. The exact form of the (strictly positive) function $\delta^c_{CPD}(\lambda)$ is unknown.

Remark 3.10 (Couplings for the CPD). In special cases such as the CPD, these bounds can be very bad: Here, the dominated CP, which has infection rate $\bar{\lambda} = 0$ and recovery rate $\delta_a > 0$, will never survive when started with finitely many infections. However, in order to ensure survival one can construct an alternative coupling between the CPD (with $\delta_d = 0$ or $\delta_d = \delta_a$) and oriented percolation by adapting the arguments in [3, p. 138–142] to our setting. This gives the existence of a critical δ^c for fixed σ , ρ and λ .

Remark 3.11 (Couplings for the CPREE). For the CPREE the dominating CP with $\lambda_{\max} = \lambda$ and $\bar{\delta}$ from Theorem 3.4 looks promising. Indeed, if we let $\sigma, \rho \to \infty$ simultaneously, we observe that

$$\overline{\delta} \to \alpha \delta_a + (1 - \alpha) \delta_d,$$

which coincides with δ^* from Theorem 3.1. See Figure 3.6 for a comparison via simulation.

Remark 3.12 (Non-existence of critical infection rate for the CPD). In the special case of the CPD, there are non-trivial choices of σ , ρ and δ_d such that the process does not survive for any choice of λ and δ_a , i.e. $\lambda^c(\sigma, \rho, \delta_d, \delta_a) = \infty$ for all δ_a . This can be shown similarly to [24, p. 2401-2403] by making a connection to continuum percolation and giving a coupling with a multi-type-branching process.

Intuitively, if the dormant state is sufficiently lethal ($\delta_d \gg 0$) and active phases are sufficiently rare ($\sigma \gg \rho$), the infection goes extinct independently of its strength during the active phase.

Remark 3.13 (Monotonicity in the switching parameters). In some special cases one can even show monotonicity in the parameters σ and ρ via the graphical construction. For example, the coupling of two CPREE's $(\mathcal{X}_t, \mathcal{A}_t)(\sigma_1, \rho_1, \delta_a, \delta_d, \lambda)$ and $(\mathcal{Y}_t, \mathcal{B}_t)(\sigma_2, \rho_2, \delta_a, \delta_d, \lambda)$ with $\sigma_1 \leq \sigma_2$, $\rho_1 \geq \rho_2$ and $\delta_d \leq \delta_a$ such that we have

 $\mathcal{B}_t \subseteq \mathcal{A}_t$ and $\mathcal{X}_t \subseteq \mathcal{Y}_t$ for all $t \ge 0$

is straightforward. This comes from the fact that the dormant state is only in favour for the infection because it decreases the recovery rate of the particles (if $\delta_d \leq \delta_a$) and does not harm the spread of the infection.

On the other hand, for the CPD with $\delta_a = \delta_d$ the dormant state is harmful for the infection in the sense that the recovery rate does not decrease and some infections are not allowed. Hence, we can show monotonicity in σ and ρ for this special case as well.

3.3. Simulations

Figure 3.5 illustrates all results of Sections 3.1 and 3.2 in comparison to a single CPS (top left). This shows that, even when σ and ρ are well within the range of the other parameters, the fast switching limit CP (top right) can give an apparently reasonably good approximation to the CPS, while neither dominating CP (below left) nor dominated CP (below right) nearly come as close.

Meanwhile, Figure 3.6 shows, as argued in Remark 3.11, that the dominating CP (right) can be a good approximation for the CPREE (left). Note that here with $\sigma = \rho = 5$, neither is switching particularly fast, nor extreme in the sense of Remark 3.9, where ρ is significantly larger than σ .

Finally, Figure 3.7 suggests that the critical parameter λ^c for (finite) survival of the CPD under consideration lies in the interval [7,8]. Since in that particular process sites are active half of the time on average, intuitively one would assume that roughly a quarter of infection arrows are successful. This argument suggests that $\lambda^c \approx 4\lambda_{CP}^c$, where λ_{CP}^c denotes the critical parameter of a CP with recovery rate $\frac{1}{2}$. (Note that this corresponds to the effective rates from Theorem 3.1.) However, as shown in [21, Theorem 1.3], $4\lambda_{CP}^c \leq 4 \cdot \frac{1}{2} \cdot 1.95 = 3.9$. Thus, intuition is off here roughly by a factor of 2. It is reasonable to assume that this factor increases significantly as switching gets slower and the CPS gets closer and closer to a CP in static environment.

4. Relation to other models and open problems

4.1. Relation to the "classical" multi-type Contact Process

The classical multi-type (in our set-up: two-type) Contact Process (MTCP) as described e.g. in [20, 23] can be obtained as a scaling limit of our model. Recall that the MTCP does not allow



FIGURE 3.5. CPS (top left), fast switching limit CP (λ^*, δ^*) (top right), dominating CP $(\lambda_{\max}, \bar{\delta})$ (below left) and dominated CP $(\bar{\lambda}, \delta_{\max})$ (below right). Red and blue: (active) infected and healthy respectively; yellow and green: dormant infected and healthy respectively.



FIGURE 3.6. CPREE (left), dominating CP $(\lambda_{\max}, \overline{\delta})$ (right).

for switches between types of infected individuals, and the healthy state does not carry a type at all. This means that if one only allows for finite switching rates ρ, σ , the classical multi-type Contact Process lies outside of our modelling framework. However, if one is willing to consider the limits of infinite switching rates (in a suitable sense), the model can be accommodated via the transitions in Figure 4.1 (left).



FIGURE 3.7. Simulations of a CPD, $\sigma = \rho = 1$, $\delta_a = \delta_d = \frac{1}{2}$, on $\mathbb{Z}/400\mathbb{Z}$ with only one initially infected individual at $\{0\}$. The plot shows for $\lambda \in \{6, 6.5, 6.75, 7, 7.25, 7.5, 7.75, 8\}$ how many infections of 500 iterations survived until time T.

4.2. Relation to the CPB of Remenik

Remenik in [24] introduced a model which is closely related to the CPD and which we will call the Contact Process with Blocking (CPB), see Figure 4.1 (right). It can be seen as a special case of our model with the convention $\delta_a = 1$ and $\delta_d = \infty$. (Choose $\sigma = \delta$ and $\rho = \alpha\delta$ for some $\alpha, \delta > 0$ to obtain the nomenclature of [24].) This model is interesting because we can couple any CPS ($\mathcal{X}_t, \mathcal{A}_t$) (with recovery rate δ_a scaled to one) with a CPB ($\mathcal{Y}_t, \mathcal{B}_t$) with identical switching rates and infection rate $\lambda = \lambda_{aa}$ via the graphical construction such that the CPB is dominated by the CPS, i.e. $\mathcal{X}_t \supseteq \mathcal{Y}_t$. Further, Remenik was able to show complete convergence for his model (cf. [24, Proposition 5.1]).



FIGURE 4.1. Left: The multi-type Contact Process; switching rates $\sigma_0 = \rho_0 \rightarrow \infty$, $\sigma_1 = \rho_1 = 0$ (MTCP). Right: State transition graph of the Contact Process with blocking (CPB); $\delta_d \rightarrow \infty$, dashed transition will never occur.

4.3. The CP with infection dormancy (CPID)

Besides the host dormancy perspective, there is also an infection dormancy perspective ("latency" or "persistence"). For instance, a pathogen might encapsulate itself to have a higher chance to survive under harsh conditions with the trade-off of no reproduction during the encapsulation. For modelling this we introduce a Contact Process with infection dormancy (CPID), where only infected individuals exhibit switching. We assume that an infected individual with a dormant infection recovers at a smaller rate than an individual with an active infection, i.e. $\delta_d < \delta_a$. In return, only individuals which carry an active infection can infect their neighbours. Again we assume a spontaneous change from active to dormant and vice versa. The transition graph for this model is given below in Figure 4.2 (left).

To make the CPID comparable to CPS, we consider it artificially as a process with states in $F = \{0, 1\} \times \{a, d\}$. One way to do this would be letting $\sigma_0 = \rho_0 \rightarrow \infty$ – similarly to MTCP. However, there is another interesting way without losing the convenient independence between switching rates and infection, which is given by introducing a *diagonal* arrow from (0, d) to (1, a) (cf. Figure 4.2, right).



FIGURE 4.2. Left: State transition graph of the CPID. Right: Equivalent characterization of the CPID on F^S .

Via such diagonal arrows, infection events directly influence activity states, and this gives rise to additional stochastic dependencies, since activity states do not evolve independently of the infection any more. Furthermore, the ability of dormant infections to block infection spread leads to the failure of the coupling arguments that we need to prove either monotonicity, additivity or attractivity, which makes the application of the methods in Sections 2.2 and 3 infeasible.

What one can do, however, is to compare such a CPID $(\mathcal{X}_t, \mathcal{A}_t)$ to a CPD $(\mathcal{Y}_t, \mathcal{B}_t)$ with identical switching and recovery rates. Then, by comparing the graphical constructions of both processes with identical initial configurations, one sees that $\mathcal{A}_t \supseteq \mathcal{B}_t$ for all t, which then implies $\mathcal{X}_t \supseteq \mathcal{Y}_t$. In turn, the CPID is also dominated by a CP with rates λ and $\overline{\delta}$ by the same arguments as in Theorem 3.4.

These couplings at least provide a basic understanding of the behaviour of the CPID. In particular, we can conclude the existence of some parameter choices such that the CPID survives and others such that the process does not. Nevertheless, a more vigorous study of the CPID is desirable.

Lastly, one could also think of taking the diagonal arrow from (0, a) to (1, d) into account, which would correspond to infections exhibiting an incubation period.

4.4. Open problems and future research

Obviously, one may consider switching models with more than just two types, and perhaps even infinitely many, opening up a wide modelling range. For the two type case, natural next steps could be the derivation of a complete convergence result, investigation of *strong* survival as well as investigating whether a critical CPS dies out.

However, it also seems natural to move beyond the contact process and to incorporate dormancy and switching mechanisms into other classical interacting particle systems such as voter models or exclusion processes, and to investigate their scaling limits. In this context, very recent work of [12] shows that microscopic dormancy can give rise to non-classical macroscopic transport laws, including up-hill diffusion, a phenomenon that cannot emerge in single-type scenarios.

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