Lecture notes on population dynamics
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Contents

Preface

These notes serve for an advanced course on the mathematical theory of stochastic population dynamics, in particular adaptive dynamics.

This course is first offered at the Goethe-Universität Frankfurt in the summer semester of 2024, where I am spending one month as a guest lecturer, which is part of the International Campus Program of the university. Since one of the goals of this program is to build and reinforce connections between the Goethe-Universität Frankfurt and foreign academic institutions (just like the Budapest University of Technology and Economics, where I am currently working), I will present a rather personal view of the area, focusing on some concrete biological examples, some of which belong to the subject of my own research with Jochen Blath and other coauthors.

There are various lecture notes of past courses by other authors on this subject, e.g. by Bansaye and Méléard [BM15] and by Bovier [B21], and these also represent somewhat personal perspectives of the area. These are great sources of knowledge about various general (and often abstract) models of the field, any they also include particular examples and applications from the authors' own particular research area. In order to be able to fit the particular biological examples that we are interested in into the time frame of my course, I need to provide lecture notes with a restricted amount of general theory compared to the aforementioned references. We will nevertheless get to know some relatively general models as well (such as the individual-based models of adaptive dynamics scaling to a trait substitution sequence, introduced by Champagnat [C06] right at the beginning). Still, these lecture notes should not be seen as an exhaustive reference on adaptive dynamics and population dynamics, neither from the biological nor from the mathematical perspective. The main goal of the lecture notes is to cover and sort the material of the course, including a small number of exercises.

Taking into account that some of the readers may and some other readers may not be familiar e.g. with the theory of multitype branching processes or qualitative systems of ODEs, I will sometimes present the methods for the concrete examples that we are interested in, without a general formalism but in a way that hopefully clarifies how to treat further examples. To some extent, my inductive approach will be similar to the way how my coauthors and I got to know this subject, coming from the related but different area of mathematical population genetics. But I also intend to be precise in presenting the technical details of at least some of the crucial steps, and for didactic reasons I will often provide more details than the corresponding articles.

1 Invasion models of adaptive dynamics and convergence to a trait substitution sequence

1.1 Introduction: a few words about population dynamics and adaptive dynamics

In population dynamics, we model populations of living creatures (or sometimes also viruses etc.) and investigate the fate of *phenotypic traits (types)*. The population size is regulated by *logistic competition* between individuals, which does not allow the number of individuals to grow larger than constant times the *carrying capacity* of the population. This contrasts with population genetics (which the reader might be familiar with from e.g. Jochen Blath's courses), where the population size is often assumed to be constant, one investigates genotypes (often determined by a difference at one single genetic locus).

In simple *invasion models* of population dynamics, a frequently studied scenario is that the population is described by a continuous-time Markov chain with values in $\mathbb{N}_0^n$ for some $n \geq 2$,[1] there is initially a *resident population* of a single type or multiple coexisting types, and a *mutant* arrives. We are interested in the fate of the mutant: Will it survive with asymptotically positive probability as the population size (equivalently, the carrying capacity) tend to infinity? If yes, what happens after a successful invasion: Will the mutant fix, making the previous resident population go extinct, or will it coexist with (some of) the former resident types?

We will soon see that due to classical results by Ethier and Kurtz [EK86], if the sizes of all subpopulations are of the order of the carrying capacity, one can approximate the subpopulation sizes divided by the carrying capacity via *deterministic systems of ordinary differential equations (ODEs)* on compact time intervals. In this field, we often refer to these systems of ODEs as *dynamical systems*, although they do not have too much to do with the (measure-preserving) dynamical systems known from ergodic theory. Understanding the qualitative behaviour of these systems (existence and local/global stability of its equilibria, possible bifurcations etc.) provides valuable information on the behaviour of the stochastic population models themselves when the carrying capacity is large, and in fact, many models of population dynamics were first introduced in the form of a system of ODEs. However, one should emphasize that this approximation is true without rescaling time, only on finite time intervals, and only if all subpopulation sizes are sufficiently large.

The invasion of a mutant in a single-type resident population in a stochastic individual-based model often consists of (up to) *three phases*. At the moment when a single mutant arrives into the population, the mutant subpopulation is clearly not large enough for an ODE approximation. In chance, if one can guarantee that the resident population size stays close to its equilibrium (in a well-defined sense) for a sufficiently long time, one may be able to approximate the mutant population size via a *branching process* (in continuous time) as long as it either dies out or becomes macroscopic, which ends the *first phase* out of three phases of the invasion.

If case the branching process is supercritical, the mutant may survive the first phase with nonvanishing probability, and afterwards the *second*, shorter *phase* starts, where now the dynamical system approximation is applicable. Depending on the properties of the dynamical system, the second phase leads the rescaled resident population size of the former resident close to extinction, or it leads the rescaled system to the vicinity of a stable coexistence equilibrium between resident and mutant, and there are also other possibilities (namely, periodic or chaotic behaviour). The *third phase* occurs in case the former resident has already gone almost extinct by the end of the second phase. Now the mutant stays close to its equilibrium population size for a long time, while the former resident can be approximated by a subcritical branching process, which tends to decay exponentially fast and dies out after an amount of time that is of the same order as the duration of the first phase.

One usually says that in population dynamics one observes populations in the *ecological time scale* (which contrasts with the *evolutionary time scale* studied in population genetics, where one typically rescales time by the constant population size in order to obtain interesting scaling limits such as the Wright–Fisher diffusion

[1] We write $\mathbb{N}_0 = \{0, 1, 2, \ldots\}$ and $\mathbb{N} = \{1, 2, \ldots\}$ throughout these lecture notes, for which we apologize to the Hungarian readers.

or the Kingman coalescent). In some cases, the approach of the areas of population genetics and population dynamics can be united in some sense, so that a series of consecutive mutations can be studied and via a suitable scaling of time a (deterministic or random) scaling limit can be obtained. This is the goal of *adaptive dynamics*, which is a simplified theoretical approach to *meso-evolution*, i.e., the study of phenotypic traits under the assumption of a separation of the time scales of ecology and evolution, i.e., a low rate of trait-changing, beneficial mutations (see [B21, Section 1.3] for more details).

While the biological motivation of the stochastic individual-based (or *microscopic*) models informally sketched above is clear, in the 1990s it became popular to study a different kind of models in adaptive dynamics: the so-called *trait substitution sequence (TSS) models*. See e.g. [M96, DL96, CFB01] and the references therein. In the latter type of models, under the assumption that no coexistence is possible between different traits, most of the time the population consists of one single resident trait, and at random times the trait of the population jumps to a new, randomly chosen value. This kind of model had no clear microscopic interpretation before the work by Champagnat [C06], which explained TSS models as the limit of individual-based models in the limit of a large carrying capacity coupled with rare mutations. Taking the consecutive limit of mutations of small effects, the TSS converges to the so-called *canonical equation of adaptive dynamics* (CEAD) [CFB01]. Many common proof techniques in population dynamics and adaptive dynamics are based on Champagnat's paper [C06], whence it will be the starting point of these lectures. Another fundamental work on stochastic adaptive dynamics was the (even earlier) paper by Fournier and Méléard [FM04], which we will also often refer to in these lecture notes, but we will not consider its model directly.

Our last caveat before turning to the mathematics is that often, our models will be biologically oversimplified or even caricaturistic compared to the complexity of living creatures, even in the case of microorganisms. We do not intend to make the impression that real-world living organisms are as simple as assumed in and suggested by these models, since this would be wrong, but we believe that even such simple models may be suitable for predicting the evolution of microbial traits or in some cases even traits of much more complex species of creatures in certain situations.

1.2 Organization of these lecture notes

In the rest of Section 1 we lay the groundwork of individual-based stochastic models of adaptive dynamics, we state the main result of [C06], and we sketch its proof, going into details regarding some proof techniques that are also essential for many other population-dynamic models, in particular regarding stability of equilibria of dynamical systems, the Poissonian construction and coupling methods, the problem of diffusion exit from a domain and Freidlin–Wentzell type large deviations, and some basic properties of one-dimensional branching processes.

Sections 2 and 3 explain the behaviour of more concrete examples of stochastic individual-based population-dynamic models based on the papers [BT20, BK98, BT23] and relying heavily on proof techniques of the papers [CCLS18, CCLLS21]. These sections investigate the effect of *dormancy* (see the beginning of Section 2 for an explanation) in two different models: a competitive Lotka–Volterra type model and an epidemic model where viruses attack microbial hosts. In these sections, we study only one single mutation and we can rely on some methods from Section 1, but we still need to expand our toolbox of proof techniques. In Section 2 the main novelty will be the multitype nature of the hosts, which brings techniques related to multitype branching processes (e.g., the Kesten–Stigum theorem) into play, and particular methods will also be needed to show global stability properties of the dynamical system. Multitype branching processes are also important for Section 3, while that section is rather heavy on methods related to systems of ODEs. In addition to the basic tools from Section 1, here also Hopf bifurcations, the Routh–Hurwitz criterium, and Lyapunov function type arguments will play a role.

In Section 4 we will again study the scaling limit of an individual-based model with multiple mutations [CMT21], in a regime where the frequency of mutations decays much slower than in [C06] as the population size diverges. Here, the main new biological feature is *horizontal gene transfer*, and the sizes of subpopulations converge to a deterministic and piecewise affine function under a logarithmic scaling of population sizing and a suitable scaling of time. The subarea of adaptive dynamics where such piecewise

affine limiting processes emerge has been researched very actively in the last 15 years. Here, proof techniques are based on branching processes, also logistic ones and ones with immigration. The description and analysis of the behaviour of the limiting process is also interesting and somewhat involved.

Section 4 is essentially independent of Sections 2 and 3, and hence the sections can also be read in the order $1 \to 4 \to 2 \to 3$. We might follow the latter order in some editions of this course.

1.3 Champagnat's individual-based model

Champagnat [C06] considered a finite number of quantitative traits in a population of one-cell organisms with asexual and haploid reproduction (in other words: clonal reproduction, binary fission) and assumed that the trait space $\mathcal{X}$ is a compact subset of $\mathbb{R}^l$, $l \geq 1$.

Champagnat's microscopic model involves the three basic mechanisms of Darwinian evolution: *heredity*, which transmits traits to new offsprings, *mutation*, driving a variation in the trait values in the population, and *selection* between these different trait values. The selection process in this model is a consequence of the competition between individuals in the population for limited resources or area. The model is defined as follows[2].

For any $x, y \in \mathcal{X}$, we introduce the following biological parameters:

- $\lambda(x) > 0$ is the rate of birth from an individual with trait x,
- $\mu(x) > 0$ is the rate of natural mortality (death by age) of an individual of trait x,
- $\alpha(x, y) > 0$ describes the competitive pressure exerted by an individual with trait y affecting an individual of trait x,
- $a(x) \in [0, 1]$ is the probability that a mutation occurs when an individual with trait x gives birth,
- $M(x, \mathrm{d}h)$ is the law of $h = y - x$ where the mutant of trait y is born from an individual of trait x. $M(\cdot, \mathrm{d}h)$ is a probability kernel from $\mathbb{R}^l$ to the Borel σ-algebra $\mathcal{B}^l$ of $\mathbb{R}^l$ to $\mathbb{R}^l$, i.e., $x \mapsto M(x, A)$ is measurable for all $A \in \mathcal{B}^l$ and $A \mapsto M(x, A)$ is a probability measure for all $x \in \mathbb{R}^l$.[3] Since y must belong to the trait space $\mathcal{X}$, the support of $M(x, \cdot)$ must be a subset of
$$\mathcal{X} - x = \{y - x \colon y \in \mathcal{X}\}.$$
- $K \in \mathbb{N}$ is the carrying capacity of the population. In this model, it will serve as a parameter rescaling $\alpha(\cdot, \cdot)$, and later we will see that large K means a large population (provided that the initial condition is proportional to K).
- $u_K \in [0, 1]$ is a parameter depending on K rescaling the probability of mutation $a(\cdot)$. Small u_K means rare mutations.

At any time $t \geq 0$, we consider a finite number N_t of individuals, each of them having a trait value in $\mathcal{X}$. Let us denote by $x_1, \ldots, x_{N_t}$ the trait values of these individuals. The state of the population at time $t \geq 0$, rescaled by K, can be described by the following *empirical measure*:
$$\nu_t^K = \frac{1}{K} \sum_{i=1}^{N_t} \delta_{x_i}, \tag{1.1}$$

[2]Here we follow Champagnat's paper [C06], often word by word, but our notation will be somewhat different from his one in order to align with the notations of certain further models better.

[3]If you are not familiar with probability kernels, think about $M(x, A) = \mathbb{P}(\text{the mutant trait is in } A | \text{ the parent trait is } x)$. E.g. if the parent trait has an absolutely continuous distribution, this is formally defined via the usual conditional distribution function/conditional density.

where δ_x is the Dirac measure at x. This is a random element of the set of finite nonnegative measures (in other words, finite point measures) on $\mathcal{X}$ defined as

$$\mathcal{M}^K = \Big\{ \frac{1}{K} \sum_{i=1}^{n} \delta_{y_i} : n \geq 0, y_1, \ldots, y_n \in \mathcal{X} \Big\}.$$

Then, somewhat informally speaking, our individual-based model is defined as follows. It is a continuous-time Markov chain with (exponential waiting times and) the following transition rates: In the population ν_t^K,

- an individual with trait x gives birth to another individual with rate $\lambda(x)$. The newborn has the same trait value as its progenitor's one with probability $1 - u_K a(x)$, and with probability $u_K a(x)$, the newborn is a mutant whose trait value y is chosen according to $y = x + h$ where h is a random variable with law $M(x, \mathrm{d}h)$.
- an individual with trait x dies with rate

$$\mu(x) + \int \alpha(x, y) \nu_t^K(\mathrm{d}y) = \mu(x) + \frac{1}{K} \sum_{i=1}^{N_t} \alpha(x, x_i).$$

  The parameter K scales the strength of competition, thus allowing the simultaneous presence of more individuals in the population.

More formally, the process $(\nu_t^K)_{t \geq 0}$ is an $\mathcal{M}^K$-valued Markov process with infinitesimal generator (Q-matrix) defined for any bounded measurable function $\phi \colon \mathcal{M}^K \to \mathbb{R}$ by

$$\begin{aligned} L^K \phi(\nu) = & \int_{\mathcal{X}} \Big(\phi(\nu + \frac{\delta_x}{K}) - \phi(\nu) \Big) (1 - u_K a(x)) \lambda(x) K \nu(\mathrm{d}x) \\ & + \int_{\mathcal{X}} \int_{\mathbb{R}^l} \Big(\phi(\nu + \frac{\delta_{x+h}}{K}) - \phi(\nu) \Big) u_K a(x) M(x, \mathrm{d}h) \lambda(x) K \nu(\mathrm{d}x) \\ & + \int_{\mathcal{X}} \Big(\phi(\nu - \frac{\delta_x}{K}) - \phi(\nu) \Big) \Big(\mu(x) + \int_{\mathcal{X}} \alpha(x, y) \nu(\mathrm{d}y) \Big) K \nu(\mathrm{d}x). \end{aligned} \tag{1.2}$$

When the measure ν is of the form (1.1), the integrals with respect to $K\nu(\mathrm{d}x)$ in (1.2) correspond to sums over all individual in the population. The first (linear) term describes the births without mutation, the second (linear) term corresponds to the births with mutation, and the third (nonlinear) term accounts for the deaths by age or competition. This logistic density-dependence models the competition in the population and hence drives the selection process.

1.4 Equilibrium population sizes, invasion fitnesses, and convergence to a TSS model

Let us denote by (A) Champagnat's following three assumptions.

(A1) $x \mapsto \lambda(x)$, $x \mapsto \mu(x)$, and $(x, y) \mapsto \alpha(x, y)$ are measurable functions, and there exist $\bar{\lambda}, \bar{\mu}, \bar{\alpha} < \infty$ such that

$$\lambda(\cdot) \leq \bar{\lambda}, \quad \mu(\cdot) \leq \bar{\mu}, \quad \alpha(x, y) \leq \bar{\alpha}.$$

(A2) For all $x \in \mathcal{X}$, $M(x, \mathrm{d}h)$ is absolutely continuous with respect to the Lebesgue measure on $\mathcal{R}^l$ with density $m(x, h)$, and there exists a function $\bar{m} \colon \mathbb{R}^l \to [0, \infty)$ such that $m(x, h) \leq \bar{m}(h)$ for all $x \in \mathcal{X}$ and $h \in \mathbb{R}^l$, and $\int \bar{m}(h) \mathrm{d}h < \infty$.

(A3) For all $x \in \mathcal{X}$, $a(x) > 0$ and $\lambda(x) - \mu(x) > 0$, and there exists $\underline{\alpha} > 0$ such that $\alpha(\cdot, \cdot) \geq \underline{\alpha}$.

Let us introduce the notation $\langle \nu, f \rangle$ for the integral of the measurable function f against the measure ν, and let $\mathbf{1}$ denote the constant 1 function. Let further $\mathcal{M}_F$ denote the set of finite nonnegative Borel measures on $\mathcal{X}$.

Then, for fixed K, under (A1) and (A2) and assuming that $\mathbb{E}(\langle \nu_0^K, \mathbf{1} \rangle) < \infty$, the existence and uniqueness in law of a process with infinitesimal generator L^K was proven in another, even earlier pioneering work on stochastic adaptive dynamics by Fournier and Méléard [FM04]; we omit the details here. (When $K \to \infty$, they also proved, under more restrictive assumptions and assuming the convergence of the initial condition, the convergence on the space $\mathbb{D}([0,\infty), \mathcal{M}_F)$ (of càdlàg paths on $\mathcal{M}_F$ with the usual Skorokhod topology [4]) of the process ν^K to a deterministic process being solution to a nonlinear integro-differential equation. Here we will only use particular cases of some of their results, stated in Section 1.7 below, that can be proved under assumptions (A1) and (A2).)

[C06] focused on the case where the coexistence between two different traits is impossible on the long time scale. To state this assumption mathematically, we first introduce and interpret a couple of further key quantities. For $x, y \in \mathcal{X}$, we denote by

$$\bar{n}_x = \frac{\lambda(x) - \mu(x)}{\alpha(x,x)}$$

the *equilibrium population size* of trait x. Vaguely speaking, it is called equilibrium population size because under suitable conditions (which we will spell out later), in absence of mutations, the size of a monomorphic population consisting only of individuals of trait x divided by K converges on any fixed finite time interval to the solution to the *logistic* ODE

$$\dot{n}_x(t) = n_x(t)(\lambda(x) - \mu(x) - \alpha(x,x) n_x(t)). \tag{1.3}$$

If $\lambda(x) > \mu(x)$ (which we assume in (A3)), then this quadratic ODE has two equilibria (i.e., points where the right-hand side becomes zero and thus the solution started from the point stays there for all times), namely 0 and $\bar{n}_x > 0$. We will see that in this case, 0 is unstable, and $\bar{n}_x$ is globally asymptotically stable in the sense that any solution with a positive initial condition converges to it as $t \to \infty$.

Exercise 1. *1. Prove that this convergence is monotone in t for any positive initial condition.*

2. Solve the ODE (1.3) *via a separation of variables.*

Next, we put

$$\beta(x) = a(x)\lambda(x)\bar{n}_x. \tag{1.4}$$

This can be interpreted as the mutation rate in a population of trait x living in equilibrium. Finally, we define

$$f(y,x) = \lambda(y) - \mu(y) - \alpha(y,x)\bar{n}_x. \tag{1.5}$$

In more recent works in stochastic adaptive dynamics and population dynamics, this quantity is commonly referred to as the *invasion fitness* of a mutant of type y in a monomorphic resident population of type x (although this expression did not yet appear in [C06]). To interpret this quantity, imagine that K is very large and there are $K(1 \pm o(1))\bar{n}_x$ residents of trait x in a population living close to equilibrium, and now a mutant of trait y emerges. Then, the progeny of this mutant will have birth rate $\lambda(y)$ and natural death rate $\mu(y)$. Further, since the competitive pressure exerted by one single individual on another individual is of order $1/K$, mutants feel essentially no competitive pressure coming from each other but only from the residents, whose population size rescaled by K is nearly constant equal to $\bar{n}_x$. This competitive pressure is expressed by the term $\alpha(y,x)\bar{n}_x$. Hence, $f(y,x)$ is the approximate net growth rate of the mutant population.

The approximation described here (resident population divided by K nearly constant, mutant population competing only with residents) will be the basis of the *branching process approximation* of the mutant population during the initial phase of an invasion, which we will get to know soon. We will see that the

[4] Here we mean the usual Skorokhod topology on the space of càdlàg paths on $\mathcal{M}_F$, see e.g. [EK86, Section 5.3] for the definition and main properties of this topology and the Skorokhod J1 metric generating it.

positivity of the invasion fitness is often equivalent to a positive probability of mutant invasion. Under a further assumption excluding coexistence, invasion will also lead to fixation, i.e., mutants will reach a population size of $K(1 \pm o(1))\bar{n}_y$ and residents will die out. Let us now provide this assumption.

(B) Given any $x \in \mathcal{X}$, Lebesgue almost any $y \in \mathcal{X}$ satisfies one of the following two conditions:

$$\text{either } (\lambda(y) - \mu(y))\alpha(x,x) - (\lambda(x) - \mu(x))\alpha(y,x) < 0, \tag{1.6}$$

$$\text{or } (\lambda(y) - \mu(y))\alpha(x,x) - (\lambda(x) - \mu(x))\alpha(y,x) > 0 \text{ and } (\lambda(x) - \mu(x))\alpha(y,y) - (\lambda(y) - \mu(y))\alpha(x,y) < 0. \tag{1.7}$$

Exercise 2. *Check that condition* (1.6) *is equivalent to* $f(y,x) < 0$*, whereas condition* (1.7) *is equivalent to* $f(y,x) > 0$ *and* $f(x,y) < 0$*.*

The TSS (trait substitution sequence) model of evolution that we obtain from this individual-based model is a Markov jump process in the trait space $\mathcal{X}$ with infinitesimal generator

$$A\varphi(x) = \int_{\mathbb{R}^l} (\varphi(x+h) - \varphi(x))\beta(x)\frac{(f(x+h,x))_+}{\lambda(x+h)} m(x,h)\mathrm{d}h \tag{1.8}$$

for any bounded measurable function $\varphi\colon \mathcal{X} \to \mathbb{R}$, where $(b)_+ = [b]_+$ denotes the positive part of $b \in \mathbb{R}$, and where $\beta(x)$ and $f(y,x)$ are defined in (1.4) and (1.5), respectively. The existence and uniqueness in law of a process with generator A holds as soon as $\beta(x)(f(y,x))_+/\lambda(y)$ is bounded (see [EK86]), which is true under assumption (A) (which yields $(f(y,x))_+/\lambda(y) \le 1$). In biological terms, thanks to the positive part function in (1.8), the TSS process can only jump from a trait x to traits $x+h$ such that $f(x+h,x) > 0$, i.e. to traits fitter than x.

For any two functions $f, g\colon (0,\infty) \to \mathbb{R}$, we write $f(K) \ll g(K)$ (or $g(K) \gg f(K)$) if and only if $f(K)/g(K) \to 0$ as $K \to \infty$. In terms of standard Landau notation, this is expressed as $f(K) = o(g(K))$ (or $g(K) = \omega(f(K))$), but it is often convenient to use the notation involving the "$\ll$" or "$\gg$" symbols. We further write $[n] = \{1, 2, \dots, n\}$ for $n \in \mathbb{N}_0$, in particular, $[0] = \emptyset$. We are now able to state the main result of [C06].

Theorem 1.1 ([C06])**.** *Assume (A) and (B). Fix a sequence* $(u_K)_{K\in\mathbb{N}}$ *in* $[0,1]^{\mathbb{N}}$ *such that*

$$\forall V > 0, \quad \exp(-VK) \ll u_K \ll \frac{1}{K \log K}. \tag{1.9}$$

Fix also $x \in \mathcal{X}$*,* $\gamma > 0$*, and a sequence of* $\mathbb{N}$*-valued random variables* $(\gamma_K)_{K\in\mathbb{N}}$ *such that* $(\gamma_K/K)_{K\in\mathbb{N}}$ *converges in probability to* γ *and is bounded in* L^1*. Consider the process* $(\nu_t^K)_{t\ge 0}$ *with generator* (1.8) *with initial state* $(\gamma_K/K)\delta_x$*. Then, for any* $n \ge 1$*,* $\varepsilon > 0$*, and* $0 < t_1 < t_2 < \ldots < t_n < \infty$*, and for any measurable subsets* $\Gamma_1, \dots, \Gamma_n$ *of* $\mathcal{X}$*,*

$$\lim_{K\to\infty} \mathbb{P}\big(\forall i \in [n], \exists x_i \in \Gamma_i\colon \mathrm{supp}(\nu^K_{t_i/(Ku_K)}) = \{x_i\} \text{ and } |\langle \nu^K_{t_i/(Ku_K)}, \mathbf{1}\rangle - \bar{n}_{x_i}| < \varepsilon\big) = \mathbb{P}(\forall i \in [n], X_{T_i} \in \Gamma_i\big) \tag{1.10}$$

where for all $\nu \in \mathcal{M}_F$*,* $\mathrm{supp}(\nu)$ *denotes the support of* ν *and* $(X_t)_{t\ge 0}$ *is the TSS process with generator* (1.8) *and initial state* x*.*

Remark 1.2. Let us interpret certain ingredients of Theorem 1.1.

(i) The time scale $1/(Ku_K)$ is the time scale of the mutation events for the process ν^K (the population size is proportional to K and the individual mutation rate to u_K).

(ii) The second inequality in Assumption (1.9) leads to the separation of the ecological time scale (i.e., the time scale of birth and death events) and the evolutionary one (i.e., the one of mutations). This will guarantee that each mutation goes to fixation or extinction with high probability before the appearance of the next mutant. Such a rareness of mutations ensures that in the scaling limit, *clonal interference*,

i.e., the competitive interaction between multiple beneficial mutations fighting simultaneously for survival plays no role. For population-dynamic models with some outlook to a possible scaling limit in the case of recurrent mutations, we refer the reader to [BS17, BS19]. We will discuss mutation regimes with less rare mutations in more general in Section 4.1.

(iii) The limit in (1.10) means that, when this time scale separation occurs, the population is monomorphic at any time with high probability, and the transition periods corresponding to the invasion of a mutant trait in the resident population (between the appearance of a mutant that invades successfully and the extinction of the former resident) are infinitesimal on this mutation time scale.

(iv) Unlike in classical population-genetic models like the Wright–Fisher or the Moran model, in our model the population size is not constant. This leads to an almost sure extinction of the population in the case of any (finite) initial condition, and the typical time scale until extinction is exponential in K. The first inequality in (1.9) is needed (unlike in the case of a constant population size) in order to ensure that mutations do not come so rarely that the population is likely to die out between them. We will discuss this assumption in more detail in Remark 1.18.

(v) Note also that the convergence in (1.10) is a *convergence of finite-dimensional distributions*.

Theorem 1.1 has the following corollary.

Corollary 1.3 ([C06])**.** *Assume additionally in Theorem 1.1 that $(\gamma_K/K)_{K\in\mathbb{N}}$ is bounded in L^p for some $p > 1$. Then the process $(\nu_{t/(Ku_K)})_{t\geq 0}$ converges in the sense of finite-dimensional distributions for the topology on $\mathcal{M}_F$ induced by the functions $\nu \mapsto \langle \nu, f\rangle$ with f bounded and measurable on $\mathcal{X}$, to the process $(Y_t)_{t\geq 0}$ defined by*

$$Y_t = \begin{cases} \gamma\delta_x, & \text{if } t = 0, \\ \bar{n}_{X_t}\delta_{X_t}, & \text{if } t > 0. \end{cases}$$

This corollary is a consequence of the following long-time moment estimates:

Lemma 1.4 ([C06])**.** *Assume (A) and that $\sup_{K\geq 1}\mathbb{E}(\langle \nu_0^K, \mathbf{1}\rangle^p) < \infty$ for some $p \geq 1$, then we have*

$$\sup_{K\geq 1}\sup_{t\geq 0}\mathbb{E}(\langle \nu_t^K, \mathbf{1}\rangle^p) < \infty,$$

and therefore, if $p > 1$, then the family of random variables $(\langle \nu_t^K, \mathbf{1}\rangle_{K\geq 1, t\geq 0}$ is uniformly integrable.

The proof of this lemma can be found in Appendix A. Based on the lemma, the proof of the corollary goes as follows.

Proof of Corollary 1.3. Let Γ be a measurable subset of $\mathcal{X}$. Let us prove that

$$\lim_{K\to\infty}\mathbb{E}\big(\langle \nu^K_{t/(Ku_K)}, \mathbb{1}_\Gamma\rangle\big) = \mathbb{E}\big(\bar{n}_{X_t}\mathbb{1}_{\{X_t\in\Gamma\}}\big). \tag{1.11}$$

Fix $\varepsilon > 0$, and observe that by Assumption (A) we have $\bar{n}_x \in [0, \bar{b}/\underline{\alpha}]$. Write $[0, \bar{b}/\underline{\alpha}] \subseteq \bigcup_{i=1}^q I_i$, where q is the smallest integer greater than $\bar{b}/\varepsilon\underline{\alpha}$ and $I_i = [(i-1)\varepsilon, i\varepsilon)$. Define $\Gamma_i = \{x \in \mathcal{X}\colon \bar{n}_x \in I_i\}$ for $1 \leq i \leq q$, and apply (1.10) to the sets $\Gamma\cap\Gamma_1, \dots, \Gamma\cap\Gamma_q$ with $n = 1$, $t_1 = t$ and ε as above. Then, by Lemma 1.4,

there exists $C > 0$ such that

$$\begin{aligned}
\limsup_{K\to\infty} \mathbb{E}(\langle \nu^K_{t/(Ku_K)}, \mathbb{1}_\Gamma\rangle) &\le \limsup_{K\to\infty} \mathbb{E}(\langle \nu^K_{t/(Ku_K)}, \mathbb{1}_\Gamma\rangle \mathbb{1}_{\{\langle \nu^K_{t/(Ku_K)}, \mathbf{1}\rangle \le C\}}) + \varepsilon \\
&\le \sum_{i=1}^{q} \limsup_{K\to\infty} \mathbb{E}\big(\langle \nu^K_{t/(Ku_K)}, \mathbb{1}_{\Gamma\cap\Gamma_i}\rangle \mathbb{1}_{\{\langle \nu^K_{t/(Ku_K)}, \mathbf{1}\rangle \le C\}}\big) + \varepsilon \\
&\le \sum_{i=1}^{q} (i+1)\varepsilon \mathbb{P}(X_t \in \Gamma \cap \Gamma_i) + \varepsilon \\
&\le \sum_{i=1}^{q} \big(\mathbb{E}(\bar{n}_{X_t} \mathbb{1}_{\{X_t\in\Gamma\cap\Gamma_i\}}) + 2\varepsilon \mathbb{P}(X_t \in \Gamma_i)\big) + \varepsilon \\
&\le \mathbb{E}(\bar{n}_{X_t} \mathbb{1}_{\{X_t\in\Gamma\}}) + 3\varepsilon.
\end{aligned}$$

A similar estimate for the lim inf finishes the proof of (1.11), which implies the convergence of one-dimensional laws for the required topology (is the latter conclusion clear?). The same method gives the required limit when we consider a finite number of times $t_1, \dots, t_n$. □

Remark 1.5. It is not claimed in Corollary 1.3 that the convergence holds in distribution in the space of càdlàg paths from $[0,\infty)$ to $\mathcal{M}_F$ w.r.t. the Skorokhod topology, and that assertion is actually not true. The problem is the missing continuity of the limit at time 0+. Regarding this, we do not go into detail here; we refer the interested reader to [C06, Proposition 1] and its proof. We find the interpretation of this discontinuity more important: Started at any initial condition γ_K/K tending to $\gamma > 0$ in probability, the process reaches an arbitrarily small (fixed) neighbourhood of the equilibrium $\bar{n}_{X_0} = \bar{n}_x$ in $o(Ku_K)$ time.

Remark 1.6 (The canonical equation of adaptive dynamics). In Theorem 1.1, the TSS model is obtained in the limit of a large carried capacity combined with rare mutations from the individual-based model. As mentioned earlier, taking the limit of small mutational effects in the TSS model, under suitable additional assumptions, we obtain the *canonical equation of adaptive dynamics* (CEAD), see [CFB01, Theorem 2]. The canonical equation reads

$$\frac{\mathrm{d}\widehat{s}}{\mathrm{d}t} = u(\widehat{s}) \frac{\sigma_0^2(\widehat{s})}{2} n(\widehat{s}) \partial_1 f(\widehat{s}, \widehat{s}),$$

where s denotes a scalar trait, $\widehat{s}(t)$ denotes the mean of the distribution of the trait value s at time $t \ge 0$, $u(s)$ is the probability that a birth from an individual of trait s gives rise to a mutation, $\sigma_0^2(s)$ denotes the variance of the distribution of the mutant trait s' born from an individual of trait s, whose probability density function is denoted by $M(s, s'-s)$ and supposed to be symmetric with respect to $s'-s$, $n(s)$ is the equilibrium population size (which is supposed to exist and be positive) in a population composed only of individuals of trait s, $\partial_1 f(s,s)$ denotes the first partial derivative of the fitness function $f(s',s)$ of a mutant individual of trait s' in a population composed only of individuals with trait s. Fixed points of the canonical equation are the points where the fitness gradient $\partial_1 f(s,s)$ vanishes, these are called *evolutionary singularities*.

While [C06] gave a clear microscopic interpretation to the TSS model, the relation between the original individual-based model and the CEAD was still not that clear based on his paper because of the two separate, consecutive limits. Later, in a paper by Baar, Bovier and Champagnat [BBC17] it was showed that the two consecutive limits in [C06] can be coupled in order to obtain the same convergence in *one step*. In models with multiple mutations and coupled limits, the traces of methods based on dynamical systems and branching processes can still be found, but the approximating objects often become more complex than in single-mutation models.

1.5 Stability of equilibria of the dynamical systems

In order to explain the idea of the proof of Theorem 1.1, we need to introduce some further definitions and notations. While the theorem tells about the empirical measure $(\nu_t^K)_{t\ge 0}$ scaled properly, the following definition provides the necessary notation for the corresponding birth-and-death processes.

Definition 1.7. (a) For any $K \geq 1$, $\lambda, \mu, \alpha \geq 0$ and for any $\mathbb{N}/K$-valued random variable Z, we will denote by $\mathbb{P}^K(\lambda, \mu, \alpha, Z)$ the law of the $\mathbb{N}/K$-valued Markov birth-and-death process with initial state Z and with transitions

$$i/K \to (i+1)/K \text{ at rate } i\lambda,$$
$$i/K \to (i-1)/K \text{ at rate } i(\mu + \alpha i/K).$$

(b) For any $K \geq 1$, $\lambda_k, \mu_k, \alpha_{kl} \geq 0$ with $k, l \in \{1, 2\}$, and for any $\mathbb{N}/K$-valued random variables Z_1 and Z_2, we will denote by

$$\mathbb{Q}^K(\lambda_1, \lambda_2, \mu_1, \mu_2, \alpha_{11}, \alpha_{12}, \alpha_{21}, \alpha_{22}, Z_1, Z_2)$$

the law of the $(\mathbb{N}/K)^2$-valued Markov birth-and-death process with initial state (Z_1, Z_2) and with transition rates

$$(i/K, j/K) \to ((i+1)/K, j/K) \text{ at rate } i\lambda_1,$$
$$(i/K, j/K) \to ((i-1)/K, j/K) \text{ at rate } i(\mu_1 + (\alpha_{11}i + \alpha_{12}j)/K),$$
$$(i/K, j/K) \to (i/K, (j+1)/K) \text{ at rate } j\lambda_2,$$
$$(i/K, j/K) \to (i/K, (j-1)/K) \text{ at rate } j(\mu_2 + (\alpha_{21}i + \alpha_{22}j)/K),$$

These two Markov chains have absorbing states at 0 and $(0, 0)$, respectively. Observe also that, when $\alpha = 0$, the Markov process of point (a) is a continuous-time binary branching process divided by K. Similarly, if each α_{ij} equals zero, the two coordinates of the Markov process of point (b) are independent binary branching processes divided by K. Now we make the connection between the rescaled birth-and-death processes appearing in Definition 1.7 and (systems of) logistic ODEs precise. The proof of the following results can be found in [EK86, Chapter 11].

Proposition 1.8 ([EK86, C06])**.** *Let $T > 0$.*

(a) Assume that $a \equiv 0$ and $\nu_0^K = N_x^K(0)\delta_x$. Then, for any $t \geq 0$, $\nu_t^K = N_x^K(t)\delta_x$, where N_x^K has the law $\mathbb{P}^K(\lambda(x), \mu(x), \alpha(x, x), N_x^K(0))$.
Assume that $N_x^K(0) \to n_x(0)$ in probability when $K \to \infty$. Then, the sequence $(N_x^K)_{K\geq 1} = ((N_x^K(t))_{t\geq 0})_{K\geq 1}$ converges in probability in $[0, T]$ in the uniform norm (supremum norm) when $K \to \infty$ to the deterministic function $n_x = (n_x(t))_{t\geq 0}$ that is solution to the ODE (1.3) with initial condition $n_x(0)$.

(b) Assume that $a \equiv 0$ and $\nu_0^K = N_x^K(0)\delta_x + N_y^K(0)\delta_y$. Then, for any $t \geq 0$, $\nu_t^K = N_x^K(t)\delta_x + N_y^K(t)\delta_y$, where (N_x^K, N_y^K) has the law

$$\mathbb{Q}^K(\lambda(x), \lambda(y), \mu(x), \mu(y), \alpha(x, x), \alpha(x, y), \alpha(y, x), \alpha(y, y), N_x^K(0), N_y^K(0)).$$

Assume that $N_x^K(0) \to n_x(0)$ and $N_y^K(0) \to n_y(0)$ in probability when $K \to \infty$. Then, (N_x^K, N_y^K) converges in probability in $[0, T]$ in the uniform norm to the deterministic function $(n_x, n_y) = ((n_x(t), n_y(t))_{t\geq 0}$ when $K \to \infty$ with initial condition $(n_x(0), n_y(0))$, which is the solution to the two-dimensional competitive Lotka–Volterra system

$$\begin{aligned} \dot{n}_x(t) &= (\lambda(x) - \mu(x) - \alpha(x, x)n_x(t) - \alpha(x, y)n_y(t))n_x(t), \\ \dot{n}_y(t) &= (\lambda(y) - \mu(y) - \alpha(y, x)n_x(t) - \alpha(y, y)n_y(t))n_y(t). \end{aligned} \tag{1.12}$$

It is easy to see that the corresponding positive orthant is *positively invariant* under both systems, i.e., if the initial condition is coordinatewise positive, so is the solution at each positive time. If this did not hold, the system would not be biologically reasonable because (rescaled) population sizes could eventually turn negative. This property will be true for all systems of ODEs that appear in these lecture notes as large-K limits of rescaled population sizes.[5]

[5]It is well-known that in general for *one-dimensional* ODEs (like (1.3)), local stability of an equilibrium and global stability of the same equilibrium on a positively invariant set containing the equilibrium coincide. It is also classical that a nonnegative solution to a one-dimensional ODE under which $[0, \infty)$ is positively invariant either tends to $+\infty$ or to an equilibrium.

We have already discussed the question of (global) stability of the equilibria (0 and $\bar{n}_x$) of the ODE (1.3). The system (1.12) has at least three equilibria: $(0,0)$, $(\bar{n}_x,0)$, and $(0,\bar{n}_y)$. The reader may already be aware of the fact that for higher-dimensional systems of ODEs, local and global stability may not coincide. The question of local stability can often be determined via linearization: One determines the Jacobi matrix consisting of the partial derivatives of the right-hand sides of the equation w.r.t. all variables and writes it in matrix form. For example, in the case of (1.12), the Jacobi matrix around an equilibrium of the form $(\widetilde{n}_x,\widetilde{n}_y)\in[0,\infty)^2$ reads as

$$A(\widetilde{n}_x,\widetilde{n}_y)=\begin{pmatrix}\lambda(x)-\mu(x)-2\alpha(x,x)\widetilde{n}_x-\alpha(x,y)\widetilde{n}_y & -\alpha(x,y)\widetilde{n}_x\\ -\alpha(y,x)\widetilde{n}_y & \lambda(y)-\mu(y)-\alpha(y,x)\widetilde{n}_x-2\alpha(y,y)\widetilde{n}_y\end{pmatrix}. \tag{1.13}$$

The following summary of (Lyapunov) stability theory may also be a repetition for some readers.

- We say that the equilibrium is *(locally) asymptotically stable* if there exists $\varepsilon>0$ such that started from anywhere in an ε-ball around the equilibrium, the solution converges to the respective equilibrium as $t\to\infty$.
- We say that the equilibrium is *unstable* if there exists $\varepsilon>0$ such that for any $\delta>0$ there exists an initial condition within the δ-ball around the equilibrium starting from which the solution eventually leaves the ε-ball forever.
- Finally, the equilibrium is *(locally) stable* if for any $\varepsilon>0$ there exists $\delta>0$ such that if the initial condition lies within a δ-neighbourhood of the equilibrium, then the solution will be within the ε-neighbourhood of the equilibrium for all sufficiently large times.[6]

If all eigenvalues of the corresponding Jacobi matrix have negative real parts, then the equilibrium is asymptotically stable. If all eigenvalues have nonzero real parts and there is an eigenvalue with positive real part, then the equilibrium is unstable. Finally, if the equilibrium is *non-hyperbolic*, i.e., at least one eigenvalue has zero real part (otherwise we call it *hyperbolic*), then both stability and instability are possible. In the case of stability, it may also be the case that the equilibrium is asymptotically stable, but unlike for hyperbolic stable equilibria, the convergence to the equilibrium typically does not happen at an exponential speed but slower. Finally, (in the non-hyperbolic case) stability does not imply asymptotic stability.[7]

Summarizing, for hyperbolic equilibria, the *local* stability of equilibria can be determined by linearization, and the linearized version of the system, i.e., the linear system of ODEs with the same Jacobi matrix at the given equilibrium, exhibits the same local stability properties. If the equilibrium is non-hyperbolic, then the stability properties of the linearized and the original system may not be the same. Further, local asymptotic stability is necessary but not sufficient in order to guarantee the convergence of solutions to the system started from distant initial conditions (which is an assertion that one often needs in population dynamics, as we will see).

In the case of the system (1.12), we have

$$A(0,0)=\begin{pmatrix}\lambda(x)-\mu(x) & 0\\ 0 & \lambda(y)-\mu(y).\end{pmatrix}$$

By Assumption (A3), $\lambda(x)>\mu(x)$ and $\lambda(y)>\mu(y)$, and hence $(0,0)$ is unstable. (The eigenvalues are of course the diagonal entries and hence they are real. An equilibrium of a two-dimensional system with two positive real eigenvalues for the Jacobi matrix is called (locally) a *source*.)

The question of stability of the other eigenvalues depends also on the competition parameters. If we have *symmetric competition*, i.e., $\alpha(x,x)=\alpha(x,y)=\alpha(y,x)=\alpha(y,y)=\alpha>0$, then $\lambda(x)-\mu(x)=\lambda(y)-\mu(y)$ is

[6] Formally, the definition of stability is not exactly the opposite of the one of instability, but in practice it can be thought of like that.

[7] A typical example is given by the linear system $\dot{x}(t)=y(t),\dot{y}(t)=-\omega x(t)$, $\omega>0$, where at $(0,0)$ the Jacobi matrix has two purely imaginary eigenvalues, and trajectories of the system not started from $(0,0)$ are concentric circles around $(0,0)$. Such an equilibrium is called a (stable) center. Note that the aforementioned example is nothing but the reformulation of the harmonic oscillator ODE $\ddot{x}(t)=-\omega x(t)$ as a two-dimensional system of ODEs.

a critical case. Then, it is not only the case that $(\bar{n}_x, 0)$ and $(0, \bar{n}_y)$ are non-hyperbolic equilibria, but even any convex combination of these two eigenvalues is again a non-hyperbolic equilibrium. We will typically exclude this case, and the case of zero invasion fitnesses is difficult to treat in general. In the hyperbolic case, the following assertion of [I00] (mentioned also in [C06]) guarantees instability resp. a certain sense of global stability for some of the equilibria.

Proposition 1.9 ([I00])**.** *If x and y satisfy* (1.6)*, then $(\bar{n}_x, 0)$ is a stable equilibrium of* (1.12)*. If x and y satisfy* (1.7)*, then $(\bar{n}_x, 0)$ is unstable, $(0, \bar{n}_y)$ is stable, and any solution to* (1.12) *with initial condition in $(0, \infty)^2$ converges to $(0, \bar{n}_y)$ when $t \to \infty$.*

Exercise 3. *Assume that both invasion fitnesses $f(x, y)$ and $f(y, x)$ are nonzero. Under what conditions can both equilibria $(\bar{n}_x, 0)$ and $(0, \bar{n}_y)$ be simultaneously stable? Under what conditions can they be simultaneously unstable? Can the latter happen in the case of symmetric competition?*

Exercise 4. *Verify that in the case of symmetric competition,* (1.12) *cannot have any coordinatewise positive coexistence equilibrium apart from the critical case $\lambda(x) - \mu(x) = \lambda(y) - \mu(y)$. This assertion is known as* competitive exclusion principle.[8]

1.6 Outline of the proof of Theorem 1.1: the three phases of an invasion

The main ideas of the proof of Theorem 1.1 in [C06] are based on two ingredients. First, when $a \equiv 0$ and ν_0^K is monomorphic with trait x, it follows from Proposition 1.8 (a) that ν^K converges to $n(t)\delta_x$, where $n(t)$ is the solution to (1.3). Any solution to this equation with a positive initial condition converges to $\bar{n}_x$ as $t \to \infty$. We will employ Freidlin–Wentzell type large-deviation arguments [FW84] to this convergence in order to show that the time during which the stochastic process stays in a small neighbourhood of its limit is of order $\exp(KV)$ for a certain $V > 0$. Now, when u_K is small, the process ν^K with a monomorphic initial condition of trait x equals the same process with $a \equiv 0$ until the first time a mutant occurs. Thanks to the first inequality in (1.9), we will be able to prove that with high probability, the first mutation event (occurring on the time scale $t/(Ku_K)$) occurs before the total density drifts away from $\bar{n}_x$.

The second ingredient of Champagnat's proof is the study of the invasion of the mutant trait y that has just appeared in a monomorphic resident population of trait x. This invasion can be divided into three steps[9] as follows.

- *Phase I*: Firstly, as long as the mutant population size $\langle \nu_t^K, \mathbb{1}_{\{y\}} \rangle$ (initially equal to $1/K$) is smaller than some fixed but small $\varepsilon > 0$, the resident dynamics is very close to what it was before the mutation, so $\langle \nu_t^K, \mathbb{1}_{\{x\}} \rangle$ stays close to $\bar{n}_x$. Then, as already anticipated, the death rate of a mutant individual is close to the constant $\mu(y) + \alpha(y, x)\bar{n}_x$. Since its birth rate is constant equal to $\lambda(y)$, we can approximate the dynamics of the (non-rescaled!) mutant population size $K\langle \nu_t^K, \mathbb{1}_{\{y\}} \rangle$ by a binary branching process (whose transition rates are *linear* in the number of individuals). Hence, the probability that $\langle \nu_t^K, \mathbb{1}_{\{x\}} \rangle$ reaches ε is approximately equal to the probability that this branching process reaches εK, which converges when first $K \to \infty$ and then $\varepsilon \downarrow 0$ to the probability of non-extinction of the branching process.
  One computes the survival probability of a continuous-time branching process using the same first-step analysis as one does it for a Galton–Watson process in discrete time (since the latter is the embedded discrete-time chain of the former), and hence this probability equals $[f(y, x)]_+/\lambda(y)$, which is positive whenever the first inequality of (1.7) is satisfied; see Section 1.9 for a proof sketch. In that case, the branching process is supercritical. In case (1.6) holds, the branching process is (strictly) subcritical.
  Due to the aforementioned large-deviation results, it will follow that the resident population stays close to equilibrium at least until the mutant population size reaches εK or 0. In the presence of mutants, in the case of a successful mutant invasion, the rescaled resident population size will leave the vicinity

[8] It can also be shown that if (1.7) or the same set of inequalities with the roles of x and y swapped holds, then the system (1.12) cannot have any coordinatewise positive equilibrium.

[9] See [C06, Section 3] for references to similar results in population-genetic settings.

of $\bar{n}_x$ in $\Theta(\log K)$ time, due to the competitive disadvantage of residents compared to the mutants (cf. the first equation of (1.7)).

- *Phase II*: Secondly, once $\langle \nu_t^K, \mathbb{1}_{\{y\}} \rangle$ has reached ε, by Proposition 1.8 (b), for large K, ν^K is close to the solution to (1.12) with initial state $(\bar{n}_x, \varepsilon)$ with high probability. By Proposition 1.9, this solution will be shown to reach the ε-neighbourhood of $(0, \bar{n}_y)$ in finite time.

- *Phase III*: Finally, once $\langle \nu_t^K, \mathbb{1}_{\{y\}} \rangle$ is close to $\bar{n}_y$ and $\langle \nu_t^K, \mathbb{1}_{\{x\}} \rangle$ is small, one can approximate the resident population size $K\langle \nu_t^K, \mathbb{1}_{\{x\}} \rangle$ by a binary branching process, which is subcritical whenever the second inequality of (1.7) holds, and thus it gets extinct almost surely.

We will see that the first phase takes $\Theta(\log K)$ time; the precise prefactor of $\log K$ does depend on ε but it can be chosen uniformly bounded over all sufficiently small $\varepsilon > 0$, and the prefactor converges to a positive number as $\varepsilon \downarrow 0$. Where does this $\log K$ come from? Conditional on survival, the approximating branching process grows roughly exponentially at a certain rate, and hence reaching a size of order K takes an order of $\log K$ time. The same time scaling (with a possibly different limiting prefactor) applies to the third phase; there, a subcritical branching process with initially order K individuals decays exponentially and then dies out.

The time scale of the dynamical system (1.12) is finite (independently of K), but the common folklore statement that Phase II takes $O(1)$ time is not entirely correct because even though the duration of Phase II stays bounded as $K \to \infty$ for any fixed $\varepsilon > 0$, it tends to infinity as $\varepsilon \downarrow 0$. Taking $K \to \infty$ (with $u_K \downarrow 0$ according to (1.9)), the total duration of a successful invasion is $\Theta(\log K)$ with a precise limiting prefactor, and unsuccessful invasions take $o(\log K)$ time (in fact, even $o(h(K))$ time for any $h\colon (0,\infty) \to (0,\infty)$ increasing that tends to infinity as $K \to \infty$).

Vaguely speaking, thanks to the second inequality of (1.9), all mutants will reach fixation or go extinct (leaving the current resident untouched) until the birth of the next mutant with high probability as $K \to \infty$, i.e., clonal interference plays no role in the limit. Scaling time by $1/(Ku_K)$, the duration of the three phases of an invasion (where both the resident and the mutant are present) vanishes, and we indeed obtain a jump process, namely the TSS model, as scaling limit.

Remark 1.10 (More general initial conditions). It was observed in [M96] that the biological heuristics leading to the TSS model extend to the case of a polymorphic initial condition where the population is composed of a finite number of distinct traits. The mathematical methods of [C06] can also be extended easily to n-morphic initial conditions, expect for one difficulty: One has to replace assumption (B) with another assumption stating that, for n under consideration, any solution to the n-morphic logistic system generalizing (1.12) converges to an equilibrium (as in Proposition 1.9), and that the equilibria of these systems are hyperbolic, in the sense that the branching processes in the first and third steps above are not critical, or, equivalently, that a first-order linear analysis of these equilibria allows to determine their local stability. Then, one could construct a polymorphic TSS model in which the number of coexisting traits is not fixed. However, the asymptotic analysis of n-dimensional logistic systems is non-trivial and may exhibit cyclic behaviour or chaos, except when $n = 1$ or $n = 2$, and analytical assumptions ensuring the condition above are difficult to find.

Remark 1.11 (More frequent mutations and clonal interference). According to the above observations regarding scaling, if one wants to study the effects of clonal interference, the most interesting scaling regime is when $u_K \asymp 1/(K \log K)$. Then, the duration of a successful invasion is of the same order as the time between two consecutive mutations. Therefore, the number of mutants trying to fix in the population is typically of finite order, but not almost always 0 or 1. A mutant surviving initial fluctuations may still not become resident in case there is an even fitter mutant who "overtakes" it and reaches the size εK earlier. Further, a mutant who has become resident may as well be wiped out from the population by another mutant who is fitter but was born too late to become resident first. Such effects were investigated in [BS17], also in the case of asymmetric competition, where the interaction between three traits was studied. The authors showed that under certain conditions, one can e.g. observe a "rock–paper–scissors cycle", where three mutants of traits are periodically resident. Such a cycle requires the lack of *transitivity* of competitive relations, where transitivity

means that $f(y,x) > 0$ and $f(z,y) > 0$ implies $f(z,x) > 0$ for all $x, y, z \in \mathcal{X}$. Indeed, a rock–paper–scissors cycle starting with the residency of, say, x, and followed by first y and then z, requires that $f(y,x) > 0$, $f(z,y) > 0$, and $f(x,z) > 0$ (and further conditions).

In [BS19] it was mentioned that $u_K \asymp 1/(K \log K)$ is the relevant mutation frequency regime in order to see an interesting scaling limit, but the case of a diverging number of mutations was not studied there. We will analyse settings with even more frequent mutations and arising interesting effects in Section 4.

The proof of Theorem 1.1 is too long and technical to present it completely at our course, but apart from this overview, we will also go into detail regarding the large-deviation estimates, important properties of branching processes (e.g., their growth rate, which determines the prefactor of $\log K$ at an invasion), and some other key techniques. Interested readers can find the (greatly written) proof in [C06].

1.7 Comparison results and Poissonian construction

To provide the results on comparison and coupling of birth-and-death processes that are essential for the proof of Theorem 1.1, let us first introduce the necessary notation and definitions. For any $K \geq 1$ and $\nu \in \mathcal{M}^K$, we will denote by $\mathbb{P}^K_\nu$ the law of the process with generator (1.2) with initial state ν, and by $\mathbb{E}^K_\nu$ the expectation with respect to $\mathbb{P}^K_\nu$. The law of a stochastic process $Z = (Z_t)_{t\geq 0}$ will be denoted by $\mathcal{L}(Z)$.

Definition 1.12 (Stochastic domination of laws in the context of [C06])**.** We will denote by $\preceq$ the following stochastic domination relation: If $\mathbb{Q}_1$ and $\mathbb{Q}_2$ are the laws of $\mathbb{R}$-valued processes, we will write $\mathbb{Q}_1 \preceq \mathbb{Q}_2$ if we can construct on the same probability space $(\Omega, \mathcal{F}, \mathbb{P})$ two processes $X^1 = (X^1_t)_{t\geq 0}$ and $X^2 = (X^2_t)_{t\geq 0}$ such that $\mathcal{L}(X^i) = \mathbb{Q}_i$ $(i = 1, 2)$ and $\forall t \geq 0$, $\forall \omega \in \Omega$, $X^1_t(\omega) \leq X^2_t(\Omega)$.

Definition 1.13. If $X^1 = (X^1_t)_{t\geq 0}$ and $X^2 = (X^2_t)_{t\geq 0}$ are two stochastic processes and T is a random time constructed on the same probability space as X^1, we will write "$X^1_t \preceq X^2_t$ for $t \leq T$" if we can construct a process $\widehat{X}^2 = (\widehat{X}^2_t)_{t\geq 0}$ on the same probability space as X^1, such that $\mathcal{L}(\widehat{X}^2) = \mathcal{L}(X^2)$ and $\forall t \leq T$, $\forall \omega \in \Omega$, $X^1_t(\omega) \leq \widehat{X}^2_t(\omega)$.

The following theorem provides various stochastic domination results.

Theorem 1.14 ([C06])**.** *(a) Assume (A). For any $K \geq 1$ and any integrable initial condition ν^K_0 of the process ν^K,*

$$\mathcal{L}(\langle \nu^K, 1\rangle) \preceq \mathbb{P}^K(2\bar{\lambda}, 0, \underline{\alpha}, \langle \nu^K_0, \mathbf{1}\rangle),$$

where we recall the law $\mathbb{P}^K$ from Definition 1.7.

(b) Under the same assumptions as in (a), let A^K_t denote the number of mutations occurring in ν^K between times 0 and t, and let $a, a_1, a_2 \geq 0$. Then, for $t \leq \inf\{s \geq 0 \colon \langle \nu^K_s, \mathbf{1}\rangle \geq a\}$,

$$A^K_t \preceq B^K_t,$$

where B^K_t is a Poisson process with parameter $Ku_K a\bar{b}$. If further $\nu^K_0 = \langle \nu^K_0, 1\rangle \delta_x$, define $\tau_1 = \inf\{t \geq 0 \colon A^K_t = 1\}$ (the first mutation time). Then, for $t \leq \tau_1 \wedge \inf\{s \geq 0 \colon \langle \nu^K_s, \mathbf{1}\rangle \notin [a_1, a_2]\}$,

$$B^K_t \preceq A^K_t \preceq C^K_t, \tag{1.14}$$

where $B^K = (B^K_t)_{t\geq 0}$ and $C^K = (C^K_t)_{t\geq 0}$ are Poisson processes with respective parameters $Ku_K a_1 a(x)b(x)$ and $Ku_K a_2 a(x)b(x)$.

(c) Fix $K \geq 1$ and take b, d, α, z as in Definition 1.7 (a). Then, for any $\varepsilon_1, \varepsilon_2, \varepsilon_3 \geq 0$ and any $\mathbb{N}/K$-valued random variable $\varepsilon/4$,

$$\mathbb{P}^K(b, d + \varepsilon_2, \alpha + \varepsilon_3, z) \preceq \mathbb{P}^K(b + \varepsilon_1, d, \alpha, z + \varepsilon_4).$$

Let $(Z^1, Z^2) = ((Z^1_t, Z^2_t))_{t\geq 0}$ be a stochastic process with law

$$\mathbb{Q}^K(b_1, b_2, d_1, d_2, \alpha_{11}, \alpha_{12}, \alpha_{21}, \alpha_{22}, z_1, z_2)$$

where the parameters are as in Definition 1.7 (b). Fix $a > 0$ and define $T = \inf\{t \geq 0 \colon Z_t^2 \geq a\}$. Then, for $t \leq T$,

$$M_t^1 \preceq Z_t^1 \preceq M_t^2,$$

where $\mathcal{L}(M^1) = \mathbb{P}^K(b_1, d_1 + a\alpha_{12}, \alpha_{11} z_1)$ and $\mathcal{L}(M^2) = \mathbb{P}^K(b_1, d_1, \alpha_{11}, z_1)$.

(d) Take (Z^1, Z^2) as above, fix $0 \leq a_1 < a_2$ and $a > 0$, and define $T = \inf\{t \geq 0 \colon Z^1 \notin [a_1, a_2]$ or $Z^2 \geq a\}$. Then, for $t \leq T$,

$$M_t^2 \preceq Z_t^2 \preceq M_t^2,$$

where $\mathcal{L}(M^1) = \mathbb{P}^K(b_2, d_2 + a_2\alpha_{21} + a\alpha_{22}, 0, z_2)$ and $\mathcal{L}(M^2) = \mathbb{P}^K(b_2, d_2 + a_1\alpha_{21}, 0, z_2)$.

Remark 1.15. Point (a) explains why it is necessary to combine simultaneously the limits $K \to \infty$ and $u_K \downarrow 0$ in order to obtain the TSS process in Theorem 1.1. The limit $K \to \infty$ taken alone leads to a deterministic dynamics (which had already been shown in [FM04] before [C06] was written), so taking the limit of rare mutations afterwards cannot lead to a stochastic process. Conversely, taking the limit of rare mutations without letting the population size diverge would lead to an immediate extinction of the population on the time scale of mutations, because the stochastic domination of Theorem 1.14 (a) is independent of u_K and because the process Z with law $\mathbb{P}^K(2\bar{b}, 0, \underline{\alpha}, \gamma_K/K)$ goes extinct a.s. in finite time.

Before proving Theorem 1.14, let us mention that Lemma 1.4 can be derived from part (a) of the theorem. The proof can be found in Appendix A (or in [C06, Section 4.1]).

Proof of Theorem 1.14. The proof is essentially intuitive if one computes upper and lower bounds of the birth and death rates for each process considered in the statement of the theorem. We will simply give an explicit construction of the process ν^K, commonly known as the *Poissonian construction*[10] of the process ν^K, and we will give the precise proof of (1.14) as an example. We leave the proofs of the remaining comparison results to the reader (as it was also done in [C06]). We start via recalling the notion of Poisson point measure and Poisson point process.

Definition 1.16. Let $(S, \mathcal{S}, \nu)$ be an arbitrary σ-finite measure space, and $(\Omega, \mathcal{F}, \mathbb{P})$ a probability space. Let $P \colon \mathcal{S} \to \{0, 1, 2, \ldots\} \cup \{\infty\}$ be such that the family $\{P(A) \colon A \in \mathcal{S}\}$ are random variables defined on $(\Omega, \mathcal{F}, \mathbb{P})$. Then P is called a *Poisson random measure* or *Poisson point measure* on S with intensity measure ν if

(i) for any $n \in \mathbb{N}$, for mutually disjoint $A_1, A_2, \ldots, A_n \in \mathcal{S}$ the random variables $P(A_1), \ldots, P(A_n)$ are independent,

(ii) for any $A \in \mathcal{S}$, $P(A)$ is Poisson distributed with parameter $\nu(S) \in [0, \infty]$[11],

(iii) $\mathbb{P}$-almost surely, P is a measure.

Let us consider the case when S is a topological space with Borel σ-algebra $\mathcal{S}$, and ν is a measure on S, which is not only σ-finite but also locally finite, see e.g. [BB09, Section 1.1]. When $S = \mathbb{R}^d$, this automatically implies that $\nu(K) < \infty$, and therefore $\mathbb{P}$-a.s. $N(K) < \infty$, for any bounded and measurable $K \subset \mathbb{R}^d$. In this case, almost surely there exists a (random!) collection of points $\Pi = (X_i)_{i \in I}$ (for some possibly also random index set I) such that for all $A \in \mathcal{S}$, $N(A) = |\{i \in I \colon X_i \in A\}|$, which we call a *Poisson point process* (or in some sources simply *Poisson process*). A Poisson point process on $\mathbb{R}^d$ is called *homogeneous* if the intensity measure μ has a constant density w.r.t. the d-dimensional Lebesgue measure, i.e., $\mu(\mathrm{d}x) = \lambda \, \mathrm{d}x$ for some $\lambda \geq 0$. A homogeneous Poisson process on $[0, \infty)$ with intensity $\lambda \geq 0$ is just the usual Poisson process with intensity measure equal to λ times the Lebesgue measure. For classical coursebooks on Poisson (point) processes, we refer the reader to [K93, LP17].

[10] For readers familiar with the content of population-genetic courses given by Jochen Blath, such a construction may be known from the topic of Λ-coalescents and their moment duals.

[11] Here, a Poisson(0) distributed random variable is defined to be almost surely equal to 0, and a Poisson(∞)-distributed random variable is defined to be almost surely equal to ∞.

For the process ν^K, a Poissonian construction was first given by Fournier and Méléard [FM04] as follows. Let $(\Omega, \mathcal{F}, \mathbb{P})$ be a sufficiently large probability space, and on this space consider the following five independent random objects:

(i) a $\mathcal{M}^K$-valued random variable ν_0^K (the initial distribution),

(ii) a Poisson point measure $N_1(\mathrm{d}s, \mathrm{d}i, \mathrm{d}v)$ on $[0,\infty) \times \mathbb{N} \times [0,1]$ with intensity measure $q_1(\mathrm{d}s, \mathrm{d}i, \mathrm{d}v) = \bar{b}\mathrm{d}s \sum_{k\geq 1} \delta_k(\mathrm{d}i)\mathrm{d}v$ (the Poisson point measure of birth without mutation),

(iii) a Poisson point measure $N_2(\mathrm{d}s, \mathrm{d}i, \mathrm{d}h, \mathrm{d}v)$ on $[0,\infty)\times\mathbb{N}\times\mathbb{R}^l\times[0,1]$ with intensity measure $q_2(\mathrm{d}s, \mathrm{d}i, \mathrm{d}h, \mathrm{d}v) = \bar{b}\mathrm{d}s \sum_{k\geq 1} \delta_k(\mathrm{d}i)\bar{m}(h)\mathrm{d}h\mathrm{d}v$ (the Poisson point measure of birth with mutation),

(iv) a Poisson point measure on $N_3(\mathrm{d}s, \mathrm{d}i, \mathrm{d}v)$ on $[0,\infty) \times \mathbb{N} \times [0,1]$ with intensity measure $q_3(\mathrm{d}s, \mathrm{d}i, \mathrm{d}v) = \bar{d}\mathrm{d}s \sum_{k\geq 1} \delta_k(\mathrm{d}i)\mathrm{d}v$ (the Poisson point process of natural death),

(v) a Poisson point measure $N_4(\mathrm{d}s, \mathrm{d}i, \mathrm{d}j, \mathrm{d}v)$ on $[0,\infty)\times\mathbb{N}\times\mathbb{N}\times[0,1]$ with intensity measure $q_4(\mathrm{d}s, \mathrm{d}i, \mathrm{d}j, \mathrm{d}v) = (\bar{\alpha}/K)\mathrm{d}s \sum_{k\geq 1} \delta_k(\mathrm{d}i) \sum_{m\geq 1} \delta_m(\mathrm{d}j)\mathrm{d}v$ (the Poisson point process of death by competition).

We will also need the following function, solving the purely notational problem of associating a number to each individual in the population: For any $K \geq 1$, let $H = (H^1, H^2, \ldots, H^k, \ldots)$ be the map from $\mathcal{M}^K$ into $(\mathbb{R}^l)^N$ defined by

$$H\Big(\frac{1}{K}\sum_{i=1}^n \delta_{x_i}\Big) = (x_{\sigma(1)}, \ldots, x_{\sigma(n)}, 0, 0, \ldots),$$

where $x_{\sigma(1)} \preceq \ldots \preceq x_{\sigma(n)}$ for the lexicographic order $\preceq$ on $\mathbb{R}^l$. For convenience, we omitted in our notation the dependence of H and H^i on K.

Then a process ν^K with generator L^K and initial state ν_0^K can be constructed as follows: For any $t \geq 0$,

$$\begin{aligned}
\nu_t^K = \nu_0^K &+ \int_0^t \int_{\mathbb{N}} \int_0^1 \mathbb{1}_{\{i\leq K\langle \nu_{s-}^K, \mathbf{1}\rangle\}} \frac{\delta_{H^i(\nu_{s-}^K)}}{K} \mathbb{1}_{\left\{v\leq \frac{[1-u_K\mu(H^i(\nu_{s-}^K))]b(H^i(\nu_{s-}^K)}{\bar{b}}\right\}} N_1(\mathrm{d}s, \mathrm{d}i, \mathrm{d}v) \\
&+ \int_0^t \int_{\mathbb{N}} \int_{\mathbb{R}^l} \int_0^1 \mathbb{1}_{\{i\leq K\langle \nu_{s-}^K, \mathbf{1}\rangle\}} \frac{\delta_{H^i(\nu_{s-}^K)+h}}{K} \mathbb{1}_{\left\{v\leq \frac{[u_K\mu(H^i(\nu_{s-}^K))]b(H^i(\nu_{s-}^K)}{b} \frac{m(H^i(\nu_{s-}^K),h)}{\bar{m}(h)}\right\}} N_2(\mathrm{d}s, \mathrm{d}i, \mathrm{d}h, \mathrm{d}v) \\
&- \int_0^t \int_{\mathbb{N}} \int_0^1 \mathbb{1}_{\{i\leq K\langle \nu_{s-}^K, \mathbf{1}\rangle\}} \frac{\delta_{H^i(\nu_{s-}^K)}}{K} \mathbb{1}_{\{v\leq \frac{\mu(H^i(\nu_{s-}^K))}{\bar{d}}\}} N_3(\mathrm{d}s, \mathrm{d}i, \mathrm{d}h) \\
&- \int_0^t \int_{\mathbb{N}} \int_{\mathbb{N}} \int_0^1 \mathbb{1}_{\{i\leq K(\nu_{s-}^K, \mathbf{1}\rangle\}} \mathbb{1}_{\{j\leq K(\nu_{s-}^K, \mathbf{1}\rangle\}} \frac{\delta_{H^i(\nu_{s-}^K)}}{K} \mathbb{1}_{\left\{v\leq \frac{\alpha(H^i(\nu_{s-}^K), H^j(\nu_{s-}^K)}{\bar{\alpha}}\right\}} N_4(\mathrm{d}s, \mathrm{d}i, \mathrm{d}j, \mathrm{d}v).
\end{aligned} \tag{1.15}$$

As written in [C06], although this formula is quite complicated, the principle is simple. For each type of event, the corresponding Poisson point process jumps faster than ν^K has to. We decide whether a jump of the process ν^K occurs by comparing v to a quantity related to the rates of the various events. The indicator functions involving i and j ensure that the i-th and j-th individuals are alive in the population at time t (because $K\langle \nu_t^K, \mathbf{1}\rangle$ is the number of individuals at that time). Here, for brevity, we do not provide the formal definition of the stochastic integral of a random integrand against a Poisson point measure, which can e.g. be found in [E19].

Under (A1), (A2), and the assumption that $\mathbb{E}(\langle \nu_0^K, 1\rangle) < \infty$, Fournier and Méléard [FM04] prove the existence and uniqueness of the solution to (1.15), and that this solution is a Markov process with infinitesimal generator (1.2).

Now, let us prove (1.14). The process A^K can be written as

$$A_t^K = \int_0^t \int_{\mathbb{N}} \int_{\mathbb{R}^l} \int_0^1 \mathbb{1}_{\{i\leq K(\nu_{s-}^K, \mathbf{1}\rangle\}} \mathbb{1}_{\left\{v\leq \frac{u_K\mu(H^i(\nu_{s-}^K))b(H^i(\nu_{s-}^K))}{\bar{b}} \frac{m(H^i(\nu_{s-}^K),h)}{\bar{m}(h)}\right\}} N_2(\mathrm{d}s, \mathrm{d}i, \mathrm{d}h, \mathrm{d}v).$$

In the case where $\nu_0^K = \langle \nu_0^K, \mathbf{1}\rangle \delta_x$, as long as $t < \tau_1$, we have $\nu_t^K = \langle \nu_t^K, \mathbf{1}\rangle \delta_x$. Therefore, for $t \le \tau_1 \wedge \inf\{s \ge 0 \colon \langle \nu_s^K, \mathbf{1}\rangle \notin [a_1, a_2]\}$,

$$\begin{aligned}\int_0^t \int_{\mathbb{N}} \int_{\mathbb{R}^l} \int_0^1 \mathbb{1}_{\{i \le K a_1\}} \mathbb{1}_{\{v \le \frac{u_K a(x) b(x)}{b} \frac{m(x,h)}{\bar{m}(h)}\}} N_2(\mathrm{d}s, \mathrm{d}i, \mathrm{d}h, \mathrm{d}v) \le A_t^K \\ \le \int_0^t \int_{\mathbb{N}} \int_{\mathbb{R}^l} \int_0^1 \mathbb{1}_{\{i \le K a_2\}} \mathbb{1}_{\{v \le \frac{u_K a(x) \lambda(x)}{b} \frac{m(x,h)}{\bar{m}(h)}\}} N_2(\mathrm{d}s, \mathrm{d}i, \mathrm{d}h, \mathrm{d}v).\end{aligned} \tag{1.16}$$

Since the intensity measure of N_2 is

$$q_2(\mathrm{d}s, \mathrm{d}i, \mathrm{d}h, \mathrm{d}v) = \bar{b} \mathrm{d}s \sum_{k \ge 1} \delta_k(\mathrm{d}i)) \bar{m}(h) \mathrm{d}h \mathrm{d}v,$$

the left-hand side and the right-hand side of (1.16) are Poisson processes with parameters $K u_K a_1 a(x) \lambda(x)$ and $K u_K a_2 a(x) \lambda(x)$, respectively. □

1.8 The problem of exit from a domain and a crash-course on large deviations

Points (a) and (b) of the following result on the birth-and-death process with law $\mathbb{P}^K(\lambda, \mu, \alpha, z)$ (cf. Definition 1.7 (b)) strengthen Proposition 1.8, while point (c) studies the problem of exit from a domain.

Theorem 1.17 ([C06]). *(a) Let $\alpha, T > 0$ and $\lambda, \mu \ge 0$, let C be a compact subset of $(0, \infty)$, and write $\mathbb{P}_z^K = \mathbb{P}^K(\lambda, \mu, \alpha, z)$ for a deterministic initial condition $z \in \mathbb{N}/K$. Let ϕ_z denote the solution to*

$$\dot{\phi} = (\lambda - \mu - \alpha\phi)\phi \tag{1.17}$$

with initial condition $\phi_z(0) = z$. Then

$$r := \inf_{z \in C} \inf_{t \in [0,T]} |\phi_z(t)| > 0 \quad \text{and} \quad R := \sup_{z \in C} \sup_{t \in [0,T]} |\phi_z(t)| < \infty.$$

Moreover, for any $\delta < r$,

$$\lim_{K \to \infty} \sup_{z \in C} \mathbb{P}_z^K \Big(\sup_{t \in [0,T]} |w_t - \phi_z(t)| \ge \delta \Big) = 0, \tag{1.18}$$

where $(w_t)_{t \ge 0}$ is the canonical process on $\mathbb{D}([0, \infty), \mathbb{R})$ (i.e., $w_t(\omega) = \omega(t)$ for $\omega \in \mathbb{D}([0, \infty), \mathbb{R})$, which has distribution $\mathbb{P}_z^K$ under the probability measure $\mathbb{P}^K(z)$).

(b) Let $T, \alpha_{ij} > 0$ and $\lambda_i, \mu_i \ge 0$ (for $i, j \in \{1, 2\}$), let C be a compact subset of $(0, \infty)^2$, and write $\mathbb{Q}_{z_1, z_2}^K = \mathbb{Q}^K(b_1, b_2, d_1, d_2, \alpha_{11}, \alpha_{12}, \alpha_{21}, \alpha_{22}, z_1, z_2)$ for deterministic initial conditions $z_1, z_2 \in \mathbb{N}/K$. Let $\phi_{z_1, z_2} = (\phi_{z_1, z_2}^1, \phi_{z_1, z_2}^2)$ denote the solution to

$$\begin{aligned}\dot{\varphi}^1 &= (\lambda_1 - \mu_1 - \alpha_{11}\phi^1 - \alpha_{12}\phi^2)\phi^1, \\ \dot{\varphi}^2 &= (\lambda_2 - \mu_2 - \alpha_{21}\phi^1, \alpha_{22}\phi^2)\phi^2\end{aligned}$$

with initial conditions $\phi_{z_1, z_2}^1(0) = z_1$ and $\phi_{z_1, z_2}^2(0) = z_2$. Then

$$r := \inf_{z \in C} \inf_{t \in [0,T]} \|\phi_{z_1, z_2}(t)\| > 0 \quad \text{and} \quad R := \sup_{z \in C} \sup_{t \in [0,T]} \|\phi_{z_1, z_2}(t)\| < \infty,$$

where $\|\cdot\|$ denotes an arbitrary norm on $\mathbb{R}^2$, which we fix for the rest of Section 1. Moreover, for any $\delta < r$,

$$\lim_{K \to \infty} \sup_{z \in C} \mathbb{Q}_{z_1, z_2}^K \big(\sup_{t \in [0,T]} \|\widehat{w}_t - \phi_{z_1, z_2}(t)\| \ge \delta \big) = 0,$$

where $\widehat{w}_t = (\widehat{w}_t^1, \widehat{w}_t^2)_{t \ge 0}$ is the canonical process on $\mathbb{D}((0, \infty), \mathbb{R}^2)$.

(c) Let $\lambda, \alpha > 0$ and $0 \le \mu < \lambda$. Observe that $(\lambda - \mu)/\alpha$ is the unique stable equilibrium of (1.17). Fix $\eta_1 \in (0, (\lambda-\mu)/\alpha)$ and $\eta_2 > 0$, and define on $\mathbb{D}((0,\infty), \mathbb{R})$

$$T^K = \inf\Big\{t \ge 0 \colon w_t \notin \Big[\frac{\lambda-\mu}{\alpha} - \eta_1, \frac{\lambda-\mu}{\alpha} + \eta_2\Big]\Big\}. \tag{1.19}$$

Then, there exists $V > 0$ such that, for any compact subset C of $(\frac{\lambda-\mu}{\alpha} - \eta_1, \frac{\lambda-\mu}{\alpha} + \eta_2)$,

$$\lim_{K\to\infty} \sup_{z\in C} \mathbb{P}_z^K\big(T^K < \mathrm{e}^{KV}\Big) = 0.$$

Proof. First, we prove (a). Since $\dot\phi < 0$ as soon as $\phi > (b-d)/a$, any solution to (1.3) with positive initial condition is bounded, which implies $R < \infty$. Moreover, a solution to (1.3) can be written as

$$\phi(t) = \phi(0) \exp\Big(\int_0^t (\lambda - \mu - \alpha\phi(s))\mathrm{d}s\Big) \ge \phi(0)\exp((\lambda - \mu - \alpha R)t),$$

from which it follows that $r > 0$.

Equation (1.18) is a consequence of large-deviation estimates for the sequence of laws $(\mathbb{P}_z^K)_{K\ge1}$. Choose functions $p, q \colon \mathbb{R} \to (0,\infty)$ that are Lipschitz continuous, bounded and uniformly bounded away from 0 and we consider the $\mathbb{Z}/K$-valued Markov jump processes with transition rates

$$\begin{aligned} i/K &\to (i+1)/K \text{at rate } Kp(i/K),\\ i/K &\to (i-1)/K \text{at rate } Kq(i/K), \end{aligned}$$

Note that the process with law $(\mathbb{P}_z^K)_{K\ge1}$ does not satisfy these assumptions, but its following variant with truncated rates does:

$$p(z) = \lambda\chi(z) \quad \text{and } q(z) = \mu\chi(z) + \alpha\chi(z)^2,$$

where $\chi(z) = z$ if $z \in [r-\delta, R+\delta]$, $\chi(z) = r - \delta$ if $z < r - \delta$; $\chi(z) = R + \delta$ if $z > R + \delta$, then p and q satisfy the assumptions above, and if $\mathbb{R}_z^K$ denotes the law of this process with initial condition z, then we have $\mathbb{R}_z^K = \mathbb{P}_z^K$ on the time interval $[0, \tau]$, where $\tau = \inf\{t \ge 0 \colon w_t \notin [r-\delta, R+\delta]\}$.

For the sequence of laws $(\mathbb{R}_z^K)_{K\in\mathbb{N}}$, thanks to [DE97, Chap. 10, Theorem 10.2.6], the large deviation principle holds with good rate function I_T given as follows: For $\phi \colon [0,T] \to \mathbb{R}^2$ càdlàg,

$$I_T(\phi) = \begin{cases} \int_0^T L(\phi(t), \dot\phi(t))\ \mathrm{d}t & \text{if } \phi \text{ is absolutely continuous on } [0,T],\\ \infty & \text{otherwise,} \end{cases} \tag{1.20}$$

for a certain function $L \colon \mathbb{R}^2 \to [0,\infty)$ such that $L(y,z) = 0$ if and only if $z = p(y) - q(y)$. See Appendix B for the precise form of L. Therefore, $I_T(\phi) = 0$ if and only if ϕ is absolutely continuous and

$$\dot\phi = p(\phi) - q(\phi). \tag{1.21}$$

What does the above assertion mean? A *rate function* I is a mapping from a given topological space $\mathcal{M}$ (here, $\mathcal{M} = \mathbb{D}([0,T],\mathbb{R})$ equipped with the Skorokhod J1 topology, cf. footnote 4) to $[0,\infty]$ that is *lower semicontinuous*, i.e., the (sub-)level set $\{z \in M \colon I(z) \le \alpha\}$ is closed in the topology of $\mathcal{M}$ for all $z \in \mathbb{R}$. I is called a *good rate function* if the aforementioned level sets are all compact in the topology of $\mathcal{M}$.[12] A good rate function always attains its infimum over closed sets. Let us note that if $\mathcal{M}$ is a metric space (more precisely, its topology is induced by a metric), then lower semicontinuity is equivalent to the assertion that

$$\liminf_{z_n\to z} I(z_n) \ge I(z), \quad \forall z \in \mathcal{M}.$$

[12] Hence, if the topological space $\mathcal{M}$ is Hausdorff, then every good rate function is a rate function (is that clear?).

The large deviation principle states, very roughly speaking, that for certain measurable sets $A \subseteq \mathcal{M}$, we have

$$\mathbb{R}_z^K(A) \approx \exp\Big(- K(1 \pm o(1)) \inf_{\psi \in A} I_T(\psi)\Big),$$

or in other words,

$$\frac{1}{K} \log \mathbb{R}_z^K(A) \approx - \inf_{\psi \in A} I_T(\psi).$$

That is, if the set A includes some ψ such that $I_T(\psi) = 0$, then the $\mathbb{R}_z^K$-probability of A does not decay exponentially, whereas if A contains no such ψ, then A is a rare event whose probability decays exponentially in K with rate approximately $\inf_{\psi \in A} I_T(\psi)$.

Let us make this more precise now: By definition, the fact that the *large deviation principle* with good rate function I_T holds for the sequence of probability measures $(R_z^K)_{K \in \mathbb{N}}$ means that for all open sets $G \subseteq \mathcal{M} = \mathbb{D}([0,T], \mathbb{R})$, the lower bound

$$\liminf_{K \to \infty} \frac{1}{K} \log \mathbb{R}_z^K(G) \geq - \inf_{\psi \in G} I_T(\psi)$$

is satisfied, whereas for all closed sets $F \subseteq \mathcal{M}$, we have the upper bound

$$\limsup_{K \to \infty} \frac{1}{K} \log \mathbb{R}_z^K(F) \leq - \inf_{\psi \in F} I_T(\psi).$$

In our particular case, it also follows from [DE97] that this large deviation principle is uniform with respect to the initial condition in the sense that for any compact subset C of $\mathbb{R}$ and F, G as above, we have

$$\liminf_{K \to \infty} \frac{1}{K} \log \inf_{z \in C} \mathbb{R}_z^K(G) \geq - \sup_{z \in C} \inf_{\psi \in G \colon \psi(0) = z} I_T(\psi) \tag{1.22}$$

and

$$\limsup_{K \to \infty} \frac{1}{K} \log \sup_{z \in C} \mathbb{R}_z^K(F) \leq - \inf_{\psi \in F \colon \psi(0) \in C} I_T(\psi). \tag{1.23}$$

Therefore, by (1.23),

$$\limsup_{K \to \infty} \frac{1}{K} \log \mathbb{R}_z^K\Big(\sup_{t \in [0,T]} |w_t - \phi_z(t)| \geq \delta\Big) \leq - \inf_{\psi \in F^\delta} I_T(\psi),$$

where

$$F^\delta = \big\{ \psi \in \mathbb{D}([0,T], \mathbb{R}) \colon \psi(0) \in C \text{ and } \exists t \in [0,T] \colon |\psi(t) - \phi_{\psi(0)}(t)| \geq \delta \big\}.$$

By the continuity of the flow $z \mapsto \phi_z(t)$ of (1.21) (which is a consequence of the fact that $z \mapsto p(z) - q(z)$ is Lipschitz and Gronwall's lemma), the set F^δ is closed. Since I_T is a good rate function, it attains its infimum on F^δ at some function that cannot be a solution to (1.21), and thus is non-zero. This finishes the proof of (1.18). The proof of (b) is very similar.

Let us now prove (c). Define the function χ on $\mathbb{R}$ by

$$\chi(z) = \begin{cases} z, & \text{if } z \in [(\lambda - \mu)/\alpha - \eta_1, (\lambda - \mu)/\alpha + \eta_2], \\ (\lambda - \mu)/\alpha - \eta_1, & \text{if } z < (\lambda - \mu)/\alpha - \eta_1, \\ (\lambda - \mu)/\alpha + \eta_2, & \text{if } z > (\lambda - \mu)/\alpha + \eta_2. \end{cases}$$

As in the proof of (a), using the functions $p(z) = \lambda \chi(z)$ and $q(z) = \mu \chi(z) + \alpha z^2$ a family of laws $(\mathbb{R}_z^K)$ such that $\mathbb{R}_z^K = \mathbb{P}_z^K$ on the time interval $[0, T^K]$ (cf. (1.19)), and such that the uniform large-deviation estimates (1.22) and (1.23) hold for the good rate function I_T defined analogously to (1.20).

Observe that all solutions to (1.21) are monotonous and converge to $(\lambda-\mu)/\alpha$ when $t\to\infty$. Therefore, the following classical estimates for the time of exit from an attracting domain [FW84, Chapter 5, Section 4] apply: There exists $\widehat{V}\geq 0$ such that for any $\delta>0$,

$$\lim_{K\to\infty}\inf_{z\in C}\mathbb{R}_z^K\big(\mathrm{e}^{K(\widehat{V}-\delta)}<T^K<\mathrm{e}^{K(\widehat{V}+\delta)}\big)=1, \tag{1.24}$$

and hence in order to finish the proof of (c), it suffices to show that $\widehat{V}>0$.

Now, the constant $\widehat{V}$ is obtained as follows (see [FW84, pages 108–109]): for any $y,z\in\mathbb{R}$, define

$$V(y,z)=\inf_{t>0\colon\,\varphi(0)=y,\varphi(t)=z} I_t(\varphi).$$

Then

$$\widehat{V}:=V\big(\frac{\lambda-\mu}{\alpha},\frac{\lambda-\mu}{\alpha}-\eta_1\big)\wedge V\big(\frac{\lambda-\mu}{\alpha},\frac{\lambda-\mu}{\alpha}+\eta_2\big).$$

(In physical terms, e.g. $V\big(\frac{\lambda-\mu}{\alpha},\frac{\lambda-\mu}{\alpha}-\eta_1\big)$ can be interpreted as the *work* that needs to be invested in order to move a point of unit mass from the attractive equilibrium $\frac{\lambda-\mu}{\alpha}$ of (1.21) to $\frac{\lambda-\mu}{\alpha}-\eta_1$ despite the "potential" attracting the mass point towards $\frac{\lambda-\mu}{\alpha}$.)

Now, [FW84, Theorem 5.4.3] states that, for any $y,z\in\mathbb{R}$, the infimum defining $V(y,z)$ is attained at some function ϕ linking y to z. This means that either there exists an absolutely continuous function ϕ defined on $[0,T]$ for some $T>0$ such that $\phi(0)=y$, $\phi(T)=z$, and $V(y,z)=I_T(\phi)=\int_0^T L(\phi(t),\dot\phi(t))\mathrm{d}t$, or there exists an absolutely continuous function ϕ defined on $(-\infty,T)$ for some $T\in\mathbb{R}$ such that $\lim_{t\to-\infty}\phi(t)=y$, $\phi(T)=z$, and $V(y,z)=\int_{-\infty}^T L(\phi(t),\dot\phi(t))\mathrm{d}t$. Since any solution to (1.21) is decreasing as long as it stays in $[(\lambda-\mu)/\alpha,\infty)$, a function ϕ defined on $[0,T]$ or $(-\infty,T)$ linking $(\lambda-\mu)/\alpha$ to $(\lambda-\mu)/\alpha+\eta_2$ cannot be a solution to (1.21), and thus V $\big(\frac{\lambda-\mu}{\alpha},\frac{\lambda-\mu}{\alpha}+\eta_2\big)>0$. Similarly, $V\big(\frac{\lambda-\mu}{\alpha},\frac{\lambda-\mu}{\alpha}-\eta_1\big)>0$, hence $\widehat{V}>0$, which concludes the proof of Theorem 1.17.

□

Remark 1.18. We see in (1.24) that in case the resident population is type x, one needs the first mutation to appear in at most $\mathrm{e}^{K\widehat{V}(1+o(1))}$ time with high probability in order that the resident population is still close to its equilibrium $\bar n_x$ at the time when the mutant emerges. The value of $\widetilde{V}$ of course also depends on the value of $\lambda-\mu$ and may tend to zero as $\lambda-\mu\downarrow 0$, and hence in the left inequality of (1.9) for simplicity we assume that the first mutant appears in subexponential time. But for example, if we assume that $\lambda(x)-\mu(x)$ only changes by a uniformly bounded number over all x if an individual of trait x suffers a mutation, then we can find a suitable $\widetilde{V}>0$ such that assuming $\exp\Big(-\widetilde{V}K\Big)\ll u_K$ instead of $\exp(-VK)\ll u_K$ for all $V>0$ suffices in order for Theorem 1.1 to hold (with all other assumptions unchanged).

1.9 Some results on branching processes

Although we do not provide all the details of the proof of Theorem 1.1, the following collection of assertions on branching processes plays a crucial role in this proof, and it is also applicable for many other models of stochastic population biology or even other fields of probability theory.

Let $\mathbb{Q}_n$ denote the law of a binary branching process with initial state $n\in\mathbb{N}$, with individual birth rate λ and individual death rate μ. Regarding this process, we have the following theorem, where we recall the canonical process $(w_t)_{t\geq 0}$ from part (a) of Theorem 1.17.

Theorem 1.19 ([C06])**.** *Let $\lambda,\mu>0$. Define, for any $\varrho\in\mathbb{R}$, on $\mathbb{D}([0,\infty),\mathbb{R})$, the stopping time*

$$T_\varrho=\inf\{t\geq 0\colon w_t=\varrho\}.$$

(In particular, T_0 is the extinction time of the process.)
Finally, let $(t_K)_{K\geq 1}$ be a sequence of positive numbers such that $t_K\gg\log K$.

(a) If $\lambda < \mu$ (subcritical case), then for any $\varepsilon > 0$,

$$\lim_{K\to\infty} \mathbb{Q}_1\big(T_0 \le t_K \wedge T_{\lfloor \varepsilon K\rfloor}\big) = 1 \tag{1.25}$$

and

$$\lim_{K\to\infty} \mathbb{Q}_{\lfloor \varepsilon K\rfloor}(T_0 \le t_K) = 1. \tag{1.26}$$

Moreover, for any $K, k, n \ge 1$,

$$\mathbb{Q}_n(T_{kn} \le T_0) \le \frac{1}{k}. \tag{1.27}$$

(b) If $\lambda > \mu$ (supercritical case), then for any $\varepsilon > 0$,

$$\lim_{K\to\infty} \mathbb{Q}_1\big(T_0 \le t_K \wedge T_{\lceil \varepsilon K\rceil}\big) = \frac{\mu}{\lambda} \tag{1.28}$$

and

$$\lim_{K\to\infty} \mathbb{Q}_1\big(T_{\lceil \varepsilon K\rceil} \le t_K\big) = 1 - \frac{\mu}{\lambda}. \tag{1.29}$$

Proof. Let $q = \mathbb{Q}_1(T_0 < \infty)$ denote the extinction probability of the branching process started with one individual. Then it follows from classical first-step analysis (for the embedded discrete-time Galton–Watson process where each individual has 2 offspring with probability $\frac{\lambda}{\lambda+\mu}$ and 0 offspring with probability $\frac{\mu}{\lambda+\mu}$) that q is the smallest solution to the quadratic equation

$$q = \frac{\lambda}{\lambda+\mu} q^2 + \frac{\mu}{\lambda+\mu} \cdot 1.$$

The roots of this equation are given by 1 and $\frac{\mu}{\lambda}$, hence the smallest solution is 1 in the subcritical case and $\frac{\mu}{\lambda}$ in the supercritical case.[13]

Assertion (1.29) follows easily from the extinction time for binary branching processes when $\lambda \ne \mu$ (cf. [AN72, p. 109]): For any $t \ge 0$ and $n \in \mathbb{N}$,

$$\mathbb{Q}_n(T_0 \le t) = \Big(\frac{\mu(1 - \mathrm{e}^{-(\lambda-\mu)t})}{\lambda - \mu\mathrm{e}^{-(\lambda-\mu)t}}\Big)^n.$$

Since $t_K \to \infty$, $\mathbb{Q}_1(T_0 \le t_K \wedge T_{\lceil \varepsilon K\rceil}) \to \mathbb{Q}_1(T_0 < \infty) = 1$, which yields (1.25) and (1.28).

The inequality (1.27) follows from the fact that if $(Z_t)_{t\ge 0}$ is a branching process with law $\mathbb{Q}_n$, then $(Z_t \exp(-(\lambda-\mu)t)_{t\ge 0}$ is a martingale (see [AN72, p. 111]). Applying Doob's optimal stopping theorem applied to the stopping time $T_0 \wedge T_{kn}$, writing $\mathbb{E}_n$ for the expectation w.r.t. $\mathbb{Q}_n$, we obtain

$$\mathbb{E}_n\big(kn\mathrm{e}^{-(\lambda-\mu)T_{kn}} \mathbb{1}_{\{T_{kn} < T_0\}}\big) = n.$$

(This is the version of the optimal stopping theorem where the stopping time is a.s. finite and the stopped martingale is bounded; in this case between 0 and kn.) Therefore, when $\lambda \le \mu$, $kn\mathbb{Q}_n(T_{kn} \le T_0) \le n$, and thus we conclude (1.27).[14]

The limiting assertion (1.29) follows from the fact that if $(Z_t)_{t\ge 0}$ is a branching process with law $\mathbb{Q}_1$, then the martingale $(Z_t\mathrm{e}^{-(\lambda-\mu)t})_{t\ge 0}$ converges a.s. when $t \to \infty$ to a random variable W, where $W = 0$ on the event $\{T_0 < \infty\}$ and $W > 0$ on the event $\{T_0 = \infty\}$ (see [AN72, p. 112]). Hence, when $\lambda > \mu$, on the event $\{T_0 < \infty\}$ we have

$$\frac{\log Z_t}{t} \to \lambda - \mu > 0 \tag{1.30}$$

a.s. Therefore, since $t_K \gg \log K$, for any $\varepsilon > 0$, $\mathbb{Q}_1(T_0 = \infty, T_{\lceil \varepsilon K\rceil} \ge t_K) \to 0$ when $K \to \infty$. Thus, using the above observation that $\mathbb{Q}_1(T_0 = \infty) = 1 - \frac{\mu}{\lambda}$, we conclude (1.29). □

[13] Note that this also applies in the critical case $\lambda = \mu$, where 1 and μ/λ coincide, although we do not treat this case here.

[14] Of course, this martingale property also holds in the critical case $\lambda = \mu$; in this case, the process $(Z_t)_{t\ge 0}$ is itself a martingale. Therefore (1.27) is also true in the critical case.

Remark 1.20. We did not provide detailed proofs for the martingale property, the positivity of the almost sure limit W of the martingale on $\{T_0 = \infty\}$, and the fact that the limit is zero on $\{T_0 < \infty\}$; the interested reader can consult [AN72]. Nevertheless, we would like to emphasize that (1.27) also has a more elementary proof based on the fact that a binary branching process is nothing but a time-changed random walk on $\mathbb{Z}$, and we only need to use that this random walk is a martingale in the symmetric case $\lambda = \mu$. Indeed, the nearest-neighbour random walk on $\mathbb{Z}$ in continuous time that jumps one step up at rate λ and one step down at rate μ has generator L defined via

$$Lf(n) = \lambda(f(n+1) - f(n)) + \mu(f(n-1) - f(n))$$

for all bounded measurable functions $f\colon \mathbb{Z} \to \mathbb{R}$. Now, the binary branching process with individual birth rate λ and individual death rate μ has generator $\mathcal{L}$ acting on all bounded measurable functions $f\colon \mathbb{N}_0 \to \mathbb{R}$ via

$$\mathcal{L}f(n) = n(\lambda(f(n+1) - f(n)) + \mu(f(n-1) - f(n))).$$

This is indeed a time-changed version of the random walk; the time change becomes zero (time stops, the process gets absorbed) at the state 0, and thus the state can never get negative. Now, let us consider the critical case $\lambda = \mu$. Then it is clear that the random walk $(S_t)_{t\geq 0}$ is itself a martingale and thus by Doob's optimal stopping theorem, defining the stopping time $T = \inf\{t \geq 0\colon S_t \in \{0, kn\}\}$, we have

$$n = \mathbb{E}[S_T | S_0 = n] = kn\mathbb{P}(S_T = kn),$$

which yields (1.27) for $\lambda = \mu$. Now, for $\lambda < \mu$, the probability under consideration can only get smaller.

Remark 1.21. Since (1.30) holds in the supercritical phase, we actually have that for any $\varepsilon > 0$,

$$\frac{T_{\lfloor \varepsilon K \rfloor}}{\log K} \overset{K\to\infty}{\longrightarrow} \frac{1}{\lambda - \mu}$$

a.s. on $\{T_0 = \infty\}$. Therefore, part (a) of Theorem 1.19 also applies when $t_K \not\gg \log K$ but there exists $\delta > 0$ such that $t_K \geq (\frac{1}{\lambda-\mu} + \delta)$ for all sufficiently large K, while it does not hold if there exists $\delta > 0$ such that $t_K \leq (\frac{1}{\lambda-\mu} - \delta)$ for all sufficiently large K. The factor $\frac{1}{\lambda-\mu}$ of $\log K$ is the reciprocal of the mean growth rate of the branching process, and this will be similar in the case of multitype branching processes (see Section 2.5). Similarly, part (b) of Theorem 1.19 also applies for the aforementioned choice of t_K for which part (a) applies. In the subcritical case, we have exponential decay instead of exponential growth. When starting with $\lceil \varepsilon K \rceil$ individuals, reaching extinction takes approximately $\frac{1}{\mu-\lambda} \log K$ time.

Below (1.5) we already interpreted $f(y, x)$ as the net growth rate of a mutant population of trait y in a resident population of trait x when the mutant population is small compared to K. These mutants have birth rate $\lambda(y)$ and death rate approximately $\mu(y) + \alpha(y, x)\bar{n}_x$ per individual. The branching process approximating the mutant population is the binary branching process precisely with these parameters, which is supercritical if and only if $f(y, x) > 0$. This branching process corresponds to Phase I of the invasion of trait y against trait x. In case the branching process is supercritical, we have a nonvanishing probability of a successful invasion of trait y, which also implies fixation (i.e. extinction of the former resident trait) due to our non-coexistence assumption (B). Now, in Phase III, when the trait y population size rescaled by K is close to $\bar{n}_y$ and the population of the former resident trait x is small compared to K, the resident population has birth rate $\mu(x)$ and death rate close to $\mu(y) + \alpha(x, y)\bar{n}_y$.[15] Hence, it can be approximated by a binary branching process with precisely these parameters, which is subcritical if and only if $f(x, y) < 0$. Under (B), $f(x, y) < 0$ is satisfied whenever $f(y, x) > 0$.

Remark 1.22 (The case of symmetric competition). Assume that $\alpha(x, y)$ equals the same positive number α for all $x, y \in \mathcal{X}$. Then, we have

$$\bar{n}_x = \frac{\lambda(x) - \mu(x)}{\alpha}.$$

[15] Of course, with a suitable initial condition, this branching process also approximates a type x mutant population born when type y is resident.

Hence, the larger the "absolute fitness" (i.e., net growth rate) $\lambda(x)-\mu(x)$ of a trait x, the higher its equilibrium population size. The invasion fitness of a mutant of trait y reads (cf. (1.5))

$$f(y,x) = \lambda(y) - \mu(y) - \alpha\bar{n}(x) = \lambda(y) - \mu(y) - (\lambda(x) - \mu(x)),$$

i.e., mutants can invade with positive probability (the corresponding branching process is supercritical) if and only if their net growth rate is positive. In other words, the invasion fitness is the difference between the absolute fitness of mutant and resident. The later a mutant with a given absolute fitness comes, the higher typically the resident fitness is, and therefore, the invasion of the mutant (if it is still possible at all) will typically take a longer and longer time. This case is also *antisymmetric* w.r.t. the invasion fitnesses, which by definition means that $f(y,x) = -f(x,y)$, and *transitive* in the sense of Remark 1.11. Thanks to the observations of Remark 1.21, antisymmetry implies that Phase I and Phase III of any invasion take the same amount of time on the $\log K$ time scale. Without antisymmetry, the same is not true in general (see e.g. the example presented in Section 2).

Remark 1.23. Apart from the nice connection of (1.27) to random walks, we should also explain at least intuitively the role of this assertion in the proof of Theorem 1.1, even if we do not spell out all details of this proof. Assertion (1.27) implies that for $k \geq 0$ fixed, subcritical branching processes started from an arbitrarily large initial condition n will go extinct before reaching size kn with probability at least $1 - 1/k$ as $n \to \infty$. In the setting of the invasion of trait y against trait x, this branching process consists of the leftover individuals of trait x after the rescaled population size of trait y has already reached a vicinity of its equilibrium population size $\bar{n}_y$. Now, one first has to show that the trait y mutants are sufficiently close to a process to which the large-deviations machinery of Section 1.8 is applicable, as long as their population is not too large, and then one has to prove that the trait x residents will go extinct with high probability before their population could become too large or the population of the mutants could leave a small neighbourhood of equilibrium. For the first item, an estimate like (1.27) is very useful: e.g. one can start with εK residents and guarantee that with high probability, the resident population never goes beyond $\sqrt{\varepsilon}K$ before dying out, and one can allow an error term of order $\sqrt{\varepsilon}K$ in the transition rates of the mutant and still apply the Freidlin–Wentzell type large-deviation results (see [CCLLS21] for a similar argument).

Remark 1.24. It is also worth observing that for $x, y \in \mathcal{X}$, the Jacobi matrix (cf. (1.13))

$$A(\bar{n}_x, 0) = \begin{pmatrix} \mu(x) - \lambda(x) & -\alpha(x,y)\bar{n}_x \\ 0 & \lambda(y) - \mu(y) - \frac{\alpha(y,x)(\lambda(x)-\mu(x))}{\alpha(x,x)} \end{pmatrix}$$

has a positive eigenvalue if and only if the first inequality of (1.7) holds, while it has two negative eigenvalues if (1.6) holds, and $A(0, \bar{n}_y)$ has two negative eigenvalues if and only if the second inequality of (1.7) holds, whereas it has a positive eigenvalue if and only if (1.6) holds with the roles of x and y swapped. In other words, $A(\bar{n}_x, 0)$ is unstable (resp. asymptotically stable) if and only if the branching process approximating the population of a trait y mutant when trait x is resident is supercritical (resp. subcritical), and $A(0, \bar{n}_y)$ is asymptotically stable (resp. unstable) if and only if the branching process approximating the population of a trait x mutant when trait y is resident is subcritical (resp. supercritical). Certainly, $A(\bar{n}_x, 0)$ and $A(0, \bar{n}_y)$ are very simple matrices whose eigenvalues are their diagonal entries, but actually we will also see such correspondences between the critical behaviour of branching processes and the stability of equilibria of ODE systems in the sequel, see Remark 2.4 below.

2 Example 1: an invasion model with competition-induced dormancy

2.1 Motivation

Champagnat's work [C06] laid the fundament for analysing the invasion of mutants in the scaling regime of rare mutations in adaptive dynamics without a coexistence between multiple traits. In this section as well as

the following one, we will incorporate further biologically relevant and interesting phenomena into the model and explain how Champagnat's methods can be extended to these cases. In particular, the approximating branching processes will often be *multitype*, and in some cases we will be able to treat some questions of *global* stability of equilibria of the underlying dynamical systems. Since in the rare mutation regime, mutations are separated from each other in time as $K \to \infty$, it is possible to study the fate of one single mutation, starting with a resident population near its equilibrium size and one single mutant (or a few mutants) at time 0, excluding further mutations.[16] The particular examples that we will focus on in these two sections will not include multiple mutations but rather just one single invasion step. The same approach is of course not applicable if we expect that the population that we want to model is substantially affected by clonal interference.

In this section, we focus on *dormancy*, which is an evolutionary trait that has emerged independently at various positions across the tree of life. It describes the ability of a microorganism to switch to a reversible and metabolically inactive state that can withstand unfavorable conditions. However, maintaining such a trait requires additional resources that could otherwise be used to increase e.g. reproductive rates. Examples include among others winter sleep of mammals, sporulation of bacteria, or dormancy of cancer cells that may eventually lead to therapy resistance and metastases. The reactivation of dormant individuals/cells is referred to as *resuscitation*. For an overview on various forms of dormancy, see the survey article by Lennon, den Hollander, Wilke Berenguer, and Blath [LdHWB21].

In what follows, we summarize the results and proofs of the paper [BT20], sometimes making excursions to classical works that are necessary for the proofs of these results. To introduce the model of the paper informally, imagine that we have a resident population consisting of identically fit one-cell individuals performing asexual and haploid reproduction (i.e., binary fission), death by age and by competition among individuals, living close to its equilibrium population size, just as one typical trait in Champagnat's model, and now a mutant (or migrant) emerges. This mutant is able to switch into a dormant state under competitive pressure (we will see in a moment what this mathematically means). Dormant mutants cannot reproduce, but they are not affected by competition, eventually they will either die naturally or resuscitate. This should ease competition for dormant individuals. The main questions are then the following:

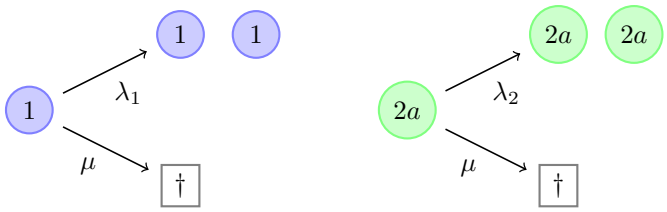
- Are there choices of the parameters when the mutant can invade the resident population (with an asymptotically positive probability as $K \to \infty$)?
- If yes, what is the asymptotic probability of a successful invasion and how much time does it typically take the mutant population to become macroscopic?
- What happens after a successful invasion? Will the mutant fix, making the former residents die out, or will it coexist with the residents?

2.2 The base model for competition-induced dormancy

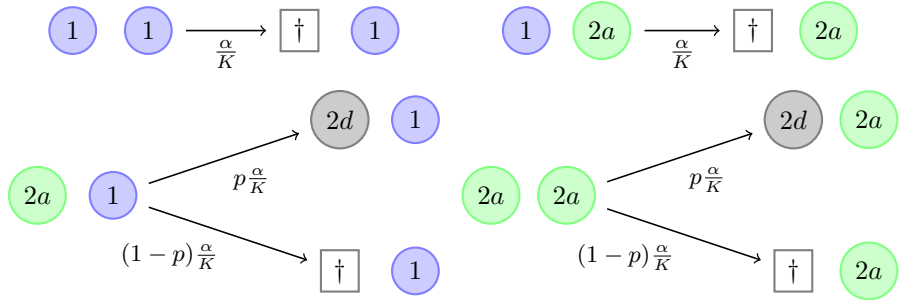
Let us now introduce the stochastic individual-based model of [BT20]. We have two traits (types), the resident one (1) and the mutant one (2). Mutant individuals can have an active (2a) and a dormant (2d) state. As an interpretation, we will sometimes say that the dormant individuals are in the *seed bank*. Informally speaking, the model is defined as follows.

- A resident individual gives birth to another such individual at rate $\lambda_1 > 0$.
- An active mutant individual gives birth to another such individual at rate $\lambda_2 \in (0, \lambda_1)$.
- Any active individual has a natural death rate $\mu \in (0, \lambda_1)$.

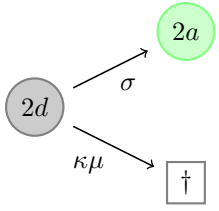
[16] In this sense, the mutant could as well be interpreted as a migrant arriving from somewhere else in the case of models involving an underlying geographic space.



- $K > 0$ is the carrying capacity of the population.
- The competitive pressure felt by an active individual from another active individual is $\alpha/K > 0$, where $\alpha > 0$. For any ordered pair (x_i, x_j) of active individuals, at rate $\alpha/K > 0$ a competitive event affecting x_i happens. We fix $p \in (0, 1)$. At a competitive event, in case x_i is a resident individual, it dies. If x_i is a mutant individual, it dies with probability $1 - p$ and becomes a dormant (mutant) individual with probability p.
  In other words, in a population with $n_1 \in \mathbb{N}_0$ (active) resident individuals and $n_{2a} \in \mathbb{N}_0$ active mutant individuals, writing $n_a = n_1 + n_{2a}$ for the total number of active individuals, a resident individual dies by competition at rate $\alpha n_a/K$, an active mutant dies by competition at rate $(1 - p)\alpha n_a/K$ and switches to dormant mutant at rate $p\alpha n_a/K$.



- For some $\kappa \geq 0$, a dormant (mutant) individual dies at rate $\kappa\mu$.[17]
- A dormant (mutant) individual becomes an active (mutant) individual at rate $\sigma > 0$.



Further necessary conditions on the parameters will be specified later in the sequel. Note that the behaviour of trait 1 in absence of trait 2 fits into the framework of [C06] with a monomorphic initial state and with mutations excluded. Actually, in Champagnat's setting we were only describing the model in terms of empirical measures because those are the right objects to study if one wants to prove some kind of convergence to a TSS. In our model without further mutations, it will suffice to focus on frequencies of subpopulations (rescaled by K if needed). To define the model more precisely, we consider, for $t \geq 0$, a finite

[17] From a biological point of view, it is reasonable to assume that dormancy reduces natural death rate too, i.e., $\kappa \leq 1$, but $\kappa > 1$ does not make a big difference mathematically and neither does $\kappa = 0$.

number $N_t \in \mathbb{N}_0$ of individuals $\{x_i \colon i \in [N_t]\}$, where for all $i \in [N_t]$ we have $x_i \in \{1, 2a, 2d\}$. We define the triple of frequency processes

$$(\mathbf{N}_t)_{t\geq 0}) = ((N_{1,t}, N_{2a,t}, N_{2d,t}))_{t\geq 0},$$

where for $x \in \{1, 2a, 2d\}$,

$$N_{x,t} = \#\{x_i \colon i \in [N_t], x_i = x\}.$$

We will often consider the triple of rescaled frequency processes

$$(\mathbf{N}^K_t)_{t\geq 0} = ((N^K_{1,t}, N^K_{2a,t}, N^K_{2d,t}))_{t\geq 0},$$

where for $x \in \{1, 2a, 2d\}$,

$$N^K_{x,t} = \frac{1}{K} N_{x,t}$$

is the number of individuals of type x rescaled by K. We also write

$$N_{2,t} = N_{2a,t} + N_{2d,t}, \qquad N^K_{2,t} = N^K_{2a,t} + N^K_{2d,t}$$

for the non-rescaled resp. rescaled total population size of mutant individuals and

$$N_t = N_{1,t} + N_{2,t}, \qquad N^K_t = N^K_{1,t} + N^K_{2,t} = \frac{N_t}{K}$$

for the total population size resp. $1/K$ times the same. Hence, $(\mathbf{N}^K_t)_{t\geq 0}$ is a $\left(\frac{1}{K}\mathbb{N}_0\right)^3$-valued Markov process with transitions

$$(n_1, n_{2a}, n_{2d}) \to \begin{cases} (n_1 + \frac{1}{K}, n_{2a}, n_{2d}) \text{ at rate } Kn_1\lambda_1, \\ (n_1, n_{2a} + \frac{1}{K}, n_{2d}) \text{ at rate } Kn_{2a}\lambda_2, \\ (n_1 - \frac{1}{K}, n_{2a}, n_{2d}) \text{ at rate } Kn_1(\mu + \alpha(n_1 + n_{2a})), \\ (n_1, n_{2a} - \frac{1}{K}, n_{2d}) \text{ at rate } Kn_{2a}(\mu + (1-p)\alpha(n_1 + n_{2a})), \\ (n_1, n_{2a} - \frac{1}{K}, n_{2d} + \frac{1}{K}) \text{ at rate } Kn_{2a}p\alpha(n_1 + n_{2a}), \\ (n_1, n_{2a}, n_{2d} - \frac{1}{K}) \text{ at rate } Kn_{2d}\kappa\mu, \\ (n_1, n_{2a} + \frac{1}{K}, n_{2d} - \frac{1}{K}) \text{ at rate } Kn_{2d}\sigma. \end{cases}$$

The rates of $(\mathbf{N}_t)_{t\geq 0}$ can be described in an analogous way.

Exercise 5. *Write down the infinitesimal generator of* $(\mathbf{N}^K_t)_{t\geq 0}$ *(acting on all bounded measurable functions* $f \colon \left(\frac{1}{K}\mathbb{N}_0\right)^3 \to \mathbb{R}$*).*

The condition $\lambda_1 > \lambda_2 > \mu$ above implies that both residents and mutants are fit, i.e., they exhibit no rapid extinction, when being on their own, but mutants have a lower reproductive rate than residents. We have seen that $\lambda_1 > \mu$ is equivalent to the long-term survival of residents if the initial condition is of order K, and because of the dormancy it is not *a priori* clear that $\lambda_2 > \mu$ is also necessary and sufficient for the same for the mutants, but we will soon see that this is indeed the case.

2.3 The dynamical system(s)

From Section 1 we already know that if all subpopulation sizes are of order K, then the population size process rescaled by K converges to the solution to the corresponding system of ODEs on any finite time interval of the form $[0, T]$, given convergence of the initial conditions. This is also true for the model with competition-induced dormancy; let us first investigate the situation when only one of the two traits is present in the system, and only afterwards the joint dynamical system of the two traits.

(1) In absence of mutants, for large K, the rescaled resident population $N^K_{1,t}$ can be approximated by $n_1(t)$, where $n_1(\cdot)$ solves the quadratic ODE

$$\dot{n}_1(t) = n_1(t)(\lambda_1 - \mu - \alpha n_1(t)), \tag{2.1}$$

which is again the *logistic* equation (cf. (1.3)) with parameters adapted. Recall the equilibria of this system and their (global) stability properties from Section 1.5. For $\lambda_1 > \mu$, the unique positive (and stable) equilibrium of this system is given as

$$\bar{n}_1 = \frac{\lambda_1 - \mu}{\alpha}.$$

Else, there is no stable positive equilibrium, and 0 is always an equilibrium of the ODE.

(2) Similarly, in absence of residents, for large K, the rescaled mutant population $(N^K_{2a,t}, N^K_{2d,t})$ can be approximated by $(n_{2a}(t), n_{2d}(t))$, where $(n_{2a}(\cdot), n_{2d}(\cdot))$ solves the two-dimensional system of ODEs

$$\begin{aligned} \dot{n}_{2a}(t) &= n_{2a}(t)(\lambda_2 - \mu - \alpha n_{2a}(t)) + \sigma n_{2d}(t), \\ \dot{n}_{2d}(t) &= p\alpha n_{2a}(t)^2 - (\kappa\mu + \sigma) n_{2d}(t). \end{aligned} \tag{2.2}$$

The Jacobi matrix at $(n_{2a}, n_{2d}) \in \mathbb{R}^2$ is given as

$$A(n_{2a}, n_{2d}) = \begin{pmatrix} \lambda_2 - \mu - 2\alpha n_{2a} & \sigma \\ 2p\alpha n_{2a} & -\kappa\mu - \sigma \end{pmatrix}. \tag{2.3}$$

Clearly, the system has no equilibrium of the form $(0, \cdot)$ or $(\cdot, 0)$ apart from $(0, 0)$. Further, we have

$$A(0,0) = \begin{pmatrix} \lambda_2 - \mu & \sigma \\ 0 & -\kappa\mu - \sigma \end{pmatrix}. \tag{2.4}$$

Let us now show that for $\lambda_2 > \mu$ we have a unique (coordinatewise) positive equilibrium, which is asymptotically stable. For an equilibrium (n_{2a}, n_{2d}) with $n_{2a} \neq 0$, dividing both equations in (2.2) by n_{2a}, we obtain

$$\frac{n_{2d}}{n_{2a}} = -\frac{\lambda_2 - \mu - \alpha n_{2a}}{\sigma} = \frac{p\alpha n_{2a}}{\kappa\mu + \sigma}. \tag{2.5}$$

From (2.5) we obtain that there is precisely one such equilibrium, with coordinates

$$\bar{n}_{2a} = \frac{(\lambda_2 - \mu)(\kappa\mu + \sigma)}{\alpha(\kappa\mu + (1-p)\sigma)} > 0, \qquad \bar{n}_{2d} = \frac{(\lambda_2 - \mu)^2 p(\kappa\mu + \sigma)}{\alpha(\kappa\mu + (1-p)\sigma)^2} > 0.$$

It is worth emphasizing that since $p > 0$, $\bar{n}_{2a}$ is strictly larger than $\frac{\lambda_2 - \mu}{\alpha}$. For $p = 0$ (and $\lambda_2 > \mu$), $(\bar{n}_{2a}, 0)$ would be the only coordinatewise nonnegative equilibrium apart from $(0, 0)$, in accordance with (2.1). Thus, we see that even if we disregard the seed bank and consider only the active population size, the equilibrium population size of the mutant population is increased by competition-induced dormancy. The above precise formula for $\bar{n}_{2d}$ will be used less frequently; what is most important is that it is positive whenever $\bar{n}_{2a}$ is positive.

Let us now analyse the local stability of the equilibria (for $\lambda_2 > \mu$) via linearization. As before, we want to identify the signs of the real parts of the eigenvalues of $A(0,0)$ and $A(n_{2a}, n_{2d})$. It is clear from (2.4) that the eigenvalues of $A(0,0)$ are its diagonal entries $\lambda_2 - \mu > 0$ and $-\kappa\mu - \sigma < 0$. Hence, $(0,0)$ is an unstable saddle point. As for the stability of $(\bar{n}_{2a}, \bar{n}_{2d})$, by (2.3) and (2.5), we obtain

$$\det A(\bar{n}_{2a}, \bar{n}_{2d}) = (\kappa\mu + \sigma)(\lambda_2 - \mu).$$

Since $\lambda_2 > \mu$, the right-hand side is positive. Further, the trace $\operatorname{Tr} A(\bar{n}_{2a}, \bar{n}_{2d})$ is negative, which follows from the fact that $\bar{n}_{2a} > \lambda_2 - \mu$ and $\kappa\mu + \sigma > 0$. Hence, the product of the two eigenvalues is positive and their sum is negative. Therefore, if both eigenvalues are real, both must be negative, and if they are complex, they have to be conjugate and hence their real parts must be negative. We conclude that both eigenvalues have a strictly negative real part, which implies that $(\bar{n}_{2a}, \bar{n}_{2d})$ is asymptotically stable.

Exercise 6 (Open problem because we have never checked it, but it should not be too difficult.)**.** *Are the eigenvalues of $A(\bar{n}_{2a}, \bar{n}_{2d})$ always real?*

In fact, the equilibrium $(\bar{n}_{2a}, \bar{n}_{2d})$ exhibits some more global stability properties, in some cases in a certain sense even in presence of the residents, as we will see below.

Exercise 7. *Show that for $\lambda_2 \leq \mu$, the system (2.2) has no coordinatewise positive equilibrium. (For $\lambda_2 < \mu$, $(0,0)$ is of course asymptotically stable.)*

(3) Given the above observations, it is not surprising that if all non-rescaled subpopulation sizes are of order K, $\mathbf{N}_t^K$ can be approximated by the solution $(\mathbf{n}(t)) = (n_1(t), n_{2a}(t), n_{2d}(t))$ to the system of ODEs

$$\begin{aligned} \dot{n}_1(t) &= n_1(t)(\lambda_1 - \mu - \alpha(n_1(t) + n_{2a}(t)), \\ \dot{n}_{2a}(t) &= n_{2a}(t)(\lambda_2 - \mu - \alpha(n_1(t) + n_{2a}(t)) + \sigma n_{2d}(t), \\ \dot{n}_{2d}(t) &= p\alpha n_{2a}(t)(n_1(t) + n_{2a}(t)) - (\kappa\mu + \sigma) n_{2d}(t). \end{aligned} \tag{2.6}$$

If we put $p = 0$ and $n_{2d}(0) = 0$, $(n_1(t), n_{2a}(t))_{t\geq 0}$ would be a classical two-type competitive Lotka–Volterra equation with symmetric competition. It is a well-known result that in that system, $(\frac{\lambda_1 - \mu}{\alpha}, 0) = (\bar{n}_1, 0)$ is asymptotically stable and $(0, \frac{\lambda_2 - \mu}{\alpha})$ is unstable if $\lambda_1 > \lambda_2 > \mu$ and the roles of asymptotic stability and instability are swapped if $\lambda_2 > \lambda_1 > \mu$.This way, intuitively speaking, for $\lambda_2 < \lambda_1$ there is no chance for a mutant invasion in absence of competition-induced dormancy, whereas for $\lambda_2 > \lambda_1$ mutants can invade even in absence of dormancy. Hence, we see that $\lambda_2 < \lambda_1$ is indeed the interesting case to study in our system, where dormancy will actually have a new effect.

To see this, let us study the coordinatewise nonnegative equilibria of the system (2.6) (for $p > 0$). Clearly, $(0,0,0)$ is an equilibrium of the system.

Exercise 8. *Show that $(0,0,0)$ is unstable whenever $\lambda_2 > \mu$ or $\lambda_1 > \mu$ (even without the assumption that $\lambda_1 > \lambda_2$ or that both λ_1 and λ_2 are larger than μ). Show further that for $\lambda_2 \neq \lambda_1$, the system (2.6) can have no equilibrium of the form $(\cdot, 0, 0)$ apart from $(\bar{n}_1, 0, 0)$ and no equilibrium of the form $(0, \cdot, \cdot)$ apart from $(0, \bar{n}_{2a}, \bar{n}_{2d})$.*

Moreover, we easily derive from our previous observations that $(\bar{n}_1, 0, 0)$ and $(0, \bar{n}_{2a}, \bar{n}_{2d})$ are both equilibria of the system. The interesting question is what we can say about their stability and whether the system can have further coordinatewise nonnegative equilibria. According to the statement of Exercise 8, such an equilibrium must be coordinatewise positive. However, we have the following lemma.

Lemma 2.1 ([BT20])**.** *Assume that*

$$\lambda_1 - \lambda_2 \neq p(\lambda_1 - \mu)\frac{\sigma}{\kappa\mu + \sigma}. \tag{2.7}$$

Then, (2.6) exhibits no coordinatewise positive equilibrium.[18]

Proof. Assume that there exists a coordinatewise positive equilibrium, say (n_1, n_{2a}, n_{2d}). Expressing n_1 from the first line of (2.6) and substituting it into the second and third line divided by n_{2a} yields

$$\frac{n_{2d}}{n_{2a}} = \frac{\lambda_1 - \lambda_2}{\sigma} = \frac{1}{\kappa\mu + \sigma} p(\lambda_1 - \mu),$$

but the last inequality contradicts (2.7). We conclude the claim. □

Exercise 9. *Determine all equilibria of (2.6) in the degenerate case $\lambda_2 - \lambda_1 = p(\lambda_1 - \mu)\frac{\sigma}{\kappa\mu+\sigma}$ (in case our usual assumptions all hold).*

[18] This lemma is only partially included in [BT20], but extending it does not requires any new ideas or computations.

To provide a biological interpretation to Lemma 2.1, the *competitive exclusion principle* applies for the system (2.6) (if (2.7) holds): Only one of the two subpopulations can remain asymptotically positive, and the other has to vanish. This is in a certain sense not only true for the deterministic dynamical system (2.6) but also for our stochastic individual-based model $(\mathbf{N}_t^K)_{t\geq 0}$ in the limit $K \to \infty$, as we will see below; the interpretation with that respect is that *invasion implies fixation*, i.e., there is no macroscopic coexistence between types 1 and 2. The competitive exclusion principle holds in general for classical two-dimensional Lotka–Volterra systems with symmetric competition (without dormancy) apart from some degenerate cases, but asymmetric competition may lead to a coexistence between the two types. (This is also the reason why in Section 1 we needed Assumption (B) to exclude coexistence.) This way, the advantage of type 2 gained from competition-induced dormancy does not qualify as asymmetric competition (or the above interpretation is not valid for our model, which is strictly speaking not a Lotka–Volterra model).

Given Lemma 2.1, the next question is which of the two equilibria $(\bar{n}_1, 0, 0)$, $(0, \bar{n}_{2a}, \bar{n}_{2d})$ are (asymptotically) stable. The following lemma will be crucial for our analysis.

Lemma 2.2 ([BT20])**.** *Assume that the condition*

$$\lambda_1 - \lambda_2 < p(\lambda_1 - \mu)\frac{\sigma}{\kappa\mu + \sigma} = p\alpha\bar{n}_1\frac{\sigma}{\kappa\mu + \sigma}. \tag{2.8}$$

holds. Then, $(\bar{n}_1, 0, 0)$ *is unstable and* $(0, \bar{n}_{2a}, \bar{n}_{2d})$ *is asymptotically stable. On the other hand, if*

$$\lambda_1 - \lambda_2 > p(\lambda_1 - \mu)\frac{\sigma}{\kappa\mu + \sigma} = p\alpha\bar{n}_1\frac{\sigma}{\kappa\mu + \sigma}, \tag{2.9}$$

then $(\bar{n}_1, 0, 0)$ *is asymptotically stable and* $(0, \bar{n}_{2a}, \bar{n}_{2d})$ *is unstable.*

Proof. Let us first assume that (2.8) holds. At any equilibrium (n_1, n_{2a}, n_{2d}), the Jacobi matrix is given as

$$B(n_1, n_{2a}, n_{2d}) = \begin{pmatrix} \lambda_1 - \mu - 2\alpha n_1 - \alpha n_{2a} & -\alpha n_1 & 0 \\ -\alpha n_{2a} & \lambda_2 - \mu - 2\alpha n_{2a} - \alpha n_1 & \sigma \\ p\alpha n_{2a} & 2p\alpha n_{2a} + p\alpha n_1 & -(\kappa\mu + \sigma) \end{pmatrix}.$$

At $(\bar{n}_1, 0, 0)$, since $\alpha n_1 = \lambda_1 - \mu$, the Jacobian matrix is

$$B(\bar{n}_1, 0, 0) = \begin{pmatrix} -\lambda_1 + \mu & -\lambda_1 + \mu & 0 \\ 0 & \lambda_2 - \lambda_1 & \sigma \\ 0 & p(\lambda_1 - \mu) & -(\kappa\mu + \sigma) \end{pmatrix}. \tag{2.10}$$

We see that $-\lambda_1 + \mu < 0$ is an eigenvalue of this matrix (with eigenvector $(1, 0, 0)$). The determinant of the matrix is

$$\det B(\bar{n}_1, 0, 0) = -(\lambda_1 - \mu)((\lambda_2 - \lambda_1)(-\kappa\mu - \sigma) - p(\lambda_1 - \mu)\sigma).$$

Now, since $\lambda_1 > \mu$, further, thanks to (2.8),

$$(\lambda_1 - \lambda_2)(\kappa\mu + \sigma) - p(\lambda_1 - \mu)\sigma < 0. \tag{2.11}$$

Since $-\lambda_1 + \mu < 0$ is an eigenvalue of $B(\bar{n}_1, 0, 0)$, the left-hand side of (2.11) is equal to the product of the two other eigenvalues of $B(\bar{n}_1, 0, 0)$ and also to the determinant of its last 2×2 block. Since it is negative, this implies that the eigenvalues of that 2×2 block are real, and thus one of them must be positive. This implies that $(\bar{n}_1, 0, 0)$ is unstable. Finally, let us consider the equilibrium $(0, \bar{n}_{2a}, \bar{n}_{2d})$. We have

$$B(0, \bar{n}_{2a}, \bar{n}_{2d}) = \begin{pmatrix} \lambda_1 - \mu - \alpha\bar{n}_{2a} & 0 & 0 \\ 0 & \lambda_2 - \mu - 2\alpha\bar{n}_{2a} & \sigma \\ p\alpha\bar{n}_{2a} & 2p\alpha\bar{n}_{2a} & -(\kappa\mu + \sigma) \end{pmatrix}.$$

Now, note that

$$\begin{aligned} \bar{n}_{2a} > \bar{n}_1 \quad &\Leftrightarrow \quad \lambda_2 - \mu > (\lambda_1 - \mu)\big(1 - \frac{p\sigma}{\kappa\mu + \sigma}\big) \\ &\Leftrightarrow \lambda_1 - \lambda_2 < (\lambda_1 - \mu)p\frac{\sigma}{\kappa\mu + \sigma} \quad \Leftrightarrow \ (2.8). \end{aligned} \tag{2.12}$$

Thus, $\lambda_1 - \mu - \alpha\bar{n}_{2a} < 0$ under condition (2.8), and this quantity is clearly an eigenvalue of the matrix $B(0, \bar{n}_{2a}, \bar{n}_{2d})$. The other two ones are the eigenvalues of the last 2×2 block of the matrix, which is the same as $A(\bar{n}_{2a}, \bar{n}_{2d})$ defined according to (2.3) (with $n_{2a} = \bar{n}_{2a}$ and $n_{2d} = \bar{n}_{2d}$). We have already seen that these eigenvalues have negative real parts for $\lambda_2 > \mu$. We conclude that $B(0, \bar{n}_{2a}, \bar{n}_{2d})$ has three eigenvalues with negative real parts and hence the equilibrium $(0, \bar{n}_{2a}, \bar{n}_{2d})$ is asymptotically stable under condition (2.8).

The proof in the case when (2.9) holds is analogous ((2.9) is equivalent to $\bar{n}_{2a} < \bar{n}_1$). □

Under the assumption (2.8), the equilibrium $(0, \bar{n}_{2a}, \bar{n}_{2d})$ turns out to be not only locally asymptotically stable, but it even attracts solutions with certain coordinatewise positive, more distant initial conditions, see Lemma 2.18 below.

2.4 Overview of the three phases of an invasion

Thanks to Lemma 2.1, there are choices of the parameters such that $(0, \bar{n}_{2a}, \bar{n}_{2d})$ is asymptotically stable and $(\bar{n}_1, 0, 0)$ is unstable, in other words, when thanks to dormancy, type 2 is fitter than type 1 despite $\lambda_2 < \lambda_1$, namely precisely the choices of parameters satisfying (2.8). This is a strong indication that for such choices of the parameters, if we start our stochastic individual-based model with approximately $K\bar{n}_1$ residents, one active mutant, and no dormant mutant, invasion and complete fixation of type 2 should be possible with asymptotically positive probability as $K \to \infty$, and we will soon be able to provide a biological interpretation for condition (2.8) in terms of our stochastic process.

Since our main question is whether the probability that a mutant population started with a single mutant individual is able to invade a resident population living in equilibrium is asymptotically positive as $K \to \infty$, we will be interested in initial conditions $\mathbf{N}_0^K = (N_{1,0}^K, N_{2a,0}^K, N_{2d,0}^K)$ such that $N_{1,0}^K \approx \bar{n}_1$ (in a sense that will be clarified later) and $(N_{2a,0}^K, N_{2d,0}^K) = (1/K, 0)$, i.e., there is one active mutant and there are zero dormant mutants at time zero. From the following analysis it is straightforward to derive how to handle the case when one starts with one dormant mutant instead of one active one, see Exercise 10 below.

The analysis is then similar to what we have seen in Section 1 in the case of one single invasion, with the main difference being that the mutant population is two-dimensional:

(I) The rescaled resident population size $N_{1,t}^K$ stays close to its equilibrium $\bar{n}_1$ for a sufficiently long time, thanks to Freidlin–Wentzell type large-deviation results [FW84] analogous to Section 1.8. Given this, we can approximate the two-type (non-rescaled) mutant population size process $(N_{2a,t}, N_{2d,t})_t$ by a two-type linear branching process until the total mutant population size $N_{2a,t} + N_{2d,t}$ reaches 0 or εK for some small $\varepsilon > 0$ (chosen independently of K). We will see that the latter happens with asymptotically positive probability (i.e., the branching process is supercritical) under condition (2.8) and it happens with vanishing probability (the branching process is subcritical) under condition (2.9) in the limit $K \to \infty$ followed by $\varepsilon \downarrow 0$. (We will ignore the critical case when neither of the aforementioned two conditions hold).
In the case of a successful invasion, reaching εK for fixed $\varepsilon > 0$ small takes $\Theta(\log K)$ time as $K \to \infty$; in the limit $\varepsilon \downarrow 0$ we will also be able to identify the asymptotic prefactor of $\log K$. In the case of an unsuccessful invasion, mutant extinction takes $o(\log K)$ time, and the rescaled resident subpopulation size will stay close to $\bar{n}_1$ until this extinction with high probability.

(II) In the latter case, once $N_{2a,t} + N_{2d,t}$ has reached εK, the three-type rescaled population size process $(\mathbf{N}_t^K)_t$ can be approximated by the dynamical system (2.6). Our aim is to show that started from a suitably chosen initial condition that $(N_{2a,t}^K, N_{2d,t}^K)_t$ reaches with high probability conditional on the survival of the branching process, the solution to the dynamical system converges to $(0, \bar{n}_{2a}, \bar{n}_{2d})$. Given

this, starting from such an initial condition, the dynamical system reaches a fixed $\widetilde{\varepsilon}$-neighbourhood of this equilibrium within $O(1)$ time.[19]

(III) After Phase II, $(N^K_{2a,t}, N^K_{2d,t})$ is close to $(\bar{n}_{2a}, \bar{n}_{2d})$ and $N_{1,t}$ is at most $\widetilde{\varepsilon}K$ for some $\widetilde{\varepsilon} > 0$. Similarly to the rescaled case of the resident population during Phase I, we show that $(N^K_{2a,t}, N^K_{2d,t})$ stays close to $(\bar{n}_{2a}, \bar{n}_{2d})$ for a sufficiently large amount of time. Given this, $N_{1,t}$ can be approximated by a branching process, which is subcritical under condition (2.8). This branching process does not become much larger again before it dies out (cf. Remark 1.23), and its extinction takes $\Theta(\log K)$ time starting from the end of Phase II, where we will again be able to identify the prefactor of $\log K$ in the limit $\widetilde{\varepsilon} \downarrow 0$ following $K \to \infty$.

2.5 Multitype branching processes I: general theory and particular heuristics

The key principles of the analysis of multitype branching processes (in continuous time) [AN72] are very similar to the ones of single-type ones explained in Section 1.9, but some important new objects arise that were trivial in the one-dimensional case, which are associated to the *mean matrix* of the branching process. The principles of the branching process approximation of a small mutant population are also unchanged: We assume that the resident population size divided by K stays close to its equilibrium and ignore non-linear interactions among the mutants, which makes the transition rates for the mutant population linear in the number of individuals. In this section, instead of presenting general results on multitype branching processes, we will focus on the case of our particular example, hoping that given this and the general one-dimensional theory presented in Section 1.9 the reader can also treat the case of other, mathematically similar examples.

The way these branching processes approximate our mutant subpopulation in Phase I resp. our resident subpopulation in Phase III will be similar to the case of branching process approximations in Section 1. We will not fully explain the couplings, but at least we will provide some crucial details in Section 2.7 below. For the moment being, let us now provide a heuristic description of the branching process approximations in Phases I and III.

Assuming that $N^K_{1,t}$ is close to $\bar{n}_1$, the principle of the branching process approximation is that we assume that $N^K_{1,t}$ is constant equal to $\bar{n}_1$. As long as $N_{2a,t}$ and $N_{2d,t}$ are small compared to K, self-competition of active mutant individuals can be ignored because competitive events involving two active mutants arise at rate $\alpha\frac{N_{2a,t}(N_{2a,t}-1)}{K}$, which is small compared to the approximate rate $\alpha N_{2a,t}\bar{n}_1$ of competitive events affecting active mutants and caused by residents. Thus, the dynamics of the mutant population size process $(N_{2a,t}, N_{2d,t})$ can be approximated by a two-type linear branching process $(\widehat{Z}_{2a}(t), \widehat{Z}_{2d}(t))$ with rates

$$(n_{2a}, n_{2d}) \to \begin{cases} (n_{2a}+1, n_{2d}) \text{ at rate } n_{2a}\lambda_2, \\ (n_{2a}-1, n_{2d}) \text{ at rate } n_{2a}(\mu + \alpha\bar{n}_1(1-p)), \\ (n_{2a}-1, n_{2d}+1) \text{ at rate } n_{2a}\bar{n}_1\alpha p, \\ (n_{2a}+1, n_{2d}-1) \text{ at rate } \sigma n_{2d}, \\ (n_{2a}, n_{2d}-1) \text{ at rate } \kappa\mu n_{2d}. \end{cases}$$

The continuous-time Markov chain with these rates is indeed a (two-type) branching process because every individual gives birth to individuals reproducing according to the same rules independently of all the other individuals (where the rates are linear in n_{2a} resp. n_{2d}). By classical results on multitype branching processes [AN72, Section 7.2], the process is supercritical, i.e., there is no almost sure convergence to $(0,0)$, if and only if the following *mean matrix* has a positive eigenvalue

$$J = \begin{pmatrix} \lambda_2 - \mu - \alpha\bar{n}_1 & p\alpha\bar{n}_1 \\ \sigma & -\kappa\mu - \sigma \end{pmatrix} = \begin{pmatrix} \lambda_2 - \lambda_1 & p(\lambda_1 - \mu) \\ \sigma & -\kappa\mu - \sigma \end{pmatrix}. \tag{2.13}$$

In general, the j-th element of the i-th row of the mean matrix of a multitype branching process with n types is the expected number of type i individuals created by the actions of a type j individual (taking also

[19] It is not entirely correct to say that Phase II takes $O(1)$ time in total, for the same reason as what we wrote in Section 1.6.

deaths into account). Note that these rates may be negative in some cases, e.g. the diagonal entries $\lambda_2 - \lambda_1$ and $-\kappa\mu - \sigma$ are negative in our model. (If there are negative off-diagonal elements, certain classical results of the theory of multitype branching processes are not always applicable, but this is luckily not the case in our model.)

In the interesting case $\lambda_2 < \lambda_1$, it is impossible that we have two positive eigenvalues because Tr $J < 0$ follows from the definition of $\bar{n}_1$. To describe the condition that J has a positive eigenvalue more explicitly, let us first consider the sign of $\det J$. In case there is precisely one positive eigenvalue, the determinant must be negative, which is equivalent to

$$\lambda_1 - \lambda_2 < p(\lambda_1 - \mu)\frac{\sigma}{\kappa\mu + \sigma} = p\alpha\bar{n}_1\frac{\sigma}{\kappa\mu + \sigma}$$

which is precisely the condition (2.8) (under which $(\bar{n}_1, 0, 0)$ was an unstable and $(0, \bar{n}_{2a}, \bar{n}_{2d})$ an asymptotically stable equilibrium of (2.6)).

Indeed, since $\kappa \geq 0$, the characteristic equation in the variable λ corresponding to the matrix J in (2.8) is

$$\lambda^2 + (\lambda_1 - \lambda_2 + \kappa\mu + \sigma)\lambda + \det J = 0.$$

This quadratic equation always has two different real solutions if $\det J$ is negative, and hence one of the eigenvalues of J must indeed be positive if (2.8) holds. The condition (2.8) turns out to be necessary and sufficient for the invasion probability to be asymptotically positive.

Remark 2.3. The biological interpretation of the supercriticality condition (2.8) is that the selective advantage $\lambda_1 - \lambda_2$ of trait 1 due to its higher reproduction rate (and thus by the same amount higher net growth rate since the natural death rates of both traits equal μ) is smaller than the selective advantage $\bar{n}_1\frac{\sigma}{\kappa\mu+\sigma}$ of trait 2. The latter quantity is the mean amount of mutant (trait 2) individuals surviving competition with the (trait 1) residents when the mutant population is small. Indeed, the per capita death rate of competitive events is $\alpha\bar{n}_1$ per mutant individual. While competitive events always cause instantaneous death for the residents, mutants survive them with probability p and then with probability $\sigma/(\kappa\mu + \sigma)$ they manage to resuscitate before they would die in a dormant state, hence their aforementioned selective advantage.

It is also remarkable that (2.8) is equivalent to $\bar{n}_1 > \bar{n}_{2a}$ (cf. (2.12)), i.e. that the active equilibrium population size of the mutants is larger than the one of the residents. One interesting aspect of this is that the dormant mutant equilibrium size $\bar{n}_{2d}$ plays no direct role here. From the analogue of Section 1 it is now at least intuitively clear that in our model there is no coexistence between residents and mutants (apart from possibly some critical cases), and hence the probability of a successful mutant invasion stays asymptotically positive as $K \to \infty$ if and only if (2.8) holds, and from Lemma 2.1 one can also suspect that such an invasion will also imply the fixation of mutants and the extinction of residents. Thus, a successful mutant invasion always increases the active equilibrium population size, just as in the case of the model of [C06] with symmetric competition (see Remark 1.22).

In the case $\kappa = 0$ of no death in the seed bank, condition (2.8) reduces to

$$\frac{\lambda_1 - \mu}{1} < \frac{\lambda_2 - \mu}{1 - p},$$

where $1 - p$ is the probability that a mutant affected by a competitive event dies. On the complementary event, this mutant will eventually become active again.[20]

We further note that the largest eigenvalue of the matrix J defined in (2.13) is given as follows:

$$\widetilde{\lambda} = \frac{1}{2}\Big((\lambda_2 - \lambda_1 - \kappa\mu - \sigma) + \sqrt{(\lambda_1 - \lambda_2 + \kappa\mu + \sigma)^2 - 4\big((\lambda_1 - \lambda_2)(\kappa\mu + \sigma) - p(\lambda_1 - \mu)\sigma\big)}\Big). \tag{2.14}$$

[20]Note that $\lambda_2 > \mu$ automatically follows from (2.8) given that $\lambda_1 > \mu$. Thus, our model is free from *evolutionary suicide*: mutants who are not able to survive on their own will not make the resident population go extinct with asymptotically positive probability. Models involving the possibility of evolutionary suicide will appear in Section 4.

According to [AN72, Section 7], $\widetilde{\lambda}$ is equal to the mean exponential growth rate of the approximating branching process $(\widehat{Z}_{2a}(t), \widehat{Z}_{2d}(t))$. What this precisely means will be clear when we have discussed the Kesten–Stigum theorem (Theorem 2.13); right now we just enlighten that conditional on survival, as $t \to \infty$, $(\widehat{Z}_{2a}(t), \widehat{Z}_{2d}(t))$ behaves approximately like $\mathrm{e}^{\widetilde{\lambda}t}$ times a deterministic vector with positive coordinates, modulo subexponential correction terms. This way, the time it takes for the branching process to reach a population size of order εK (where $\varepsilon > 0$ is small but independent of K) is about $(\frac{1}{\widetilde{\lambda}} + \phi(\varepsilon)) \log K$, where $\phi(\varepsilon)$ is some positive and increasing function tending to 0 as $\varepsilon \downarrow 0$. Thanks to our branching process approximation, Phase I of a successful invasion for our original stochastic individual-based model will also take approximately the same amount of time conditional on survival. On the other hand, conditional on extinction, $(\widehat{Z}_{2a}(t), \widehat{Z}_{2d}(t))$ tends to $(0, 0)$ almost surely, and hence in our model, an unsuccessful invasion will take $o(\log K)$ time as $K \to \infty$. All this is similar to Remark 1.21 in the case of single-type setting, where the largest eigenvalue of the 1×1 mean matrix is of course equal to its only entry.

Using our multitype branching process approach, now we can compute the *extinction probability* under condition (2.8) with $\lambda_1 > \lambda_2 > \mu$. Define

$$s_a = \mathbb{P}\big(\exists t < \infty \colon \widehat{Z}_{2a}(t) + \widehat{Z}_{2d}(t) = 0 \big| (\widehat{Z}_{2a}(0), \widehat{Z}_{2d}(0)) = (1, 0)\big). \tag{2.15}$$

By [AN72, Section 7], s_a is the first coordinate of the unique solution to the system of equations

$$\begin{aligned} \lambda_2(s_a^2 - s_a) + p(\lambda_1 - \mu)(s_d - s_a) + (\mu + (1-p)(\lambda_1 - \mu))(1 - s_a) = 0, \\ \sigma(s_a - s_d) + \kappa\mu(1 - s_d) = 0, \end{aligned} \tag{2.16}$$

in $[0, 1]^2 \setminus \{(1, 1)\}$, while the second coordinate of the same solution is the extinction probability given that the branching process is started from $(0, 1)$. The system of generating equations (2.16) is obtained via the same first-step analysis as the extinction probability of a one-type branching process (cf. the proof of Theorem 1.19).

- E.g., the term $\lambda_2(s_a^2 - s_a)$ is obtained as follows. If one starts the branching process with one active individual and it reproduces first (which happens at rate λ_2), then it will create another identical individual, so that from the new state one has to kill two independent copies of the original process in order to make the branching process extinct, which has probability s_a^2.
- The term $p(\lambda_1 - \mu)(s_d - s_a)$ expresses that if the first action an active individual takes is going dormant (which happens at rate $p(\lambda_1 - \mu)$, then in the resulting state one has to kill a dormant individual instead of an active one, which has probability s_d.
- Finally, the term $(\mu + (1-p)(\lambda_1 - \mu))(1 - s_a)$ explains that if the first action the active individual takes is death, which happens naturally at rate μ or via competition at rate $(1-p)(\lambda_1 - \mu)$, then the process immediately goes extinct, so the probability of extinction becomes 1.

The rates in the second line of (2.16) are obtained similarly, now starting with one dormant individual instead of an active one.

Remark 2.4. It is worth observing that the mean matrix J (cf. (2.13)) is identical to the transpose of the last 2×2 block of the Jacobi matrix $B(\bar{n}_1, 0, 0)$ of the dynamical system (2.6) at the equilibrium $(\bar{n}_1, 0, 0)$ (cf. (2.10)). This is not a coincidence; in general the following is true in models of population dynamics. Put a mutant into a (possibly multitype) resident population whose size rescaled by K is initially close to an equilibrium whose projection to the resident coordinates is asymptotically stable w.r.t. the subsystem of the corresponding dynamical system consisting of the resident types (here the equilibrium is $(\bar{n}_1, 0, 0)$, its projection is $\bar{n}_1$ and the sub-system is (2.1)). Then, the mean matrix of the branching process will be equal to the transpose of the submatrix of the Jacobi matrix of the entire dynamical system at this equilibrium given by the indices corresponding to the mutant types. The same was of course also true in the setting of Remark 1.24, where the mean matrix was 1×1.

In general, apart from critical cases, the linearization of the dynamical system around the equilibrium (consisting in deriving the Jacobi matrix at the equilibrium and determining its local stability) can be seen as the *same* linearization as the transformation of the corresponding stochastic population process to the branching process with the resident subpopulation sizes fixed and nonlinear interactions between mutants ignored. Since the projection of the equilibrium was asymptotically stable, eigenvalues of the Jacobi matrix corresponding only to the resident types all have negative real parts, and hence all eigenvalues of the Jacobi matrix have negative real parts if and only if the branching process is subcritical, whereas there exists an eigenvalue with positive real part if and only if the branching process is supercritical.

We should however emphasize right at the beginning that this is only a *local* correspondence between the world of dynamical systems and stochastic individual-based models, which does not allow one to draw any conclusion e.g. about the global stability of equilibria of the dynamical system or the corresponding convergence of the rescaled stochastic process to the equilibria starting from distant initial conditions for large K.

As for Phase III, after the second phase of invasion, the population rescaled by $1/K$ is close to the equilibrium $(0, \bar{n}_{2a}, \bar{n}_{2d})$. To be more precise, the resident population size is of order εK for some $\varepsilon > 0$ small. It remains to show that for large K, with probability tending to one, the resident population dies out within $\Theta(\log K)$ time, while the mutant population stays close to equilibrium, and to identify the corresponding prefactor of $\log K$. Now, as long as $(N^K_{2a,t}, N^K_{2d,t})$ is near $(\bar{n}_{2a}, \bar{n}_{2d})$ and the resident population is small compared to K, the competitive pressure that the resident individuals feel comes essentially only from the mutant population. This implies that $N_{1,t}$ can be approximated by a branching process $\widehat{Z}_1(t)$ with rates

$$n_1 \to \begin{cases} n_1 + 1 & \text{at rate } n_1\lambda_1, \\ n_1 - 1 & \text{at rate } n_1(\mu + \alpha\bar{n}_{2a}) \end{cases}.$$

In order to show that this branching process goes extinct almost surely, we have to verify that it is subcritical, i.e., the rate $n \to n+1$ is smaller than the rate $n \to n-1$. But this assertion is equivalent to the inequality (2.8).

The branching process decays approximately like $\widehat{Z}_1(0)\mathrm{e}^{-\widehat{\lambda}t}$, where

$$\widehat{\lambda} = \alpha\bar{n}_{2a} + \mu - \lambda_1 = \alpha(\bar{n}_{2a} - \bar{n}_1) > 0 \tag{2.17}$$

can also be interpreted as the only element of the 1×1 mean matrix of the branching process. Hence, it takes about $1/\widehat{\lambda}$ time for the branching process (and the resident population in the individual-based model) to go extinct, again with ε-dependent correction terms.

Summarizing, we expect that under condition (2.8), the duration T_{fix} of an entire successful mutant invasion until complete fixation of the mutants and extinction of the residents satisfies

$$\lim_{K\to\infty} \frac{T_{\mathrm{fix}}}{\log K} = \frac{1}{\widetilde{\lambda}} + \frac{1}{\widehat{\lambda}}$$

in probability (note that here, there is reference to ε anymore), and the probability of success of the invasion tends to $1-q \in (0,1)$. In the next section, we will state our main results, which include a somewhat stronger version of this assertion and also treat the case of a failed mutant invasion.

Remark 2.5. Continuing Remark 2.4, it is again not a coincidence that all eigenvalues of the Jacobi matrix $B(0, \bar{n}_{2a}, \bar{n}_{2d})$ have negative real parts if and only if the branching process $(\widehat{Z}_1(t))_{t\geq 0}$ is subcritical, and similarly, it is true that the Jacobi matrix has an eigenvalue with positive real part if and only if the branching process is supercritical. In general, the conclusions of Remark 2.4 are also true for Phase III of an invasion, with the caveat that linearization does not tell about global stability, and it is only reasonable to study Phase III if Phase II indeed leads the dynamical system/the stochastic individual-based model to the vicinity of the corresponding equilibrium. It could happen for example (not in the model of [BT20] but in other ones) that the resident and the mutant stably coexist, whence with high probability, a small neighbourhood of the one-type equilibrium of the mutant will never be reached.

Of course, one could always study the reverse invasion direction, e.g. in the case of the competition-induced dormancy model one could ask whether a trait 1 mutant can invade a trait 2 population initially living in equilibrium. Then, Phase I would correspond to the original Phase III and vice versa, and therefore it is straightforward to conjecture when invasion would be possible and when not. However, this invasion direction was not studied in [BT20], and handling Phase II of the reverse invasion direction presumably requires additional ideas that are not included in [BT20] (but likely follow from ideas of [BT21] corresponding to the reverse invasion direction in an extended version of the model with the additional feature of horizontal gene transfer).

2.6 Main results of [BT20] and discussion

Recall that we have assumed $\lambda_1 > \lambda_2 > \mu > 0$, and recall also the stable equilibrium $(\bar{n}_{2a}, \bar{n}_{2d})$, which is the unique solution to the system of equations (2.5) under the assumption $\lambda_2 > \mu$. For $\beta > 0$ define the *invasion set*

$$S_\beta = \{0\} \times [\bar{n}_{2a} - \beta, \bar{n}_{2a} + \beta] \times [\bar{n}_{2d} - \beta, \bar{n}_{2d} + \beta], \tag{2.18}$$

a stopping time at which $\mathbf{N}_t^K$ reaches this set:

$$T_{S_\beta} := \inf\{t > 0 \colon \mathbf{N}_t^K \in S_\beta\}, \tag{2.19}$$

and the first time when the rescaled mutant population size reaches a threshold $x \geq 0$ (from below or above):

$$T_x^2 := \inf\{t > 0 \colon N_{2,t} = \lfloor xK \rfloor\}. \tag{2.20}$$

In particular, T_0^2 is the extinction time of trait 2. Recall also the eigenvalue $\widetilde{\lambda}$ defined in (2.14) and the extinction probability s_a from (2.16). The first main result of [BT20] characterizes the probability of mutant invasion in the large-population limit.

Theorem 2.6 ([BT20]). *Assume that* (2.8) *holds. Assume further that*

$$N_1^K(0) \underset{K\to\infty}{\to} \bar{n}_1$$

in probability and

$$(N_{2a}^K(0), N_{2d}^K(0)) = (\tfrac{1}{K}, 0).$$

Then for any $0 < \beta < \min\{\bar{n}_{2a}, \bar{n}_{2d}\}$*, we have*

$$\lim_{K\to\infty} \mathbb{P}\Big(T_{S_\beta} < T_0^2\Big) = 1 - s_a.$$

Next, we identify the time of fixation of mutants in the case of a successful invasion.

Theorem 2.7 ([BT20]). *Under the assumptions of Theorem 2.6, we have that on the event* $\{T_{S_\beta} < T_0^2\}$*,*

$$\lim_{K\to\infty} \frac{T_{S_\beta}}{\log K} = \frac{1}{\widetilde{\lambda}} + \frac{1}{\mu + \alpha\bar{n}_{2a} - \lambda_1} = \frac{1}{\widetilde{\lambda}} + \frac{1}{\widehat{\lambda}} \tag{2.21}$$

in probability.

Finally, we show that in case of an unsuccessful mutation, with high probability, the extinction takes a sub-logarithmic time (in particular, the extinction happens during the first phase of the invasion), and at the time of extinction the resident population is close to its equilibrium population size.

Theorem 2.8 ([BT20]). *Under the assumptions of Theorem 2.6, we have that on the event* $\{T_0^2 < T_{S_\beta}\}$*,*

$$\lim_{K\to\infty} \frac{T_0^2}{\log K} = 0 \tag{2.22}$$

and

$$\mathbb{1}\{T_{S_\beta} > T_0^2\}\Big|\mathbf{N}_{T_0^2}^K - (\bar{n}_1, 0, 0)\Big| \underset{K\to\infty}{\to} 0, \tag{2.23}$$

both in probability.

Now we can see the precise meaning of "invasion implies fixation" in our model: With probability tending to 1 as $K \to \infty$, we either have a complete fixation of mutants and an extinction of residents (which happens with probability tending to $1 - s_a$) or a rapid extinction of mutants with nearly unaffected residents (which occurs with probability tending to s_a).

Exercise 10 (Starting with one dormant individual)**.** *Using the equation* (2.16)*, provide the analogoues of Theorems 2.6, 2.7, and 2.8 for the case when* $\lim_{K\to\infty} N^K_{1,0} = \bar{n}_1$ *in probability and* $(N^K_{2a,0}, N^K_{2d,0}) = (0, 1/K)$ *(and express* s_d *in terms of* s_a*).*

Remark 2.9. We have seen that the two-type mutant population is able to survive on its own if $\lambda_2 > \mu$, and if (2.8) holds, then the mutants will invade the population with positive probability even if $\lambda_2 < \lambda_1$. We also noted that without the mutants having a dormancy trait (i.e., for $p = 0$), even though mutants can still survive on their own as soon as $\lambda_2 > \mu$, invasion is not possible (because the branching process remains subcritical) as long as $\lambda_2 < \lambda_1$.

For $\kappa > 0$, it is not even the case that mutants are fit on their own if the switching from activity to dormancy is not competition-induced but *spontaneous*, i.e., if an active mutant individual switches to dormancy at some fixed rate $\sigma' > 0$. There, in absence of residents, for large K, the rescaled mutant population is approximated by the system of ODEs

$$\begin{aligned} \dot{n}_{2a}(t) &= n_{2a}(t)(\lambda_2 - \mu - \alpha n_{2a}(t) - \sigma') + \sigma n_{2d}(t), \\ \dot{n}_{2d}(t) &= \sigma' n_{2a}(t) - (\kappa\mu + \sigma) n_{2d}(t). \end{aligned} \tag{2.24}$$

Hence, the origin is asymptotically stable if and only if $(\lambda_2 - \mu - \sigma')(-\kappa\mu - \sigma) - \sigma\sigma' < 0$, i.e.,

$$\lambda_2 < \mu + \frac{\kappa\mu\sigma'}{\kappa\mu + \sigma}. \tag{2.25}$$

I.e., there are values $\lambda_2 > \mu$ such that the mutant population dies out with high probability if $K \to \infty$. The right-hand side of (2.25) is the *effective death rate*: indeed, an active individual dies at rate μ, but additionally at rate σ' it becomes dormant, where it dies with probability $\frac{\kappa\mu}{\kappa\mu+\sigma}$ before ever becoming active (and capable of reproduction) again.

2.7 Outline of the proof

The general strategy of the proof of the above three theorems is very similar to the treatment of the three phases of one invasion step within the proof of [C06]; for example, the Freidlin–Wentzell type large deviations and results on diffusion exit from a domain in Phase I are applied in a way that is similar to Section 1.8. This leads to the following proposition, where for $\varepsilon > 0$ we define

$$R_\varepsilon = \inf\{t \geq 0 \colon \left|N^K_{1,t} - \bar{n}_1\right| > \varepsilon\},$$

the first time when the resident population leaves an ε-neighbourhood of its equilibrium size $\bar{n}_1$.

Proposition 2.10 ([BT20], based on methods of [CCLLS21])**.** *Assume that* (2.8) *holds. Let* $K \mapsto m^K_1$ *be a function from* $(0,\infty)$ *to* $[0,\infty)$ *such that* $m^K_1 \in \frac{1}{K}\mathbb{N}_0$ *and* $\lim_{K\to\infty} m^K_1 = \bar{n}_1$*. Then there exists a function* $f \colon (0,\infty) \to (0,\infty)$ *tending to zero as* $\varepsilon \downarrow 0$ *such that for any* $\xi \in [1/2, 1]$*,*

$$\limsup_{K\to\infty} \left|\mathbb{P}\Big(T^2_{\varepsilon^\xi} < T^2_0 \wedge R_{2\varepsilon}, \Big|\frac{T^2_{\varepsilon^\xi}}{\log K} - \frac{1}{\tilde{\lambda}}\Big| \leq f(\varepsilon) \,\Big|\mathbf{N}^K_0 = \big(m^K_1, \frac{1}{K}, 0\big)\Big) - (1 - s_a)\right| = o_\varepsilon(1) \tag{2.26}$$

and

$$\limsup_{K\to\infty} \left|\mathbb{P}\Big(T^2_0 < T^2_{\varepsilon^\xi} \wedge R_{2\varepsilon} \,\Big|\mathbf{N}^K_0 = \big(m^K_1, \frac{1}{K}, 0\big)\Big) - s_a\right| = o_\varepsilon(1), \tag{2.27}$$

where $o_\varepsilon(1)$ *tends to zero as* $\varepsilon \downarrow 0$*.*

We refer the reader to [BT20] for a proof; many of the proof techniques applied here are borrowed from Coron, Costa, Leman, Laroche, and Smadi [CCLLS21], who studied a mathematically similar invasion model with a different biological motivation (namely, the emergence of homogamy in a two-loci stochastic population model), given that the multitype setting of their paper is much more convenient to adapt than the single-type methods of [C06]. A major step in the proof of Proposition 2.10 is the following lemma.

Lemma 2.11 ([BT20], based on methods of [CCLLS21])**.** *Under the assumptions of Proposition 2.10, there exists a positive constant ε_0 such that for any $\xi \in [1/2, 1]$ and $0 < \varepsilon \le \varepsilon_0$,*

$$\limsup_{K\to\infty} \mathbb{P}\big(R_{2\varepsilon} \le T^2_{\varepsilon^\xi} \wedge T^2_0\big) = 0.$$

The proof of this lemma ([BT20, Lemma 4.2]) can be found in [BT20]. The first step of the proof is to use the bound $\varepsilon^\xi K$ on the mutant subpopulation sizes in order to couple the resident population size between two (ε-dependent) birth-and-death processes scaling to two (ε-dependent) ODEs with qualitative behaviour analogous to (2.1). Given this, we can perform a large-deviation analysis similar to Section (1.8) to show that the rescaled resident population size stays close to equilibrium for a sufficiently long amount of time. Having this, we borrow some additional moment estimates and arguments of linear algebra from [CCLLS21] to finish the proof. Given the lemma, we can finally (at least informally) explain *how* the branching process $(\widehat{Z}_{2a}(t), \widehat{Z}_{2d}(t))$ approximates $(N_{2a,t}, N_{2d,t})$ on $[0, T^2_{\varepsilon^\xi} \wedge T^2_0]$ on the event $\{R_{2\varepsilon} > T^2_{\varepsilon^\xi} \wedge T^2_0\}$. We define branching processes $(Z^{K,\varepsilon,-}_{2a,t}, Z^{K,\varepsilon,-}_{2d,t})$ and $(Z^{K,\varepsilon,+}_{2a,t}, Z^{K,\varepsilon,+}_{2d,t})$ on $\mathbb{N}^2_0$ depending on K and ε such that for all $\upsilon \in \{a, d\}$,

$$\begin{aligned} Z^{K,\varepsilon,-}_{2\upsilon,t} &\le \widehat{Z}_{2\upsilon}(t) \le Z^{K,\varepsilon,+}_{2\upsilon,t}, \\ Z^{K,\varepsilon,-}_{2\upsilon,t} &\le N_{2\upsilon,t} \le Z^{K,\varepsilon,+}_{2\upsilon,t}. \end{aligned} \tag{2.28}$$

That is, we do not directly compare $(N_{2a,t}, N_{2d,t})$ with $(\widehat{Z}_{2a}(t), \widehat{Z}_{2d}(t))$, but we sandwich both of them between the two auxiliary branching processes, which are also supercritical for large K and small ε. Using standard methods by [AN72] we can finish the proof of Proposition 2.10 (see [BT20, Section 4.1] for details).

Even if we do not present the full proof of Proposition 2.10 and Lemma 2.11, we would like to draw the attention to the exponent $\xi \in [1/2, 1]$ of ε in their statements. The assertion of the proposition tells that one can even guarantee that the resident population size rescaled by K stays within a neighbourhood of size at most 2ε of $\bar{n}_1$ as long as the total mutant population size is not yet extinct but still below $\sqrt{\varepsilon}K$, which is substantially larger than the bound $2\varepsilon K$ on the error term of the resident population size (and similarly for the lemma). It is not stated in the proposition but will also be important that the branching process approximation of mutants is valid on the entire time interval $[0, T^2_0 \wedge T^2_{\sqrt{\varepsilon}}]$ on the event $\{T^2_0 < T^2_{\varepsilon^\xi} \wedge R_{2\varepsilon}\}$.

Our multitype setting increases the dimension of the system ODEs to three. This makes it more difficult to show global stability properties of equilibria that are strong enough to guarantee that given a successful mutant invasion in Phase I, $\mathbf{N}^K_t$ will reach a small neighbourhood of $(0, \bar{n}_{2a}, \bar{n}_{2d})$ in Phase II. We need to find some set A of initial conditions (n_1, n_{2a}, n_{2d}) such that the following two assertions are satisfied:

(i) The solution $(n_1(t), n_{2a}(t), n_{2d}(t))_{t\ge 0}$ to (2.6) started from A converges to $(0, \bar{n}_{2a}, \bar{n}_{2d})$, further, $\varepsilon \le n_{2a} + n_{2d} \le \sqrt{\varepsilon}$, $|n_1 - \bar{n}_1| \le 2\varepsilon$, and for large K, for any $(n_1, n_{2a}, n_{2d}) \in A$, and

(ii) on the event $\{T^2_{\sqrt{\varepsilon}} < T^2_0\}$, $\mathbf{N}^K_t$ reaches A with probability tending to 1 as $K \to \infty$ followed by $\varepsilon \downarrow 0$.

The idea of [CCLLS21] is to use the Kesten–Stigum theorem for the approximating branching process to identify a set of initial conditions satisfying (i). As we will see below, this theorem guarantees that the proportion between the number of active and dormant mutants gets close to a certain deterministic, positive number at some time between T^2_ε and $T^2_{\sqrt{\varepsilon}}$ with high probability on the event $\{T^2_{\sqrt{\varepsilon}} < T^2_0\}$. Then, in order to guarantee (i), we shall show that if we have this total size and active/dormant proportion of mutants and residents are still close to equilibrium, then the dynamical system (2.6) started from a corresponding initial condition tends to $(0, \bar{n}_{2a}, \bar{n}_{2d})$ as $t \to \infty$. This convergence result was the part of the proof of [BT20] that required some novel ideas, as we will explain below.

The analysis of the third phase, where $(N_{2a,t}^K, N_{2d,t}^K)$ stays close to $(\bar{n}_{2a}, \bar{n}_{2d})$ and the former residents go extinct, is rather similar to the one of the first phase. Here, apart from [CCLLS21], we also use some proof techniques from the same authors without Laroche [CCLS18] on a stochastic individual-based model for speciation via mating preferences. The main result of [BT20] corresponding to Phase III, the proof of which we will also omit, is the following, where we recall the number $\widehat{\lambda}$ defined in (2.17).

Proposition 2.12 ([BT20], based on methods of [CCLS18])**.** *There exist $\varepsilon_0, C_0 > 0$ such that for all $\varepsilon \in (0, \varepsilon_0)$, under condition* (2.8)*, if there exists $\eta \in (0, 1/2)$ that satisfies*

$$\left|N_{2a}^K(0) - \bar{n}_{2a}\right| \le \varepsilon \quad \text{and} \quad \left|N_{2d}^K(0) - \bar{n}_{2d}\right| \le \varepsilon \quad \text{and} \quad \eta\varepsilon/2 \le N_1^K(0) \le \varepsilon/2,$$

then

$$\forall \widetilde{C} > \widehat{\lambda}^{-1} + C_0\varepsilon, \qquad \mathbb{P}(T_{S_\varepsilon} \le \widetilde{C} \log K) \underset{K\to\infty}{\longrightarrow} 1,$$
$$\forall 0 \le \widetilde{C} < \widehat{\lambda}^{-1} - C_0\varepsilon, \qquad \mathbb{P}(T_{S_\varepsilon} \le \widetilde{C} \log K) \underset{K\to\infty}{\longrightarrow} 0.$$

2.8 Multitype branching processes II: the Kesten–Stigum theorem and its application for our model

The Kesten–Stigum theorem can be found in various forms in the literature, let us now cite the variant [GB03, Theorem 2.1] that was also cited by [CCLLS21]. Let $(Z_t)_{t\ge0} = (Z_{i,t})_{i\in S, t\ge0}$ be a continuous-time supercritical branching process where the types of individuals are elements of a finite set S, and for $i \in S$ let $\mathbb{P}^i$ denote the probability measure under which $Z_0 = (0, \dots, 0, \underbrace{1}_{i\text{-th}}, 0, \dots, 0)$, i.e., at time 0 there is one single individual, which is of type i, and let $\mathbb{E}^i$ denote the expectation associated to $\mathbb{P}^i$. For $i, j \in S$, let N_{ij} denote the expected number of type j offspring of a type i individual. We say that the mean matrix J of the branching process (or any square matrix) is *irreducible* if it is not similar via a permutation to a block upper triangular matrix (i.e., there exists no permutation matrix P and block upper triangular matrix U such that $J = PUP^{-1}$). The interpretation of irreducibility here is similar to that of irreducibility for Markov chains: If the branching process is irreducible, than individuals of any type can create individuals of any other type (perhaps indirectly, i.e. using multiple parent→child steps).

As we have seen, in the supercritical case, if $T_0 = \inf\{t \ge 0\colon Z_t = 0\}$ denotes the extinction time of the process, we have $\mathbb{P}^i(T_0 < \infty) < 1$, and the mean matrix has a positive eigenvalue $\lambda > 0$. Then it follows from general Perron–Frobenius theory that J has a coordinatewise positive left eigenvalue $\pi = (\pi_i)_{i\in S}$ and a coordinatewise positive right eigenvalue $h = (h_i)_{i\in S}$ associated with λ, i.e., $\pi^T J = \lambda\pi^T$ and $Jh = \lambda h$, which are by convention normalized as

$$\sum_{i\in S} \pi_i = 1 = \sum_{i\in S} \pi_i h_i.$$

The first equality here makes it possible to interpret π as a probability distribution of S. It is related to the asymptotic proportion of types conditional on the survival of the branching process as follows.

Theorem 2.13 (Kesten–Stigum)**.** *Let us consider the supercritical case $\lambda > 0$ and assume that J is irreducible.*

(a) For all $i, k \in S$ we have

$$\frac{Z_k(t)}{\sum_{j\in S} Z_j(t)} \underset{t\to\infty}{\longrightarrow} \pi_k, \quad \mathbb{P}^i\text{-almost surely on } \{T_0 = \infty\}.$$

(b) There exists a nonnegative random variable W such that

$$\lim_{t\to\infty} Z(t)\mathrm{e}^{-\lambda t} = W\pi, \quad \mathbb{P}^i\text{-almost surely},$$

and $\mathbb{P}^i(W>0)>0$ *for all* i *if and only if*

$$\mathbb{E}(N_{ij}\log N_{ij})<\infty \text{ for all } i,j\in S. \tag{2.29}$$

In this case $\{W>0\}=\{T_0=\infty\}$ *holds* $\mathbb{P}^i$*-a.s., and* $h_i=\mathbb{E}^i(W)$.

See the references in [GB03] for a proof.

Remark 2.14. In the one-dimensional case, of course $\pi_1=h_1=1$ is the right choice. Given the proof of Theorem 1.19, it is no surprise that $t\mapsto Z(t)\mathrm{e}^{-\lambda t}$ is a nonnegative martingale. Under the condition (2.29), started from one individual, we have that its almost sure limit W has expectation 1, which is exactly $Z(0)$. This means that the martingale converges in L^1, equivalently, it is uniformly integrable. Thus, a consequence of the Kesten–Stigum theorem is that uniform integrability holds if and only if (2.29) is satisfied.

Example 2.15. In the case of our approximating branching process $(\widehat{Z}_{2a}(t),\widehat{Z}_{2d}(t))$, we have $S=\{2a,2d\}$, $\mathbb{P}(N_{2a,2a}=2,N_{2a,2d}=0)=\frac{\lambda_1}{\lambda_1+\mu+\alpha\bar{n}_1}$, $\mathbb{P}(N_{2a,2a}=0,N_{2a,2d}=0)=\frac{\mu+(1-p)\alpha\bar{n}_1}{\lambda_1+\mu+\alpha\bar{n}_1}$, $\mathbb{P}(N_{2a,2a}=0,N_{2a,2d}=1)=\frac{p\alpha\bar{n}_1}{\lambda_1+\mu+\alpha\bar{n}_1}$, $\mathbb{P}(N_{2d,2a}=0,N_{2d,2d}=0)=\frac{\kappa\mu}{\kappa\mu+\sigma}$, $\mathbb{P}(N_{2d,2a}=1,N_{2d,2d}=0)=\frac{\sigma}{\kappa\mu+\sigma}$. Since all these random variables take finitely many values, condition (2.29) is of course satisfied. In the supercritical case, the normalized coordinatewise positive left eigenvector $\pi=(\pi_{2a},\pi_{2d})$ is characterized by (with $\widetilde{\lambda}$ given as in (2.14))

$$\begin{aligned}\pi_{2a}(\lambda_2-\mu-\alpha\bar{n}_1)+\pi_{2d}\sigma&=\widetilde{\lambda}\pi_{2a},\\ \pi_{2a}p\alpha\bar{n}_1-\pi_{2d}(\kappa\mu+\sigma)&=\widetilde{\lambda}\pi_{2d},\\ \pi_{2a}+\pi_{2d}&=1.\end{aligned}$$

Proposition 2.16 ([BT20])**.** *There exists* $C>0$ *sufficiently large such that for* $\delta>0$ *such that* $\pi_{2a}\pm\delta\in(0,1)$*, under the same assumptions as Proposition 2.10,*

$$\begin{aligned}\liminf_{K\to\infty}\mathbb{P}\Big(&\exists t\in\big[T^2_\varepsilon,T^2_{\sqrt{\varepsilon}}\big],\frac{\varepsilon K}{C}\le N_{2,t}\le\sqrt{\varepsilon}K,\\ &\pi_{2a}-\delta<\frac{N_{2a,t}}{N_{2a,t}+N_{2d,t}}<\pi_{2a}+\delta\Big|T^2_{\sqrt{\varepsilon}}<T_0^2\wedge R_{2\varepsilon}\Big)\ge 1-o_\varepsilon(1).\end{aligned} \tag{2.30}$$

Note also the factor of $1/C$ in the term $\frac{\varepsilon K}{C}$: One cannot guarantee that after the total population size of mutants has reached εK, it will never drop below it, but for $C>0$ sufficiently small (and independent of ε and K) one can guarantee it with high probability as $K\to\infty$ followed by $\varepsilon\downarrow 0$.

The full proof of this proposition ([BT20, Proposition 4.4]) uses a Poissonian construction similar to the one that we saw in Section 1.7 and some ingredients of stochastic analysis for processes with jumps; it can be found in Appendix D. Let us provide an informal outline of the proof here. The proof employs many arguments from the one of [CCLLS21, Proposition 3.2]. It is based on the Kesten–Stigum theorem, but to employ that theorem for the original individual-based model (and not just the approximating branching process), one needs to use a semimartingale decomposition of the proportion $\frac{N^K_{2a,T^2_\varepsilon}}{N^K_{2a,T^2_\varepsilon}+N^K_{2d,T^2_\varepsilon}}$. This decomposition is of the form

$$\frac{N^K_{2a,T^2_\varepsilon}}{N^K_{2a,T^2_\varepsilon}+N^K_{2d,T^2_\varepsilon}}=\frac{N^K_{2a,t}}{N^K_{2a,t}+N^K_{2d,t}}+M_2(t)+V_2(t),\qquad t\ge T^2_\varepsilon,$$

where $t\mapsto M_2(t)$ is a martingale and $t\mapsto V_2(t)$ a finite-variation process, see Appendix D for the precise form of $M_2(t)$ and $V_2(t)$ as well as an informal explanation regarding how this decomposition is obtained. One first controls the predictable quadratic variation of the martingale[21] between times T^2_ε and $T^2_{\sqrt{\varepsilon}}$, which

[21] **Quadratic variation**. Let $(N_t)_{t\ge0}$ be any square-integrable martingale (in continuous time). Then,

$$[N]_t=\lim_{n\to\infty}\sum_{k\le n-1}\Big(N_{t\frac{k+1}{n}}-N_{t\frac{k}{n}}\Big)^2$$

guarantees that with high probability, the martingale only fluctuates by at most ε on these time intervals. On the other hand, the finite-variation process is deterministic and for large K close to the solution of a one-dimensional ODE, whose unique equilibrium is precisely given by π_{2a}. From this we can derive that with high probability (as $K \to \infty$ followed by $\varepsilon \downarrow 0$), the proportion of active individuals will enter $[\pi_{2a}-\delta, \pi_{2a}+\delta]$.

2.9 Convergence of the dynamical system

A particularly useful result in the context of the qualitative behaviour of *two-dimensional* autonomous systems of ODEs is the *Bendixson criterion* (see e.g. [DLA06, Theorem 7.10]), which tells that if the divergence of a two-dimensional autonomous system of ODEs has constant and nonzero sign on a simply connected domain of $\mathbb{R}^2$, then on that domain the system has no periodic orbit. (If the system is of the form

$$\begin{aligned} \dot{x}(t) &= f(x(t), y(t)), \\ \dot{y}(t) &= g(x(t), y(t)), \end{aligned}$$

then its divergence at $(x, y) \in \mathbb{R}^2$ is given as $\frac{\partial f}{\partial x}(x, y) + \frac{\partial g}{\partial y}(x, y)$, and for the assertion to hold, one has to assume that $f, g \colon \mathbb{R}^2 \to \mathbb{R}$ are totally differentiable.) It is also a classical result that solutions to any two-dimensional autonomous systems of ODEs will always converge to $\pm\infty$, to an equilibrium or to a periodic orbit as $t \to \infty$. On the other hand, solutions to three-dimensional ones can already show chaotic behaviour. Therefore, before attacking the system (2.6) directly, it may be a good idea to learn about the global qualitative behaviour of the sub-system

$$\begin{aligned} \dot{n}_{2a}(t) &= n_{2a}(t)(\lambda_2 - \mu - \alpha n_{2a}(t)) + \sigma n_{2d}(t), \\ \dot{n}_{2d}(t) &= p\alpha n_{2a}^2(t) - (\kappa\mu + \sigma) n_{2d}(t), \end{aligned} \tag{2.31}$$

corresponding to types 2a and 2d, which we introduced in (2.2). Let us recall that this system has an asymptotically stable equilibrium $(\bar{n}_{2a}, \bar{n}_{2d})$ and an unstable one $(0, 0)$ under the assumption that $\lambda_2 > \mu$.

Lemma 2.17 ([BT20])**.** *In case* $(n_{2a}(0), n_{2d}(0)) \in [0, \infty)^2 \setminus \{(0, 0)\}$*, we have*

$$\lim_{t\to\infty} (n_{2a}(t), n_{2d}(t)) = (\bar{n}_{2a}, \bar{n}_{2d}).$$

Proof. Observe that the active coordinate of the stable equilibrium,

$$\bar{n}_{2a} = \frac{(\lambda_2 - \mu)(\kappa\mu + \sigma)}{\alpha(\kappa\mu + (1-p)\sigma)} > 0$$

satisfies

$$\frac{\lambda_2 - \mu}{\alpha} < \bar{n}_{2a} \le \frac{\lambda_2 - \mu}{(1-p)\alpha}, \tag{2.32}$$

where the second inequality is an equality if and only if $\kappa = 0$. Further, the dormant coordinate $\bar{n}_{2d}$ is positive. Note further that the divergence of the system is given as

$$\lambda_2 - \mu - 2\alpha n_{2a}(t) - (\kappa\mu + \sigma).$$

This is certainly negative if $n_{2a} \ge \frac{\lambda_2 - \mu}{2\alpha}$, $n_{2d} \ge 0$, and at least one of the latter two inequalities is strict. In particular, the Bendixson criterion implies that there is no nontrivial periodic solution in the open and simply connected set

$$U = \big\{(n_{2a}, n_{2d}) \in \mathbb{R}^2 \colon n_{2a} > \tfrac{\lambda_2 - \mu}{2\alpha}, n_{2d} > 0\big\}.$$

is called the *quadratic variation* of $(N_t)_{t\ge0}$. On the other hand, $\langle N_t \rangle$, the *preditable quadratic variation* is defined as the unique process that is increasing and predictable (i.e. adapted to the filtration generated by the left-continuous processes) and such that $(N_t^2 - \langle N \rangle_t)_{t\ge0}$ is a martingale. (Its existence follows from the Doob–Meyer decomposition.) If the martingale $(N_t)_{t\ge0}$ is continuous, then so is $([N]_t)_{t\ge0}$, which is of course also adapted to the same filtration as the martingale itself, so that it is also predictable. In this case, we have $\langle N \rangle_t = [N]_t$. This footnote originates from Nicolas Perkowski's lecture "Interacting particles and stochastic PDEs" at HU Berlin in 2017/18.

Since this is a two-dimensional system and all solutions of the system with coordinatewise nonnegative initial conditions are bounded, this implies that any solution starting from U converges to the equilibrium $(\bar{n}_{2a}, \bar{n}_{2d}) \in U$. It remains to show that any solution started from $[0,\infty)^2 \setminus (\{(0,0)\} \cup U)$ will enter the open set U after finite time.

Now, observe that if $n_{2a}(0) > 0$ and $n_{2d}(0) \geq 0$, then $\dot{n}_{2a}$ is positive and bounded away from zero until n_{2a} reaches $\frac{\lambda_2-\mu}{2\alpha}$, hence n_{2a} will reach this level. If $n_{2d}(0) > 0$ and $n_{2a}(0) = 0$, then there exists $\delta > 0$ such that $n_{2a}(\delta) > 0$ and $n_{2d}(\delta) > 0$, and hence n_{2a} will also reach the level $\frac{\lambda_2-\mu}{2\alpha}$ in finite time. Further, for $t > 0$, if $n_{2a}(t) = \frac{\lambda_2-\mu}{2\alpha}$ and $n_{2d}(t) \geq 0$, then plugging in the first inequality of (2.32) to the first equation of (2.31) implies that $\dot{n}_{2a}(t) > 0$. This implies that if $n_{2d}(t) > 0$, then

$$(n_{2a}(t+\varepsilon), n_{2d}(t+\varepsilon)) \in U, \qquad \forall \varepsilon > 0 \text{ sufficiently small.} \tag{2.33}$$

Else, $\dot{n}_{2a}(t) = 0$ but $\dot{n}_{2d}(t) > 0$, and hence the observations of the previous case imply that $\dot{n}_{2a}(t+\varepsilon) > 0$ for all sufficiently small $\varepsilon > 0$, thus (2.33) also holds. □

Now, we show convergence of the original 3-dimensional system to $(0, \bar{n}_{2a}, \bar{n}_{2d})$ as $t \to \infty$ for initial conditions corresponding to Proposition 2.16. In other words, we verify some global attractor properties of this equilibrium, which are not as general as for the two-dimensional system but sufficient for the present invasion analysis.

Lemma 2.18 ([BT20])**.** *Let us consider the system of ODEs* (2.6)*. If the initial condition* $(n_1, n_{2a}, n_{2d}) = (n_1(0), n_{2a}(0), n_{2d}(0))$ *satisfies*

$$\frac{p\alpha(n_1+n_{2a})}{\kappa\mu+\sigma} > \frac{n_{2d}}{n_{2a}} > \frac{\mu-\lambda_2+\alpha(n_1+n_{2a})}{\sigma}, \qquad n_1 \geq 0, n_{2a}, n_{2d} > 0, \tag{2.34}$$

then

$$\lim_{t\to\infty} (n_1(t), n_{2a}(t), n_{2d}(t)) = (0, \bar{n}_{2a}, \bar{n}_{2d}). \tag{2.35}$$

Before proving this lemma, let us mention how it corresponds to Proposition 2.16.

Lemma 2.19 ([BT20], based on methods of [CCLLS21])**.** *Let C be chosen according to Proposition 2.16, further, $n_1, n_{2a}, n_{2d} > 0$ such that $n_1 \in (\bar{n}_1 - 2\varepsilon, \bar{n}_1 + 2\varepsilon)$, $n_{2a} + n_{2d} \in (\varepsilon/C, \sqrt{\varepsilon})$, and $\frac{n_{2d}}{n_{2a}} = \frac{\pi_{2d}}{\pi_{2a}}$. Then, if $\varepsilon > 0$ is sufficiently small, then (n_1, n_{2a}, n_{2d}) satisfies* (2.34)*.*

The proof of Lemma 2.19 is elementary and therefore we only present it in Appendix C. Let us now proceed with the proof of Lemma 2.18.

Proof of Lemma 2.18. Let us assume that for some $t \geq 0$, $(n_1(t), n_{2a}(t), n_{2d}(t)) = (n_1, n_{2a}, n_{2d})$. Then the first inequality in (2.34) is equivalent to the statement that $\dot{n}_{2d}(t) > 0$ and the second one is equivalent to the statement that $\dot{n}_{2a}(t) > 0$. Hence, as long as (2.34) holds, $t \mapsto n_{2a}(t)$ and $t \mapsto n_{2d}(t)$ are strictly increasing.

Let us assume that condition (2.34) holds for $(n_1, n_{2a}, n_{2d}) = (n_1(0), n_{2a}(0), n_{2d}(0))$. We claim that then it also holds for all $t > 0$ with $(n_1, n_{2a}, n_{2d}) = (n_1(t), n_{2a}(t), n_{2d}(t))$, unless eventually $n_{2a}(t) = \bar{n}_{2a}$ and $n_{2d}(t) = \bar{n}_{2d}$. Indeed, let us assume that for some $t > 0$, $(n_1(t), n_{2a}(t), n_{2d}(t))$ lies on the boundary of the set

$$G = \{(n_1, n_{2a}, n_{2d}) \in [0,\infty) \times (0,\infty) \times (0,\infty) \colon (n_1, n_{2a}, n_{2d}) \text{ satisfies (2.34)}\} \tag{2.36}$$

with $n_{2a}, n_{2d} \geq 0$, in such a way that $(n_1(s), n_{2a}(s), n_{2d}(s))$ is contained in the set G for all $0 \leq s < t$. Then $n_{2a}(t), n_{2d}(t) > 0$ holds because $n_{2a}, n_{2d} > 0$ by assumption, moreover, $s \mapsto n_{2a}(s)$ and $s \mapsto n_{2d}(s)$ are increasing on $[0,t)$. Hence, one of the following conditions holds:

(i) $\dot{n}_{2d}(t) = 0$, $\dot{n}_{2a}(t) > 0$,

(ii) $\dot{n}_{2a}(t) = 0$, $\dot{n}_{2d}(t) > 0$,

(iii) $\dot{n}_{2a}(t) = \dot{n}_{2d}(t) = 0$.

In case (i) we have

$$\dot{\left(\frac{n_{2d}}{n_{2a}}\right)}(t) = \frac{-\dot{n}_{2a}(t)n_{2d}(t)}{n_{2a}(t)^2} < 0.$$

The case (ii) yields

$$\dot{\left(\frac{n_{2d}}{n_{2a}}\right)}(t) = \frac{\dot{n}_{2d}(t)n_{2a}(t)}{n_{2a}(t)^2} > 0.$$

In case (iii) we have (thanks to the condition that $n_{2a}, n_{2d} > 0$) that $(n_{2a}, n_{2d}) = (\bar{n}_{2a}, \bar{n}_{2d})$. We conclude that if $(n_1, n_{2a}, n_{2d}) = (n_1(0), n_{2a}(0), n_{2d}(0))$ satisfies (2.34), then $t \mapsto (n_1(t), n_{2a}(t), n_{2d}(t))$ never enters the complement of the closure of the set G apart from $(\bar{n}_{2a}, \bar{n}_{2d})$, which implies the claim.

Now, given that condition (2.34) holds for $(n_1, n_{2a}, n_{2d}) = (n_1(0), n_{2a}(0), n_{2d}(0))$, $t \mapsto n_{2a}(t)$ and $t \mapsto n_{2d}(t)$ are nonnegative, bounded, increasing, and strictly increasing unless $(n_{2a}(t), n_{2d}(t)) = (\bar{n}_{2a}, \bar{n}_{2d})$ eventually, in which case both coordinates would immediately become constant. Further, $t \mapsto n_1(t)$ is also bounded and nonnegative. Hence, $(n_1(t), n_{2a}(t), n_{2d}(t))$ converges along a subsequence to $(n_1^*, \bar{n}_{2a}, \bar{n}_{2d})$ for some $n_1^* \geq 0$. Now we argue that n_1^* must be equal to zero. Indeed, taking limits of (2.34) implies that

$$\frac{p\alpha(n_1^* + \bar{n}_{2a})}{\kappa\mu + \sigma} \geq \frac{\bar{x}_d}{\bar{x}_a} \geq \frac{\mu - \lambda_2 + \alpha(n_1^* + \bar{n}_{2a})}{\sigma}. \tag{2.37}$$

Observe that (2.37) holds for $n_1^* = 0$ thanks to (2.5). Taking this into account, any subsequential limit has to satisfy

$$\frac{p\alpha n_1^*}{\kappa\mu + \sigma} \geq \frac{\alpha n_1^*}{\sigma}.$$

Since by our assumptions, $\frac{p}{\kappa\mu+\sigma} < \frac{1}{\sigma}$, we conclude that $n_1^* = 0$. Hence, (2.35) follows. □

3 Example 2: the Beretta–Kuang host–virus model extended with recovery and dormancy

In this section we will discuss another invasion model with an interesting biological motivation, namely a host–virus model, also related to (a different kind of) dormancy. The underlying dynamical system has features that are rather different from the properties of the system (2.6), which give a good opportunity to discuss some further methods of stability theory and a well-known type of bifurcations during this course. However, the qualitative behaviour of this system is tedious to investigate analytically, and many related questions are still open.

3.1 The Beretta–Kuang host–virus model (with recovery, without dormancy)

The dynamical system corresponding to the three-dimensional, dormancy-free base variant of the model was introduced by Beretta and Kuang [BK98]. They did not study its stochastic version, which we will explain below (and which is derived analogously to the previous sections). We introduced this individual-based model as well as its full, four-dimensional version with contact-mediated host dormancy in [BT23]. Our agenda for Section 3 starts with the presentation of the individual-based model scaling to the dynamical system introduced by Beretta and Kuang, and to explain some fundamental results on the qualitative behaviour of this dynamical system as well as heuristics on its branching process counterpart. The full four-dimensional dynamical system is more difficult to study and we only have partial results with regard to its behaviour; this is one motivation to start with the dormancy-free model, another is that this way we can investigate the effect of dormancy compared to the original model. The description of the full model starts in Section 3.5, and the main results of [BT23] are presented in Section 3.7.

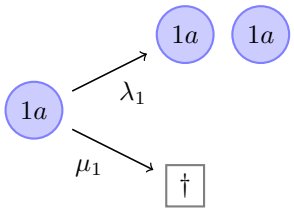
In the dormancy-free model, there is a *host type* (type 1) and a *virus type* (type 2). In absence of viruses, type 1 only appears in its active form (type 1a), featuring binary reproduction and logistic competition,

just as in the case of the model of [C06] with mutations ignored. Type 1 can be thought of as a one-cell microorganism featuring asexual and haploid (clonal) reproduction. However, type 1a individuals are susceptible to an infection by a lytic virus of type 2. If a host meets a free virus particle (also called *virion*), it will become infected (type 1i) and the virus will disappear (becoming part of the infected individual). Infected hosts do not reproduce and do not feel competitive pressure, but they exert competitive pressure on the active cells, and they will eventually either recover (i.e., become active again), or they will become a virus factory, eventually ejecting a fixed number $m \in \mathbb{N}$ of new virus particles and dying. Such a kind of virus reproduction is called *lytic*. Finally, virus particles cannot reproduce on their own, but they "die" (degrade) at a certain rate.[22]

Informally speaking, the definition of the model is the following.

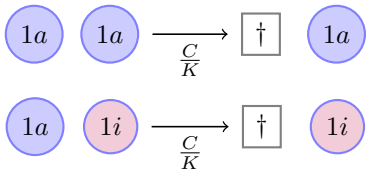
(i) An active (type 1a) individual gives birth to another such individual at rate $\lambda_1 > 0$.

(ii) A type 1a individual has a natural death rate $\mu_1 \in (0, \lambda_2)$.

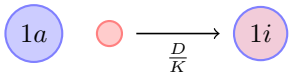


(iii) $K > 0$ is the carrying capacity of the population.

(iv) For some $C > 0$, for any ordered pair consisting of one active (1a) host cell and one other host cell (of either type 1a or 1i), at rate C/K, a death due to competition/overcrowding happens, affecting the first active individual, which is removed from the population.[23]



(v) For any ordered pair of individuals containing one active (type 1a) cell and one virion (type 2), a virus attack happens at rate D/K. In this case, the host cell gets infected (i.e. switches from 1a to 1i) and the free virus (2) is 'removed' (in the sense that it enters the cell).

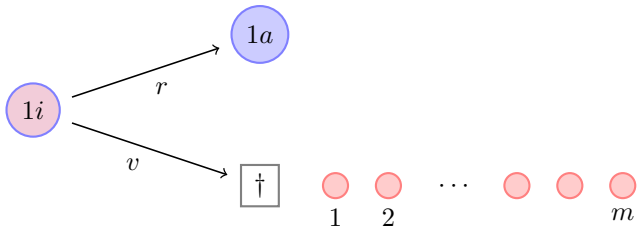


(vi) An infected (type 1i) individual recovers (i.e. switches back from 1i to 1a) at rate $r \geq 0$.

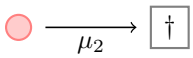
(vii) An infected individual produces $m \in \mathbb{N}$ new virions and then gets removed (lysis), at rate $v > 0$. The parameter m is called the *burst size* (a realistic value of m to imagine is several dozens or at most a few hundreds).

[22] We will sometimes also call type 2 particles "individuals" for convenience, even though this is biologically not entirely correct.

[23] Note that the competition parameter is now called C. Regarding the infected individuals, the above modelling choice originates from [BK98, Section 1], where the authors argue that this is a reasonable assumption because the mortality of infected individuals is almost completely due to lysis. (In contrast, if one considers chronically infected cells, their lifespan is typically much longer than the one of lytically infected ones, and hence the competitive pressure that they feel is not negligible, cf. [GW18].)



(viii) Virions (type 2) do not reproduce individually (but instead indirectly via infection of a host cell, see below) and die/degrade at rate $\mu_2 > 0$.



The corresponding population process is formally defined as a continuous time Markov chain $\mathbf{N} = (\mathbf{N}_t)_{t\geq 0}$ on $\mathbb{N}_0^4$, where

$$(\mathbf{N}_t)_{t\geq 0} = (N_{1a,t}, N_{1i,t}, N_{2,t})_{t\geq 0} \tag{3.1}$$

is interpreted as

$$N_{x,t} = \#\{\text{individuals of type } x_i \text{ alive at time } t\}, \tag{3.2}$$

for $x_i \in \{1a, 1i, 2\}$. According to the above description, $\mathbf{N}$ is then the unique Markov process with transitions

$$(n_{1a}, n_{1i}, n_2) \to \begin{cases} (n_{1a}+1, n_{1i}, n_2) \text{ at rate } \lambda_1 n_{1a}, \\ (n_{1a}-1, n_{1i}, n_2) \text{ at rate } (\mu_1 + C\frac{n_{1a}+n_{1i}}{K})n_{1a}, \\ (n_{1a}-1, n_{1i}+1, n_2-1) \text{ at rate } \frac{Dn_{1a}n_2}{K}, \\ (n_{1a}+1, n_{1i}-1, n_2) \text{ at rate } rn_{1i}, \\ (n_{1a}, n_{1i}-1, n_2+m) \text{ at rate } vn_{1i}, \\ (n_{1a}, n_{1i}, n_2-1) \text{ at rate } \mu_2 n_2. \end{cases}$$

Its only absorbing state is $(0,0,0)$, which corresponds to the extinction of all the three types. The underlying dynamical system is now easily seen to be

$$\begin{aligned} \frac{\mathrm{d}n_{1a}(t)}{\mathrm{d}t} &= n_{1a}(t)\big(\lambda_1 - \mu_1 - C(n_{1a}(t)+n_{1i}(t)) - Dn_2(t)\big) + rn_{1i}(t), \\ \frac{\mathrm{d}n_{1i}(t)}{\mathrm{d}t} &= Dn_{1a}(t)n_2(t) - (r+v)n_{1i}(t), \\ \frac{\mathrm{d}n_2(t)}{\mathrm{d}t} &= mvn_{1i}(t) - Dn_{1a}(t)n_2(t) - \mu_2 n_2(t). \end{aligned} \tag{3.3}$$

The positive orthant of $\mathbb{R}^3$ is invariant under this system. Note that in [BK98], recovery was absent (the authors studied a "microbial virus epidemic with a mortality rate of 100%" [24]), and the notation was different, especially regarding the logistic competition. The qualitative differences between the cases $r = 0$ and $r > 0$ that we will mention below originate from [BT23].

[24] Note that there are some epidemics in nature that are nearly always fatal, e.g. untreated rabies in humans or dogs, or certain variants of the African swine pest in swine. Also, the relevance of the case $r > 0$ is not entirely clear because while e.g. humans can recover from influenza although some of their cells have died, it is not entirely clear if single cells can also survive after having been infected, and hence the recovery of one-cell individuals is debatable.

3.2 The dynamical system I: stability of simple equilibria and existence of a coexistence equilibrium

$(0,0,0)$ is clearly an equilibrium of the system (3.3), under which the positive orthant is again invariant, and since we have assumed that $\lambda_1 > \mu_1$, $\bar{n}_{1a} := \frac{\lambda_1 - \mu_1}{C}$ is also an equilibrium. At $(0,0,0)$ we have the Jacobi matrix

$$A(0,0,0) = \begin{pmatrix} \lambda_1 - \mu_1 & r & 0 \\ 0 & -(r+v) & 0 \\ 0 & mv & -\mu_2 \end{pmatrix}.$$

The eigenvalues of this matrix are its diagonal entries, and since $\lambda_1 - \mu > 0$, it follows that $(0,0,0)$ is always unstable.

On the other hand, at $(\bar{n}_{1a}, 0, 0)$, the Jacobi matrix is given as follows

$$A(\bar{n}_{1a}, 0, 0) = \begin{pmatrix} -(\lambda_1 - \mu_1) & r & -D\bar{n}_{1a} \\ 0 & -(r+v) & D\bar{n}_{1a} \\ 0 & mv & -\mu_2 - D\bar{n}_{1a} \end{pmatrix} \tag{3.4}$$

We see that $-(\lambda_1 - \mu_1) < 0$ is an eigenvalue of $A(\bar{n}_{1a}, 0, 0)$ with eigenvector $(1,0,0)^T$, and the remaining two eigenvalues are the eigenvalues of the two eigenvalues of the last 2×2 block of the matrix. Since the trace of this block is negative, at least one eigenvalue has negative real part, and thus if there is an eigenvalue with positive real part, then both eigenvalues must be real. This way, $A(\bar{n}_{1a}, 0, 0)$ is asymptotically stable if and only if the determinant of this block is positive. Whenever $mv > r + v$, this condition is equivalent to

$$\bar{n}_{1a} < \frac{\mu_2(r+v)}{D(mv - (r+v))}, \tag{3.5}$$

and unstable if and only if this determinant is negative, i.e.,

$$\bar{n}_{1a} > \frac{\mu_2(r+v)}{D(mv - (r+v))}. \tag{3.6}$$

Before we give a biological interpretation for condition (3.6), let us point out that the following.

Lemma 3.1 ([BK98] for $r = 0$, [BT23] for $r > 0$)**.** *Condition* (3.6) *together with the condition that* $mv > r + v$ *is equivalent to the existence of a coordinatewise positive equilibrium of the system* (3.3)*. If such an equilibrium* $(\widetilde{n}_{1a}, \widetilde{n}_{1i}, \widetilde{n}_2)$ *exists, it is also unique, and its active coordinate is given by*

$$\widetilde{n}_{1a} = \frac{\mu_2(r+v)}{D(mv - (r+v))}. \tag{3.7}$$

If $mv \leq r + v$*, then there is no coordinatewise positive equilibrium.*

Proof. A coexistence equilibrium (as in the lemma) needs to satisfy

$$\widetilde{n}_{1i} = \frac{D\widetilde{n}_{1a}\widetilde{n}_2}{r+v} \tag{3.8}$$

and

$$\widetilde{n}_{1i} = \frac{D\widetilde{n}_{1a}\widetilde{n}_2 - \mu_2\widetilde{n}_2}{mv}.$$

This implies that we must have

$$\frac{D\widetilde{n}_{1a}}{r+v} = \frac{D\widetilde{n}_{1a} - \mu_2}{mv}, \tag{3.9}$$

in other words,

$$\widetilde{n}_{1a} = \frac{\mu_2(r+v)}{D(mv - (r+v))}$$

(which is the expression in (3.7)), provided that $mv > r + v$. The right-hand side equals the active (type 1a) coordinate of *any* coexistence equilibrium. It is clear from (3.9) that for $mv \le r + v$ there can be no coordinatewise positive equilibrium. If $mv > r + v$, then since $\widetilde{n}_{1a} > 0$, by (3.8), $\widetilde{n}_{1i}$ and $\widetilde{n}_2$ have the same sign. Using also the first equation of (3.3), we obtain

$$\frac{\widetilde{n}_{1i}}{\widetilde{n}_{1a}} = \frac{-(\lambda_1 - \mu_1 - C(\widetilde{n}_{1a} + \widetilde{n}_{1i}) - D\widetilde{n}_2)}{r} = \frac{D\widetilde{n}_2}{r + v}.$$

We see that if $\widetilde{n}_{1a} > \frac{\lambda_1 - \mu_1}{C} = \bar{n}_{1a}$, i.e., if the determinant of $A(\bar{n}_1, 0, 0)$ is nonpositive, then $\widetilde{n}_{1i} \le 0$ and therefore there can be no coexistence equilibrium. On the other hand, if $\widetilde{n}_{1a} < \bar{n}_{1a}$, then since by the first equation of (3.3) we have

$$\widetilde{n}_{1a}(\lambda_1 - \mu_1 - C(\widetilde{n}_{1a} + \widetilde{n}_{1i}) - D\widetilde{n}_2) + r\widetilde{n}_{1i} = 0,$$

using also (3.8) we obtain

$$\lambda_1 - \mu_1 - C(\widetilde{n}_{1a} + \widetilde{n}_{1i}) - (r + v)\frac{\widetilde{n}_{1i}}{\widetilde{n}_{1a}} + r\frac{\widetilde{n}_{1i}}{\widetilde{n}_{1a}} = 0.$$

That is,

$$\lambda_1 - \mu_1 - C(\widetilde{n}_{1a} + \widetilde{n}_{1i}) = v\frac{\widetilde{n}_{1i}}{\widetilde{n}_{1a}}.$$

Now, if the left-hand side was negative, this would imply that $\widetilde{n}_{1i} > 0$, but then the equality could not be true. Hence, it must be the case that the right-hand side is positive, which implies that $\widetilde{n}_{1i} > 0$, and therefore $\widetilde{n}_2$ is also positive thanks to (3.8). (These two coordinates can be expressed with the help of the model parameters explicitly, but these expressions are rather involved, and therefore we omit them.) □

Remark 3.2. Note that the last paragraph of the proof implies that if (3.6) holds, i.e., $\widetilde{n}_{1a} < \bar{n}_{1a}$, then we even have $\widetilde{n}_{1a} + \widetilde{n}_{1i} < \bar{n}_{1a}$. This can be interpreted as follows: The coexistence with viruses reduces the total host population, even if we take infected hosts into account. Intuitively speaking, the reason for this is that the hosts cannot fully invest in their own reproduction but they are forced to use part of their energy to produce viruses. We have seen that for $mv \le r + v$ there is no coexistence equilibrium. Heuristically, this condition means that the "net growth rate" of viruses is negative due to inefficient lytic reproduction: Each virus attack leads to the loss of one virus, and the host who gets infected during this attack will produce m viruses with probability $\frac{v}{r+v}$ and 0 viruses otherwise. Hence, the net increase in the number of viruses due to this attack is on average $m\frac{v}{r+v} - 1$, which is positive if and only if $mv > r + v$.

Note also that if all parameters but λ_1, μ_1, C are fixed in such a way that $mv > r + v$, then one can always make $\bar{n}_{1a}$ so large (via choosing λ_1, μ_1, C suitably) that (3.6) becomes true. This can be interpreted as follows: More host individuals yield more host–virus contacts and hence a higher danger of outbreak of a large virus epidemic.

3.3 The branching process counterpart of the previous section

Similarly to Proposition 1.8, the dynamical system (3.3) describes the limit of the three-coordinate population size process rescaled by K on compact time intervals. We have seen that the instability of the equilibrium $(\bar{n}_{1a}, 0, 0)$ is equivalent to the existence of the coordinatewise positive equilibrium $(\widetilde{n}_{1a}, \widetilde{n}_{1i}, \widetilde{n}_2)$, and also to condition (3.6). By now, given the results of Sections 1 and 2, it is not surprising that this is also equivalent to the supercriticality of the two-type branching process that approximates the type 1i and 2 population during the first phase of the invasion. The initial condition here consists of $\approx K\bar{n}_{1a}$ type 1a individuals, one type 2 individual, and no type 1i individuals. That is, we start with a large active and susceptible host population at equilibrium, and we would like to see if one single virus can initiate a macroscopic virus epidemic with asymptotically positive probability, and if yes, with what probability this happens and how much time it takes asymptotically, and how the system will behave after a successful virus invasion. (We could also start with one type 1i individual and no type 2 ones, cf. Exercise 13 below).

Similarly to the branching process approximation in the previous sections, ignoring the precise details of couplings, the idea is that the rescaled type 1a population size $N_{1a,t}/K$ stays close to $\bar{n}_{1a}$ with high probability as long as $(N_{1i,t}, N_{2,t})$ reaches εK for some small $\varepsilon > 0$ or 0, and hence, considering $N_{1a,t}/K$ as constant equal to $\bar{n}_{1a}$, $(N_{1i,t}, N_{2,t})_{t\geq 0}$ can be approximated by the two-type branching process $(Z_{1i,t}, Z_{2,t})_{t\geq 0}$ with transitions

$$(n_{1i}, n_2) \to \begin{cases} (n_{1i}+1, n_2-1) & \text{at rate } D\bar{n}_{1a}n_2, \\ (n_{1i}-1, n_2) & \text{at rate } rn_{1i}, \\ (n_{1i}-1, n_2+m) & \text{at rate } vn_{1i}, \\ (n_{1i}, n_2-1) & \text{at rate } \mu_2 n_2 \end{cases}$$

and with the same initial condition. This branching process has mean matrix

$$J = \begin{pmatrix} -(r+v) & mv \\ D\bar{n}_{1a} & -D\bar{n}_{1a} - \mu_2 \end{pmatrix}. \tag{3.10}$$

This matrix has at least one eigenvalue with negative real part since its trace is negative. Thus, the matrix has a positive eigenvalue if and only if its determinant is negative, which is equivalent to the condition (3.6), as wanted. Again, the mean matrix J equals the transpose of the last 2×2 block of the Jacobi matrix $A(\bar{n}_{1a}, 0, 0)$.

Remark 3.3. From the point of view of the sub- or supercriticality of the branching process, the burst size (number of viruses ejected at a lysis event) need not be constant equal to m, we could also have i.i.d. random burst sizes (independent of everything else in the process) with expectation m and still obtain the same mean matrix. This would yield a multitype branching process with more possible kinds of transitions, and it is easy to imagine that all results we list in these lecture notes about the virus model (also with dormancy) hold in this case as well, but we do not want to spell out any details.

We will focus on other key quantities (largest eigenvalue of the mean matrix, extinction probability etc.) in Section 3.5, after introducing the full model with dormancy (there, the dormancy-free model corresponds to the degenerate case $q = 0$, to which the results also apply after forgetting the dormant coordinate). Right now, we will return to the study of the dynamical system, which is substantially easier in the case without dormancy (due to the lack of a fourth dimension, as already mentioned), having the branching process counterpart of the deterministic system (3.3) in mind.

3.4 The dynamical system II: Hopf bifurcations, the effect of recovery, and the paradox of enrichment

In what follows, we will fix all model parameters but the burst size m, which we keep varying as a *bifurcation parameter*. (We will see below why we call it like that.)[25]

If m is very small, namely, $mv < r + v$, then $(\widetilde{n}_{1a}, \widetilde{n}_{1i}, \widetilde{n}_2)$ does not exist as a coordinatewise positive equilibrium of (3.3) while $(\bar{n}_{1a}, 0, 0)$ is not only locally asymptotically stable but it satisfies some global attractive properties (the case where this is easiest to see is $r = 0$, see Section 3.6 for details).

Let us denote by m^* the value of m such that (3.6) holds with an equality. For $m = m^*$, $(\bar{n}_{1a}, 0, 0)$ formally equals $(\widetilde{n}_{1a}, \widetilde{n}_{1i}, \widetilde{n}_2)$, and for $m > m^*$, $(\widetilde{n}_{1a}, \widetilde{n}_{1i}, \widetilde{n}_2)$ is coordinatewise positive (while it has a negative coordinate for $m < m^*$). For $m > m^*$, $(\bar{n}_{1a}, 0, 0)$ is not just not locally asymptotically stable but repelling in a stronger sense, see Section 3.6 below. Further, we have the following two lemmas.

Lemma 3.4 ([BK98])**.** *Under condition* (3.6)*, the Jacobi matrix*

$$A(\widetilde{n}_{1a}, \widetilde{n}_{1i}, \widetilde{n}_2) = \begin{pmatrix} \lambda_1 - \mu_1 - 2C\widetilde{n}_{1a} - \widetilde{n}_{1i} - D\widetilde{n}_2 & r & -D\widetilde{n}_{1a} \\ D\widetilde{n}_2 & -(r+v) & D\widetilde{n}_{1a} \\ -D\widetilde{n}_2 & mv & -D\widetilde{n}_{1a} - \mu_2. \end{pmatrix}$$

of (3.3) *has negative determinant and negative trace.*

[25] Most results in this section originate from [BK98], but since they only treated the case $r = 0$ and the scaling of parameters was a bit different there, we will often use somewhat different proofs here, which partially come from [BT23].

Proof. The last 2×2 block of $A(\widetilde{n}_{1a},\widetilde{n}_{1i},\widetilde{n}_2)$ has zero determinant thanks to the definition of $\widetilde{n}_{1a}$ (cf. (3.7)). Hence, the determinant equals

$$-rD^2\widetilde{n}_{1a}\widetilde{n}_2+rD\widetilde{n}_2(D\widetilde{n}_{1a}+\mu_2)-D^2mv\widetilde{n}_{1a}\widetilde{n}_2+D^2\widetilde{n}_{1a}\widetilde{n}_2(r+v)=rD\widetilde{n}_2\mu_2+D^2(r+v-mv)\widetilde{n}_{1a}\widetilde{n}_2$$
$$=rD\widetilde{n}_2\mu_2-D(r+v)\widetilde{n}_2\mu_2=-\mu_2v\widetilde{n}_2. \quad (3.11)$$

This implies that the determinant is negative.

As for the trace, it suffices to show that the first diagonal entry of the matrix is negative. Since $(\widetilde{n}_{1a},\widetilde{n}_{1i},\widetilde{n}_2)$ is an equilibrium of (3.3) with three positive coordinates, we have

$$\lambda_1-\mu_1-C\widetilde{n}_{1a}-C\widetilde{n}_{1i}-D\widetilde{n}_2=-r\frac{\widetilde{n}_{1i}}{\widetilde{n}_{1a}},$$

so that

$$\lambda_1-\mu_1-2C\widetilde{n}_{1a}-C\widetilde{n}_{1i}-D\widetilde{n}_2<-r\frac{\widetilde{n}_{1i}}{\widetilde{n}_{1a}}\le 0.$$

We conclude the lemma. □

Lemma 3.5 ([BK98])**.** *For $m^*>m$ sufficiently close to m, $(\widetilde{n}_{1a},\widetilde{n}_{1i},\widetilde{n}_2)$ is locally asymptotically stable.*

Proof. In the extreme case $m=m^*$ when $A(\widetilde{n}_{1a},\widetilde{n}_{1i},\widetilde{n}_2)=A(\bar{n}_{1a},0,0)$, $\lambda_1-\mu_1-C\widetilde{n}_{1a}=\lambda_1-\mu_1-C\bar{n}_{1a}=-(\lambda_1-\mu_1)$. This number is negative and an eigenvalue of the matrix (cf. (3.4) and the paragraph thereafter). For $m=m^*$, the determinant of the last 2×2 block of $A(\bar{n}_{1a},0,0)=A(\widetilde{n}_{1a},\widetilde{n}_{1i},\widetilde{n}_2)$ has zero determinant and negative trace, meaning that its eigenvalues are 0 and a negative real number.

Now, as $m\downarrow m^*$, by continuity, all eigenvalues of the Jacobi matrix tend to those corresponding to $m=m^*$. The two eigenvalues that are negative and real for $m=m^*$ must therefore also have negative real parts for $m>m^*$ sufficiently close to m^*. Thus, in order to prove the lemma, the last thing to exclude is that the real part of the third eigenvalue tends to 0 from above as $m\downarrow m^*$. If that was the case, then this eigenvalue would be real for $m>m^*$ sufficiently close to m^* (since the other two eigenvalues have negative real parts). But then, the determinant of $A(\widetilde{n}_{1a},\widetilde{n}_{1i},\widetilde{n}_2)$ would be positive, which is impossible thanks to Lemma 3.4. □

Exercise 11. *Using arguments of the proof of Lemma 3.5, show that apart from the special choice of parameters when the two negative eigenvalues of the Jacobi matrix $A(\widetilde{n}_{1a},\widetilde{n}_{1i},\widetilde{n}_2)$ coincide for $m=m^*$, for $m>m^*$ sufficiently close to m^* the three eigenvalues have different real parts and must therefore be real.*

The value m^* is called the *transcritical bifurcation point*. In general, during a transcritical bifurcation, a new stable equilibrium branches out of another equilibrium that stays present but loses its stability at this point.

The crucial question is now whether $(\widetilde{n}_{1a},\widetilde{n}_{1i},\widetilde{n}_2)$ preserves its stability for all $m>m^*$. If we write the characteristic equation of a 3×3 matrix B in variable λ as

$$\lambda^3+a_1\lambda^2+a_2\lambda+a_3=0,$$

then a_1 is minus the trace of the matrix, a_3 is minus its determinant, and a_2 is the coefficient of λ in $\det(\lambda I-B)$. The *Routh–Hurwitz criterion* tells that all eigenvalues of B have strictly negative real parts if and only if $a_1,a_3>0$ and $a_1a_2<a_3$. Further, if $a_1,a_3>0$ and $a_1a_2>a_3$, then there exists an eigenvalue with positive real part: Since its determinant and trace are negative, the eigenvalue with the largest absolute value is real and negative, but the two other eigenvalues have positive real parts (they could both be real or complex and conjugate). We already know from Lemma 3.4 that $a_1,a_3>0$ under condition (3.3).

Lemma 3.6 ([BK98] for $r=0$, [BT23] for $r>0$)**.** *If $r=0$ or $r>0$ is sufficiently small, then $(\widetilde{n}_{1a},\widetilde{n}_{1i},\widetilde{n}_2)$ is unstable for all sufficiently large $m>m^*$. In contrast, if $r>v$, then $(\widetilde{n}_{1a},\widetilde{n}_{1i},\widetilde{n}_2)$ is stable for all sufficiently large $m>m^*$.*

In words, a virus infection with very high mortality leads to the loss of stability for large m. In contrast, if $r > v$, i.e. if the mortality is below 50%, then for large m the coexistence equilibrium stays stable. We will see in the proof of the lemma below that the condition $r > v$ can presumably be relaxed. Hence, we have identified a qualitative effect of recovery on the behaviour of the model. Having Lemma 3.5, it is also straightforward to conjecture that if $(\widetilde{n}_{1a}, \widetilde{n}_{1i}, \widetilde{n}_2)$ is stable for m large, then in fact it is stable for all $m > m^*$, but such an assertion has not been proven so far.

Proof of Lemma 3.6. Note that according to (3.7), we have $\lim_{m\to\infty} \widetilde{n}_{1a} = 0$. For $m > m^*$, letting the right-hand side of the first equation of (3.3) be equal to zero, and dividing it with $\widetilde{n}_{1a} > 0$ we obtain

$$\lambda_1 - \mu_1 - C(\widetilde{n}_{1a} + \widetilde{n}_{1i}) - D\widetilde{n}_2 = \frac{r}{r+v} D\widetilde{n}_2.$$

Hence, it follows that

$$\lim_{m\to\infty} \lambda_1 - \mu_1 - C\widetilde{n}_{1i} - \frac{v}{r+v} D\widetilde{n}_2 = 0,$$

and thus in particular

$$\limsup_{m\to\infty} \widetilde{n}_2 \le \frac{\lambda_1 - \mu_1}{D} \frac{r+v}{v}.$$

In particular, $\widetilde{n}_2$ is bounded as a function of m. Hence, from (3.8) we conclude that $\lim_{m\to\infty} \widetilde{n}_{1i} = 0$ and thus

$$\lim_{m\to\infty} \widetilde{n}_2 = \frac{\lambda_1 - \mu_1}{D} \frac{r+v}{v}.$$

Hence, we obtain

$$\lim_{m\to\infty} a_1 = \lim_{m\to\infty} -(\lambda_1 - \mu_1 - D\widetilde{n}_2) + r + v + \mu_2 = (\lambda_1 - \mu_1)\frac{r}{v} + (r+v) + \mu_2 > 0,$$

further,

$$\lim_{m\to\infty} a_2 = \lim_{m\to\infty} -(\lambda_1 - \mu_1 - D\widetilde{n}_2)(r+v) - D\widetilde{n}_2 r + (\lambda_1 - \mu_1)\frac{r}{v}\mu_2 + \mu_2(r+v) - D\widetilde{n}_{1a} m v = (\lambda_1 - \mu_1)\frac{r}{v}\mu_2, \quad (3.12)$$

and, using (3.11)

$$\lim_{m\to\infty} a_3 = -\lim_{m\to\infty} \det A(\widetilde{n}_{1a}, \widetilde{n}_{1i}, \widetilde{n}_2) = \lim_{m\to\infty} \mu_2 v \widetilde{n}_2 = \frac{\lambda_1 - \mu_1}{D}(r+v)\mu_2 > 0$$

since $\lambda_1 > \mu_1$.

In the case $r = 0$ of no recovery, we see that $\lim_{m\to\infty} a_1 = v + \mu_2$, $\lim_{m\to\infty} a_2 = 0$, $\lim_{m\to\infty} a_3 = (\lambda_1 - \mu_1)v\mu_2$, and hence $\lim_{m\to\infty} a_1 a_2 - a_3 < 0$. Therefore for $r = 0$, for all sufficiently large $m > m^*$, the Jacobi matrix at $(\widetilde{n}_{1a}, \widetilde{n}_{1i}, \widetilde{n}_2)$ has two eigenvalues with positive real parts, and thus $(\widetilde{n}_{1a}, \widetilde{n}_{1i}, \widetilde{n}_2)$ is unstable. By continuity, the same assertion also holds for $r > 0$ sufficiently small, as asserted. On the other hand, for $r > v$ we obtain that

$$\begin{aligned}\liminf_{m\to\infty} a_1 a_2 - a_3 &> ((\lambda_1 - \mu_1) + (r+v) + \mu_2)((\lambda_1 - \mu_1)\mu_2) - (\lambda_1 - \mu_1)(r+v)\mu_2 \\ &> (r+v)(\lambda_1 - \mu_1)\mu_2 - (\lambda_1 - \mu_1)(r+v)\mu_2 = 0.\end{aligned}$$

Thus, for $r > v$, for all $m > m^*$ sufficiently large, $(\widetilde{n}_{1a}, \widetilde{n}_{1i}, \widetilde{n}_2)$ is asymptotically stable, as claimed. □

By Lemma 3.4, the only way $(\widetilde{n}_{1a}, \widetilde{n}_{1i}, \widetilde{n}_2)$ can be (hyperbolically) unstable for $m > m^*$ is that it has a negative real eigenvalue and a pair of complex eigenvalues with positive real parts. Indeed, by continuity, since for $m > m^*$ sufficiently close to m all eigenvalues have negative real parts, to obtain two eigenvalues with positive real parts, both such eigenvalues need to cross the imaginary axis of $\mathbb{C}$ at some values of $m > m^*$. But if any of these eigenvalues became zero for some $m > m^*$, then the determinant would vanish for such m, which would contradict Lemma 3.4. It also follows that for $r \ge 0$ sufficiently small (given all

the other parameters including v), there exists some $m^{**} > m^*$ such that at m^{**}, the Jacobi matrix has a negative (real) eigenvalue and a pair of purely imaginary eigenvalues. When this is not the case (since r is relatively large compared to v), we will put $m^{**} = \infty$.

We say that at $m^{**} < \infty$ the system (3.3) undergoes a *supercritical Hopf bifurcation*. That is, a pair of complex conjugate eigenvalues crosses the imaginary axes from negative to positive at a nonzero speed, and the corresponding eigenvalue (here: $(\widetilde{n}_{1a}, \widetilde{n}_{1i}, \widetilde{n}_2)$) loses its stability, and instead a stable hyperbolic periodic trajectory[26] surrounding the equilibrium emerges and starts attracting solutions started close to the equilibrium.[27] Right before the bifurcation point, the eigenvalue is locally a *stable focus*, which means coordinatewise oscillatory convergence of solutions to (3.3) started sufficiently close to $(\widetilde{n}_{1a}, \widetilde{n}_{1i}, \widetilde{n}_2)$ as $t \to \infty$, and right after it, the eigenvalue is locally an *unstable focus*, which means also spiral-like behaviour but repellence of the equilibrium and convergence of the solutions from any sufficiently small neighbourhood of the unstable equilibrium to the stable periodic orbit. That we indeed have a supercritical Hopf bifurcation in this case follows from the fact that a_1, a_2 are positive whenever $m > m^*$ and $a_1 a_2 - a_3$ is a smooth function of the parameter m (given all the other parameters) in $(m*, \infty)$ under the conditions of Lemma 3.6 implying $m^{**} < \infty$ with nonzero derivative at m^{**}, see the proof of [BK98, Proposition 3.1] for details.

This implies in particular that there exists $m' < m^{**}$ such that the Jacobi matrix for $m^* < m < m'$ the eigenvalues of the Jacobi matrix of (3.3) at $(\widetilde{n}_{1a}, \widetilde{n}_{1i}, \widetilde{n}_2)$ are all real, while for $m > m'$ two eigenvalues are complex (and conjugate). This is always true when there is a Hopf bifurcation at $m^{**} < \infty$, apart from the critical case when for $m = m^*$ two eigenvalues of the Jacobi matrix already coincide (cf. Exercise (11)). In that case, it is not known if there is a complex pair of eigenvalues for all $m > m^*$.

Simulations indicate that the periodic behaviour is true for any $m > m^*$ whenever $(\widetilde{n}_{1a}, \widetilde{n}_{1i}, \widetilde{n}_2)$ eventually loses its stability, with increasing amplitude and minimal subpopulation sizes within the cycle tending to 0 as $m \to \infty$. For $r = 0$ or $r > 0$ very small, this is actually also to prove theoretically, as shown by the following remark.

Remark 3.7. Let us note that a_1, a_2, a_3 are all increasing as functions of m on $[m^*, \infty)$ (where in the boundary case $m = m^*$ we identify $(\widetilde{n}_{1a}, \widetilde{n}_{1i}, \widetilde{n}_2)$ with $(\bar{n}_{1a}, 0, 0)$). From this together with the fact that $a_1 > 0$ and $a_3 = 0$ holds for $m = m^*$ and thus arguments of the proof of Lemma 3.5 imply that $a_2 < 0$ for this choice of m, we can deduce that $a_1 a_2$ is decreasing in m. But then, if $r = 0$, since a_3 tends to a positive limit as $m \to \infty$ but a_2 tends to zero, it follows that $a_3 - a_1 a_2$ as a function of m has a unique zero in (m^*, ∞), i.e. there is a unique Hopf bifurcation point, see [BK98, Proposition 3.2] and its proof. By continuity, this uniqueness also holds for $r > 0$ sufficiently small.

Although the solution to the autonomous system of ODEs (3.3) of course never hits 0, in the corresponding stochastic system this phenomenon comes with an increased risk of extinction of all subpopulations, which relates our model to the phenomenon of *paradox of enrichment* known from predator–prey models, cf. Remark 3.8 below.

Is there also $m' > m^*$ such that a pair of eigenvalues becomes complex at m' also in case $m^{**} < \infty$? Numerical simulations suggest a positive answer (see Section 3.9). This suggests that while we have seen that for $m > m^*$ close to m^* the eigenvalues of the Jacobi matrix at $(\widetilde{n}_{1a}, \widetilde{n}_{1i}, \widetilde{n}_2)$ are always real (and negative) and therefore the convergence to this equilibrium from nearby initial conditions is eventually coordinatewise monotone, increasing m leads to oscillatory convergence, which in the case of small r leads to divergence from the equilibrium and convergence to the stable periodic trajectory above the Hopf bifurcation point. Summarizing, we expect the behaviour of the system as shown in Tables 1 and 2 under the assumption that the three eigenvalues of the Jacobi matrix are pairwise distinct for $m = m^*$. We should however note that we have not proved the existence of $m' > m^*$ such that for all $m^* < m < m'$ the Jacobi matrix has three real eigenvalues and for $m > m'$ it has a pair of complex eigenvalues. Simulations still support this conjecture,

[26] We will not define here what hyperbolicity for a stable periodic orbit means, see [K98, Section 2.2.3] for a definition, but we note that hyperbolicity implies exponentially fast convergence to the periodic orbit started from all initial conditions sufficiently close to the orbit w.r.t. a suitable notion of distance, similarly to how hyperbolicity of an asymptotically stable equilibrium implies convergence of solutions started from nearby initial conditions to the equilibrium at an exponential speed.

[27] A *subcritical* Hopf bifurcation would correspond to a situation where right before the bifurcation, there is a stable equilibrium and an unstable periodic orbit, and right after the bifurcation, there is an unstable equilibrium and no periodic orbit.

Region	I.	II.	III.	IV.
Characterization	$0 < m < m^*$	$m^* < m < m'$	$m' < m < m^{**}$	$m > m^{**}$
Stability of $(\bar{n}_{1a},0,0,0)$	stable	unstable	unstable	unstable
Existence of $(\widetilde{n}_{1a},\widetilde{n}_{1i},\widetilde{n}_2)$	does not exist	exists	exists	exists
Stability of $(\widetilde{n}_{1a},\widetilde{n}_{1i},\widetilde{n}_2)$	-	stable	stable	unstable
Asymptotic behaviour of positive solutions to (3.3)	eventually coord. monotone convergence to $(\bar{n}_{1a},0,0,0)$	eventually coord. monotone convergence to $(\widetilde{n}_{1a},\widetilde{n}_{1i},\widetilde{n}_2)$	oscillatory convergence to $(\widetilde{n}_{1a},\widetilde{n}_{1i},\widetilde{n}_2)$	periodic behaviour

Table 1: Expected behaviour of the system (3.3) in the case when r is small compared to v (e.g. $r = 0$).

Region	I.	II.	III.
Characterization	$0 < m < m^*$	$m^* < m < m'$	$m > m'$ $(m^{**} = \infty)$
Stability of $(\bar{n}_{1a},0,0,0)$	stable	unstable	unstable
Existence of$(\widetilde{n}_{1a},\widetilde{n}_{1i},\widetilde{n}_2)$	does not exist	exists	exists
Stability of$(\widetilde{n}_{1a},\widetilde{n}_{1i},\widetilde{n}_2)$	-	stable	stable
Asymptotic behaviour of positive solutions to (3.3)	eventually coord. monotone convergence to $(\bar{n}_{1a},0,0,0)$	eventually coord. monotone convergence to $(\widetilde{n}_{1a},\widetilde{n}_{1i},\widetilde{n}_2)$	oscillatory convergence to $(\widetilde{n}_{1a},\widetilde{n}_{1i},\widetilde{n}_2)$

Table 2: Expected behaviour of the system (3.3) in the case when r is small compared to v (e.g. $r > v$ or larger).

see Figure 1. We have merely shown that for $m^{**} < \infty$ there is a complex pair of eigenvalues for all $m > m^{**}$ and also for $m < m^{**}$ sufficiently close to m^{**}.

Remark 3.8 (Paradox of enrichment). The fact that the coexistence equilibrium can lose its stability is a variant of the phenomenon called *paradox of enrichment* in ecology, which was introduced by Rosenzweig [R71] and is well-known from the context of predator–prey type dynamical systems (see e.g. [MM90] for an overview). It rests on a bifurcation that appears in the model studied in [BK98] (and also for $r > 0$ if r is small compared to v) in the following way: When the burst size m reaches a critical threshold, the coexistence equilibrium emerges and is initially stable. However, further increase in the burst size destabilizes it, giving rise to periodic limiting behaviour.

In the predator–prey context, the analogue of the burst size expresses how much energy the predator can gain out of a consumed unit prey, and the analogue of the equilibrium population size $\bar{n}_{1a}$ of active hosts is the carrying capacity of the prey population. An example for a system where this phenomenon occurs (see e.g. [M72])

$$\dot{x} = x\Big(1 - \frac{x}{K}\Big) - y\frac{x}{1+x}$$
$$\dot{y} = \delta y\frac{x}{1+x} - \gamma y,$$

for $\delta, \gamma > 0$, where $K > 0$ is the carrying capacity of the prey population.

Now, increasing the carrying capacity of the system while keeping all other parameters constant leads to periodic cycles with increasing amplitudes, where the lowest population size during a period approaches zero for both for the prey and the predators. This corresponds to an increased danger of extinction due to small stochastic fluctuations in the underlying individual-based model. The 'paradox' consists in the counter-intuitive effect that increasing carrying capacities may actually increase the risk of extinction for the whole system.

In our model, for r small compared to v and q not too close to 1, a similar high-amplitude periodicity (with low minimum value) can be observed. While varying $\bar{n}_{1a}$ (which can e.g. be achieved via fixing $\lambda_1 - \mu_1$ and varying C, or the other way around) would be analogous to the predator–prey setting, we used m as a bifurcation parameter. Nevertheless, we know from [BK98, Sections 3 and 5] that under suitable assumptions on the other parameters that we fix, a Hopf bifurcation can also be observed while varying $\bar{n}_{1a}$

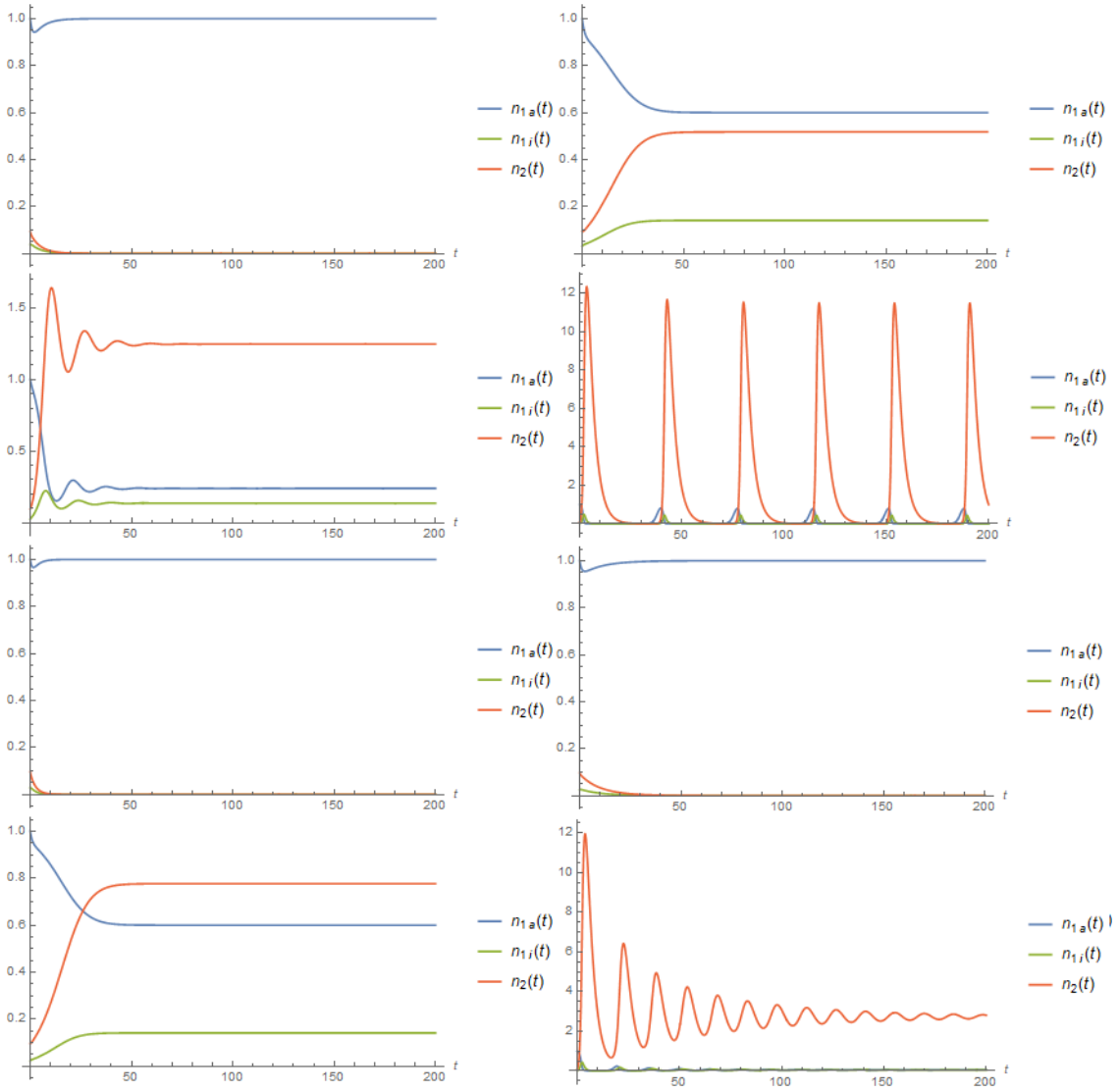


Figure 1: Behaviour of the system (3.3) for various values of m. First two rows: $r = 0, v = 1.1$. $m = 1$ ($mv \not> r + v$): Case I, there is no coexistence equilibrium, coordinatewise eventually monotone convergence to $(\bar{n}_{1a}, 0, 0)$. $m = 2$: Case II, we have coordinatewise eventually monotone convergence to the stable coexistence equilibrium $(\widetilde{n}_{1a}, \widetilde{n}_{1i}, \widetilde{n}_2)$. $m = 3$: Case III, $(\widetilde{n}_{1a}, \widetilde{n}_{1i}, \widetilde{n}_2)$ still stable but the convergence is already oscillatory in each coordinate. $m = 20$: After $(\widetilde{n}_{1a}, \widetilde{n}_{1i}, \widetilde{n}_2)$ lost its stability via the Hopf bifurcation, the limiting behaviour of the system is periodic.
Last two rows: Same choice of the parameters apart from r, whose value is now $0.55 = v/2$. $m = 1$: case I. $m = 2$: still case I. $m = 3$: (still) case II. $m = 20$ (and any $m > 20$): (still) case III. We see that the effect of recovery is not just hindering Hopf bifurcation but also increasing m^* and m', leading to a better scenario for the hosts and a worse one for the viruses (not surprisingly).

(more precisely, the analogue of $\bar{n}_{1a}$ in a rescaled variant of the system (3.3) for $r = 0$). Note that if we use $\bar{n}_{1a}$ as a bifurcation parameter, fixing m, it is crucial to assume that $mv > r + v$, otherwise the coexistence equilibrium will never exist and thus it cannot lose its stability.

It is further remarkable that as long as the coexistence equilibrium exists, its active coordinate $\widetilde{n}_{1a}$ does not depend on λ_1, μ_1, C (which are the only parameters $\bar{n}_{1a}$ depends on) but on the other parameters of the model. This is in analogy to the fact that in certain predator–prey models, the prey coordinate of the coexistence equilibrium between predators and prey does not depend on the carrying capacity of the prey, see e.g. [KC09, Section 2].

There are further, finer, and more global stability results regarding the system (3.3). However, at this point we will not continue with these, but we will incorporate dormancy into our model and present the main results of [BT23] regarding the full model, which will include some additional (partial) results about the qualitative behaviour of the extended, four-dimensional version of the dynamical system, which will also have some implications regarding the original system.

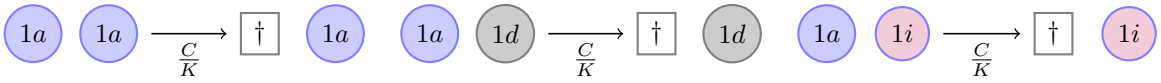
3.5 The full model of [BT23] with contact-mediated host dormancy

The form of dormancy that we will consider in an extension of the Beretta–Kuang model (with recovery) is *contact-mediated host dormancy*. Informally speaking, this means that the host may inactivate due to a contact with a virion. In mathematical terms, not all virus attacks will successfully lead to the infection of the affected host cell, but with probability $q \in (0, 1)$ they will make the host cell dormant, while the virus particle will not be lost. Dormant cells can later resuscitate or die, but they cannot get infected before resuscitation. Similarly to the infected cells, they do not reproduce and they feel no competitive pressure from the other host cells, but they exert competitive pressure on the active hosts.

In the biological literature, it has been reported that infected bacteria can enter a dormant state as part of a CRISPR-Cas immune response, thereby curbing phage epidemics (cf. [JF19] resp. [MNM19]). Moreover, it has been suggested that dormancy of hosts may even be initiated upon mere contact of virus particles with their cell hull, so that the dormant host may entirely avoid infection, cf. Bautista et al [BZW15]. Indeed, in experiments, Bautista et al observed that *Sulfolobus islandicus* (an archeon) populations may switch almost entirely into dormancy within hours after being exposed to the *Sulfolobus spindle-shape virus SSV9*, even when the initial virus-to-host ratio is relatively small.

We now modify the model of Section 3.1 in order to introduce dormancy as follows. We keep (i)-(iii) and (vi)-(viii) unchanged, whereas (iv) changes to

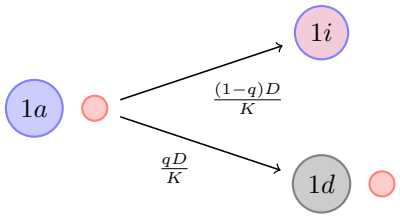
(iv') For some $C > 0$, for any ordered pair consisting of one active (1a) host cell and one other host cell (of either type 1a or 1i or 1d), at rate C/K, a death due to competition/overcrowding happens, affecting the first active individual, which is removed from the population.[28]



Moreover, (v) changes to

(v') We fix a number $q \in (0, 1)$. For any ordered pair of individuals containing one active (type 1a) cell and one virion (type 2), a virus attack happens at rate D/K. In this case, independently of the pre-history of the process, with probability $1 - q$ the host cell gets infected (i.e. switches from 1a to 1i) and the free virus (2) is 'removed' (in the sense that it enters the cell), whereas with probability q the host cell becomes dormant (i.e. switches from 1a to 1d) and the free virus (2) stays unaffected.

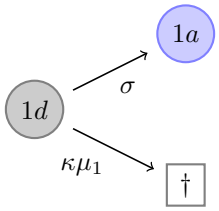
[28] While the model presented in this section can be seen as an extension of the model of [BK98] with dormancy and recovery, ODE-based preliminary models featuring contact-mediated dormancy and some other properties of the ODE (3.13) corresponding to the full model were already considered in [GW16, GW18]. In [BT23] we opted to follow [GW16] in including competition with dormant host individuals in order to stay close to their modeling frame, even though from the biological point of view this is debatable.



Finally, we extend the model with the following two points on the fate of dormant individuals:

(ix) A dormant (type 1d) individual resuscitates (i.e. switches back from 1d to 1a) at rate $\sigma > 0$.

(x) For some $\kappa \geq 0$, a dormant individual dies at rate $\kappa\mu_1$ (where we recall that μ_1 is the natural death rate of the active host population).



Note that there is no competition-induced dormancy here; the presence of type 1d is merely due to the virus attacks.

The corresponding Markov chain will still be denoted by $(\mathbf{N}_t)_{t\geq 0}$, but now it has four coordinates: $\mathbf{N}_t = (N_{1a,t}, N_{1d,t}, N_{1i,t}, N_{2,t})$, where $N_{1d,t}$ is defined analogously to (3.2) (it denotes the number of dormant individuals at time t). It has state space $\mathbb{N}_0^4$ and transitions

$$(n_{1a}, n_{1d}, n_{1i}, n_2) \to \begin{cases} (n_{1a}+1, n_{1d}, n_{1i}, n_2) \text{ at rate } \lambda_1 n_{1a}, \\ (n_{1a}-1, n_{1d}, n_{1i}, n_2) \text{ at rate } (\mu_1 + C\frac{n_{1a}+n_{1d}+n_{1i}}{K})n_{1a}, \\ (n_{1a}-1, n_{1d}, n_{1i}+1, n_2-1) \text{ at rate } \frac{(1-q)Dn_{1a}n_2}{K}, \\ (n_{1a}-1, n_{1d}+1, n_{1i}, n_2) \text{ at rate } \frac{qDn_{1a}n_2}{K}, \\ (n_{1a}+1, n_{1d}, n_{1i}-1, n_2) \text{ at rate } rn_{1i}, \\ (n_{1a}, n_{1d}, n_{1i}-1, n_2+m) \text{ at rate } vn_{1i}, \\ (n_{1a}, n_{1d}, n_{1i}, n_2-1) \text{ at rate } \mu_2 n_2, \\ (n_{1a}+1, n_{1d}-1, n_{1i}, n_2) \text{ at rate } \sigma n_{1d}, \\ (n_{1a}, n_{1d}-1, n_{1i}, n_2) \text{ at rate } \kappa\mu_1 n_{1d}. \end{cases}$$

Its only absorbing state is $(0,0,0,0)$, which corresponds to the extinction of all the four types. We will write $\mathbf{N}_t^K = (N_{1a,t}^K, N_{1d,t}^K, N_{1i,t}^K, N_{2,t}^K)$ for the rescaled process, where $N_{x,t}^K = N_{x,t}/K$ for $x \in \{1a, 1d, 1i, 2\}$. The underlying dynamical system is clearly

$$\begin{aligned} \frac{\mathrm{d}n_{1a}(t)}{\mathrm{d}t} &= n_{1a}(t)\big(\lambda_1 - \mu_1 - C(n_{1a}(t) + n_{1i}(t)) - Dn_2(t)\big) + rn_{1i}(t) + \sigma n_{1d}(t), \\ \frac{\mathrm{d}n_{1d}(t)}{\mathrm{d}t} &= qDn_{1a}(t)n_2(t) - (\kappa\mu_1 + \sigma)n_{1d}(t), \\ \frac{\mathrm{d}n_{1i}(t)}{\mathrm{d}t} &= (1-q)Dn_{1a}(t)n_2(t) - (r+v)n_{1i}(t), \\ \frac{\mathrm{d}n_2(t)}{\mathrm{d}t} &= mvn_{1i}(t) - (1-q)Dn_{1a}(t)n_2(t) - \mu_2 n_2(t), \end{aligned} \tag{3.13}$$

which gives back (3.3) for $q = 0$ after ignoring the dormant coordinate $n_{1d}(t)$, given that $n_{1d}(0) = 0$. The positive orthant of $\mathbb{R}^4$ is obviously invariant under this system. We write $(\mathbf{n}(t))_{t\geq 0} = (n_{1a}(t), n_{1d}(t), n_{1i}(t), n_2(t))_{t\geq 0}$

for the unique solution to (3.13) (given the initial condition). Arguing very similarly to Section 3.2, we obtain the following result (for the missing bits of the proofs see [BT23]).

Proposition 3.9 ([BK98]). *Let $q = 0$ and consider the dynamical system* (3.13).

1. $(0, 0, 0, 0)$ *is always an unstable equilibrium of the system.*
2. $(\bar{n}_{1a}, 0, 0, 0)$ *is asymptotically stable if the determinant of the Jacobi matrix at* $(\bar{n}_{1a}, 0, 0, 0)$ *is positive, which is equivalent to*
$$\bar{n}_{1a} < \frac{\mu_2(r+v)}{(1-q)D(mv-(r+v))} \tag{3.14}$$
and unstable if the determinant is positive, i.e.,
$$\bar{n}_{1a} > \frac{\mu_2(r+v)}{(1-q)D(mv-(r+v))}. \tag{3.15}$$
3. *If* (3.14) *holds, the system has no coordinatewise positive coexistence equilibrium. If* (3.15) *holds, there is a unique coordinatewise positive equilibrium* $(\widetilde{n}_{1a}, \widetilde{n}_{1d}, \widetilde{n}_{1i}, \widetilde{n}_2)$*, whose active coordinate satisfies*
$$\widetilde{n}_{1a} = \frac{\mu_2(r+v)}{(1-q)D(mv-(r+v))}. \tag{3.16}$$

In the initial phase of the epidemic, considering $N_{1a,t}/K$ as constant equal to $\bar{n}_{1a}$, $(N_{1d,t}, N_{1i,t}, N_{2,t})_{t\geq 0}$ can be approximated by the two-type branching process $(Z_{1d,t}, Z_{1i,t}, Z_{2,t})_{t\geq 0}$ with transitions

$$(n_{1d}, n_{1i}, n_2) \to \begin{cases} (n_{1d}, n_{1i}+1, n_2-1) & \text{at rate } (1-q)D\bar{n}_{1a}n_2, \\ (n_{1d}+1, n_{1i}, n_2) & \text{at rate } qD\bar{n}_{1a}n_2, \\ (n_{1d}, n_{1i}-1, n_2) & \text{at rate } rn_{1i}, \\ (n_{1d}, n_{1i}-1, n_2+m) & \text{at rate } vn_{1i}, \\ (n_{1d}, n_{1i}, n_2-1) & \text{at rate } \mu_2 n_2, \\ (n_{1d}-1, n_{1i}, n_2) & \text{at rate } (\kappa\mu_1+\sigma)n_{1d} \end{cases} \tag{3.17}$$

and with the same initial condition. This has mean matrix

$$\widetilde{J} = \begin{pmatrix} -\kappa\mu_1-\sigma & 0 & 0 \\ 0 & -(r+v) & mv \\ qD\bar{n}_{1a} & (1-q)D\bar{n}_{1a} & -(1-q)D\bar{n}_{1a}-\mu_2 \end{pmatrix}.$$

We immediately see that $-\kappa\mu_1 - \sigma < 0$ is an eigenvalue of the matrix (with left eigenvector $(1, 0, 0)$). The other two eigenvalues are therefore the eigenvalues of the last 2×2 block, which itself has a negative trace, so that at least one eigenvalue is always negative. Not surprisingly given the results of Section 3.3 and Proposition 3.9, the third eigenvalue of $\widetilde{J}$ is positive if and only if (3.15) holds, i.e. if and only if the system (3.13) has a coexistence equilibrium and $(\bar{n}_{1a}, 0, 0, 0)$ is unstable, while the third eigenvalue is negative if and only if (3.14) holds, i.e. the system has no coexistence equilibrium and $(\bar{n}_{1a}, 0, 0, 0)$ is locally asymptotically stable.

Remark 3.10 (The effect of contact-mediated host dormancy). Note that condition (3.15) implies that large reproduction rates λ_1 can be hazardous when facing a virus infection (with or without dormancy mechanism). This is analogous to the case $q = 0$, and the quantitative difference is that for $q > 0$, the threshold for $\bar{n}_{1a}$ above which the branching process is supercritical (i.e. there is an asymptotically positive chance of a major epidemic) is $\frac{1}{1-q}$ times the same threshold for $q = 0$.

At first glance, there may be hypothetical scenarios where a population threatened by recurring virus invasions might not realize its full reproductive potential in order to avoid persistent epidemics. The way

to maximize its long-term average fitness in the face of virus epidemics could then be to invest remaining resources into a dormancy-defence, which allows for higher carrying capacities during infections, and the 'reproductive trade-off' vanishes (at least to some degree). However, such a self-constraining strategy might be vulnerable to the invasion of *selfish cheaters*, i.e. of other species investing in a higher reproduction rate instead of dormancy. Investigating the balance of classical fitness (in competition with other species) and strategies (e.g. dormancy-based) reducing reproductive rates in order to cope with recurring infections could be a topic for future work.

Nevertheless, there are also some qualitative differences between the case $q = 0$ and the one $q > 0$. Namely, the mean matrix $\widetilde{J}$ is not irreducible, unlike J and the last 2×2 block of $\widetilde{J}$, which we will denote by J_2. This can be interpreted as follows. Starting the branching process (where we fix the active population size and consider resuscitations as deaths) from an initial condition where there are dormant individuals only, no infected individuals and no viruses will ever be created and the population size of the branching process will tend monotonically to 0. Hence, one has to be careful when one wants to perform an invasion analysis including a multitype branching process approximation similar to the one in Section 2. Indeed, e.g. knowing just that the branching process reaches total population size εK, one cannot be sure if these individuals are not all dormant, and therefore one cannot guarantee that the risk of extinction of the invading types (1d, 1i, and 2) is over on the short term.

Instead, we will work with the projection $(Z_{1i,t}, Z_{2,t})_{t\geq 0}$ of the branching process on its infected and virus coordinate. Thanks to the fact that transitions in (3.17) depending on the value of n_{1d} only influence the transition rates in the dormant coordinate and that dynamics of the dormant individuals does not influence the one of infected individuals and viruses, $(Z_{1i,t}, Z_{2,t})_{t\geq 0}$ is an autonomous Markov chain and itself a 2-type branching process. Its mean matrix is the last 2×2 block J_2 of $\widetilde{J}$, which is the same as J in (3.10) but with D replaced by $(1-q)D$ everywhere. Thus,

> the three-type branching process is supercritical (resp. subcritical) if and only if this two-dimensional projection is supercritical (resp. subcritical).

If we can guarantee that this branching process approximates $(N_{1i,t}, N_{2,t})$ well until the latter dies out or reaches total size $\approx \varepsilon K$, then we have a good chance for a useful branching approximation similarly to Section 2, and the number of dormant host individuals will also reach the same order of magnitude as the one of infected host individuals and virions. (Note however that such an autonomy is only true for the branching process approximation, due to the assumption that the active population size is fixed. For example, the behaviour $(N_{1i,t}, N_{2,t})$ is influenced by the one of $N_{1d,t}$ because the latter has an impact on $N_{1a,t}$ via competition and resuscitation.)

Whenever J_2 has a positive eigenvalue, we will denote it by $\widetilde{\lambda}$ (there will be no clash with the same notation in Section 2).

Exercise 12 (easy)**.** *In case J_2 has no positive eigenvalue, what is the largest eigenvalue of $\widetilde{J}$?*

The survival probability of the branching process (either the full three-dimensional one or its projection on the infected and virus coordinates) started with one single virus and no dormant or infected hosts will be denoted by s_2. Of course, $s_2 < 1$ if the branching process is supercritical, i.e. (3.15) holds, whereas $s_2 = 1$ if the branching process is subcritical, i.e. (3.14) holds.

Exercise 13. *Using a first-step analysis, derive the system of generating equations (analogous to* (2.16)*) for s_{1d}, s_{1i}, and s_2, where s_{1d} (resp. s_{1i}) denotes the survival probability of the branching process started with one single dormant (resp. infected) individual.*

1. *Convince yourself that $s_{1d} = 1$ follows from your system of equations.*
2. *Show that $s_2 < 1$ if and only if $s_{1i} < 1$, and show that these conditions are equivalent to* (3.15)*.*

3.6 The dynamical system III: some global properties

Before stating the main results of [BT23], it is useful to formulate some related assertions on the global behaviour of the dynamical system (3.13). This system is more difficult to study than (3.3) due to the extra dimension, and in some cases we will have to restrict to partial results. E.g., is clear by continuity that for $r = 0$ or for $r > 0$ very small compared to v the system (3.13) also exhibits a Hopf bifurcation, whereas for $r > 0$ large enough compared to v it does not, but specifying which choices of parameters belong to which of the two cases is tedious even numerically. Consequently, after a successful virus invasion, we will not be able to fully determine the fate (stable coexistence vs. periodic behaviour) of the stochastic host–virus system. Nevertheless, we have some global results (based on assertions of [BK98] for $r = q = 0$) that hold regardless of the presence or absence of a Hopf bifurcation. The first one tells us that starting from an initial condition with only positive coordinates, the mono-type equilibrium $(\bar{n}_{1a}, 0, 0, 0)$ will never be reached under the coexistence condition (3.15).

Proposition 3.11 (Non-extinction of the virus epidemic; [BK98, BT23])**.** *Consider the dynamical system* (3.13)*. Assume that* (3.15) *holds, and* $(n_{1a}(0), n_{1d}(0), n_{1i}(0), n_2(0)) \in (0,\infty)^4$*. Then* $(n_{1a}(t), n_{1d}(t), n_{1i}(t), n_2(t))$ *does not tend to* $(\bar{n}_{1a}, 0, 0, 0)$ *as* $t \to \infty$*, not even along a diverging subsequence of time-points.*

Before we prove this proposition, let us mention some of its consequences. Since coordinatewise nonnegative solutions of (3.13) are bounded, Proposition 3.11 together with a simple compactness argument implies that started from any initial condition $(n_{1a}(0), n_{1d}(0), n_{1i}(0), n_2(0)) \in (0,\infty)^4$, there exists a $\varrho > 0$ such that

$$\liminf_{t\to\infty} \big\|(n_{1a}(t), n_{1d}(t), n_{1i}(t), n_2(t)) - (\bar{n}_{1a}, 0, 0, 0)\big\|_1 \geq \varrho. \tag{3.18}$$

This assertion is known as $(\bar{n}_{1a}, 0, 0, 0)$ being a *uniform strong repeller*; cf. [BK98, Corollary 4.2] for its analogue in the recovery- and dormancy-free three-dimensional case. The following corollary is analogous to [BK98, Lemma 2.3 and Theorem 4.2], but since that paper provides no explicit proof and our setting is more complex, we present a proof for completeness in Appendix E.

Corollary 3.12 (Population bounds; [BK98, BT23])**.** *Consider the dynamical system* (3.13)*. Assume that* (3.15) *holds, and* $(n_{1a}(0), n_{1d}(0), n_{1i}(0), n_2(0)) \in (0,\infty)^4$*. Then*

$$\liminf_{t\to\infty} n_j(t) > 0$$

holds for all $j \in \{1a, 1d, 1i, 2\}$*, and*

$$\limsup_{t\to\infty}\ n_{1a}(t) + n_{1d}(t) + n_{1i}(t) < \bar{n}_{1a}.$$

Further,

$$\limsup_{t\to\infty} n_2(t) < \frac{mv\bar{n}_{1a}}{\mu_2}.$$

The positivity of the lim inf's of the coordinates $n_{1d}(t), n_{1i}(t), n_2(t)$ is called the *uniform strong persistence* of the system (3.13). By our uniform approximation result, in this case, the macroscopic virus epidemic will also be present for long times (with high probability conditional on a successful invasion) in the stochastic model with large enough carrying capacities K.

Exercise 14 (Initial conditions in Corollary 3.12)**.** *Coordinatewise positivity cannot be replaced by* $n_{1a}(0)$ *and at least one of the coordinates* $n_{1d}(0), n_{1i}(0), n_2(0)$ *being positive. To see this, determine whether* $\liminf_{t\to\infty} n_j(t) > 0$ *holds for all* $j \in \{1a, 1d, 1i, 2\}$ *in case*

1. *if* $n_{1a}(0) > 0$*,* $n_{1d}(0) > 0$*, but* $n_{1i}(0) = n_2(0) = 0$*,*
2. *and if* $n_{1a}(0) > 0$ *and* $(n_{1d}(0), n_{1i}(0), n_2(0)) \in [0,\infty)^3$ *is such that* $\max\{n_{1i}(0), n_2(0)\} > 0$*.*

Now we proceed with the proof of Proposition 3.11.

Proof of Proposition 3.11. Proposition 3.11 is the analogue of the assertion [BK98, Lemma 4.1] that treated the case without dormancy or recovery and slightly with different competition. Our proof (which originates from the proof of Proposition 2.4 in [BT23]) is indeed the analogue of the one in [BK98], which relies on the idea of Chetaev's instability theorem [C61].

Let $V\colon [0,\infty)^4 \to \mathbb{R}$, $(\widehat{n}_{1a}, \widehat{n}_{1d}, \widehat{n}_{1i}, \widehat{n}_2) \mapsto w_{1i}\widehat{n}_{1i} + w_2\widehat{n}_2$ for some $w_{1i}, w_2 > 0$. Let us write the system (3.13) as $\dot{\mathbf{n}}(t) = f(\mathbf{n}(t))$ and fix $\varepsilon > 0$. Then, the standard Euclidean scalar product of the gradient of V and f at $(\widehat{n}_{1a}, \widehat{n}_{1d}, \widehat{n}_{1i}, \widehat{n}_2) \in [0,\infty)^4$ with $\widehat{n}_{1a} > \bar{n}_{1a} - \varepsilon$ equals

$$\begin{aligned}\langle \nabla V, f\rangle|_{(\widehat{n}_{1a},\widehat{n}_{1d},\widehat{n}_{1i},\widehat{n}_2)} &= w_{1i}((1-q)D\widehat{n}_{1a}\widehat{n}_2 - \widehat{n}_{1i}(r+v)) + w_2(-(1-q)D\widehat{n}_{1a}\widehat{n}_2 + mv\widehat{n}_{1i} - \mu_2\widehat{n}_2)\\ &= \widehat{n}_{1i}\big[mvw_2 - w_{1i}(r+v)\big] + \widehat{n}_2\big[(1-q)D\widehat{n}_{1a}w_{1i} - (1-q)D\widehat{n}_{1a}w_2 - \mu_2 w_2\big]\\ &> \widehat{n}_{1i}\big[mvw_2 - w_{1i}(r+v)\big] + \widehat{n}_2\big[(1-q)D(\bar{n}_{1a}-\varepsilon)w_{1i} - (1-q)D\widehat{n}_{1a}w_2 - \mu_2 w_2\big].\end{aligned}$$

Hence, $\langle \nabla V, f\rangle|_{(\widehat{n}_{1a},\widehat{n}_{1d},\widehat{n}_{1i},\widehat{n}_2)}$ is positive (in other words, V is *positive definite* w.r.t. the dynamical system) once

$$mvw_2 > (r+v)w_{1i} \qquad \text{and} \qquad (1-q)w_{1i}D(\bar{n}_{1a}-\varepsilon) > ((1-q)D(\bar{n}_{1a}-\varepsilon) + \mu_2)w_2, \tag{3.19}$$

in other words,

$$\frac{mv}{r+v}w_2 > w_{1i} > w_2\frac{(1-q) + \frac{\mu_2}{D(\bar{n}_{1a}-\varepsilon)}}{1-q} = w_2\Big[1 + \frac{\mu_2}{(1-q)D(\bar{n}_{1a}-\varepsilon)}\Big].$$

Since $w_{1i} > 0, w_2 > 0$, this requires

$$\bar{n}_{1a} - \varepsilon > \frac{\mu_2(r+v)}{(1-q)D(mv-(r+v))},$$

which holds whenever $\varepsilon \in (0, \bar{n}_{1a} - n_{1a})$, where we recall that $\bar{n}_{1a} > \widetilde{n}_{1a}$ under the condition (3.15). Then we can indeed choose $w_{1i}, w_2 > 0$ satisfying (3.19), and thus we can find $d > 0$ such that for such a choice of w_{1i}, w_2, and ε, we have

$$\nabla V > dV \qquad \text{on } B_\varepsilon((\bar{n}_{1a},0,0,0)) \cap (0,\infty)^4 \tag{3.20}$$

where for $x \in \mathbb{R}^4$ and $\varrho > 0$, $B_\varrho(x)$ denotes the open ℓ^2-ball of radius ϱ around x.

Now, let us assume that $(n_{1a}(0), n_{1d}(0), n_{1i}(0), n_2(0)) \in (0,\infty)^4$. Then it is clear that for all $t > 0$, $n_{1i}(t) \neq 0$ and $n_2(t) \neq 0$. Now, if $\lim_{t\to\infty}(n_{1a}(t), n_{1d}(t), n_{1i}(t), n_2(t)) = (\bar{n}_{1a}, 0, 0, 0)$, there exists $t_0 > 0$ such that for all $t > 0$, $(n_{1a}(t), n_{1d}(t), n_{1i}(t), n_2(t)) \in B_\varepsilon((\bar{n}_{1a},0,0,0)) \cap (0,\infty)^4$. Hence, by (3.20), $\lim_{t\to\infty} V(n_{1a}(t), n_{1d}(t), n_{1i}(t), n_2(t)) = \infty$, which contradicts the assumption that $\lim_{t\to\infty}(n_{1i}(t), n_2(t)) = (0,0)$.

From this it is in fact easy to derive that $(n_{1a}(t), n_{1d}(t), n_{1i}(t), n_2(t))$ cannot even converge to $(\bar{n}_{1a}, 0, 0, 0)$ along any diverging sequence of times, but let us provide the details for completeness. Since V is positive definite on $\mathcal{B} := B_\varepsilon((\bar{n}_{1a},0,0,0)) \cap (0,\infty)^4$, the ω-limit set Ω_0 of any solution to (3.13) (i.e., the set of subsequential limits of the solution as $t \to \infty$) started from $\mathcal{B}$ satisfies

$$\Omega_0 \cap \overline{\mathcal{B}} \subseteq \{\langle \nabla V, f\rangle = 0\}$$

where $\overline{\mathcal{B}}$ is the closure of $\mathcal{B}$. In terms of these objects, we have already verified that $(\bar{n}_{1a},0,0,0) \in \{\langle \nabla V, f\rangle = 0\}$ and that $(\bar{n}_{1a},0,0,0) \neq \Omega_0 \cap \overline{\mathcal{B}}$.

Using the definition of V and the fact that $[0,\infty)^4$ is positively invariant under (3.13), we conclude that $\Omega_0 \cap \overline{\mathcal{B}}$ contains only points of the form $(\widehat{n}_{1a}, \widehat{n}_{1d}, 0, 0)$, where $\widehat{n}_{1a}, \widehat{n}_{1d} > 0$. However, if a coordinatewise nonnegative solution to (3.13) started from $(0,\infty)^4$ is such that its infected and virus coordinate tend to zero, then its dormant coordinate must also tend to zero and hence its active coordinate to $\bar{n}_{1a}$. We conclude that $\Omega_0 \cap \overline{\mathcal{B}} \subseteq \{(\bar{n}_{1a},0,0,0)\}$. But since $(\bar{n}_{1a},0,0,0) \neq \Omega_0 \cap \overline{\mathcal{B}}$, it follows that $\Omega_0 \cap \overline{\mathcal{B}} = \emptyset$, and thus the proposition is proven. □

To get a flavour of using Lyapunov functions to verify stability of an equilibrium, the reader can solve the following exercise, which also explains the definition of a *Lyapunov function* and a *strong Lyapunov function*. (The solution to this exercise can be found in [BK98], but it is presumably more tedious to reproduce that proof than to solve the exercise independently.) The assertion in the exercise implies that the equilibrium $(\bar{n}_{1a}, 0, 0)$ of the 3-dimensional system (3.3) is globally asymptotically stable on $(0,\infty)^3$ for $q = r = 0$.

Exercise 15 (Global stability of $(\bar{n}_{1a}, 0, 0)$ for $q = r = 0$ in case there is no coexistence equilibrium)**.** *Consider the set*

$$\Omega = \Big\{(n_{1a}, n_{1i}, n_2) \in [0,\infty)^3 \colon n_{1a} + n_{1i} \le \bar{n}_{1a}, n_2 \le \frac{mv\bar{n}_{1a}}{\mu_2}\Big\}.$$

It was shown in [BK98, Proposition 2.2] that the set Ω is a global attractor on $[0,\infty)^3$ in the sense that any solution to (3.3) *started from $[0,\infty)^3$ will eventually either enter the interior Ω^o of Ω or converge to a point on $\partial\Omega$. Using this, for $w_{1i}, w_2 > 0$ consider the function $V\colon (0,\infty)^3 \to \mathbb{R}$,*

$$V(n_{1a}, n_{1i}, n_2) = Cn_{1a} - (\lambda_1 - \mu)\log n_{1a} + w_{1i}n_{1i} + w_2 n_2$$

Assume that (3.5) *holds. Show that there exists a choice $w_{1i}, w_2 > 0$ such that*

(A) $\langle \nabla V, f\rangle \le 0$ on Ω^o, and

(B) V has a unique strict global minimum w.r.t. Ω in $(\bar{n}_{1a}, 0, 0)$.

The two properties are the defining properties of V being a Lyapunov function *(corresponding to the equilibrium $(\bar{n}_{1a}, 0, 0)$), which implies that $(\bar{n}_{1a}, 0, 0)$ is (Lyapunov) stable. Of course, we have already known this before, but now observe that $\langle \nabla V, f\rangle = 0$ holds only in the point $(\bar{n}_{1a}, 0, 0)$.*[29] *Thus, thanks to the positive invariance of Ω,* Lyapunov's stability theorem *(a.k.a.* Lyapunov–Le Salle theorem*) implies that $(\bar{n}_{1a}, 0, 0)$ is globally asymptotically stable on Ω^o (see also [BK98, Section 4]). Thanks to the above mentioned attractivity of Ω, it follows that $(\bar{n}_{1a}, 0, 0)$ is globally asymptotically stable on $(0,\infty)^3$.*[30]

3.7 Main results of [BT23] and discussion

What do we expect from the stochastic system started from $\approx K\bar{n}_{1a}$ active hosts and one single virus under the condition (3.15), i.e. when (3.13) has a coexistence equilibrium and the branching process is supercritical? The first phase of the virus invasion should work similarly to the first phase of the invasions studied in Sections 1 and 2. Dormant hosts, infected hosts, and virions should either die out rapidly (in $o(\log K)$ time) or they should start growing exponentially and the total population size of *infected hosts and virions* should reach εK (for K large and ε small) in $\frac{1}{\widetilde{\lambda}}(1 \pm o(1))\log K$ time, where we recall that $\widetilde{\lambda}$ is the only positive eigenvalue of the mean matrix J_2 in the supercritical case.

What happens after this first phase is less clear than in the case of Section 2 because we do not know when precisely $(\widetilde{n}_{1a}, \widetilde{n}_{1d}, \widetilde{n}_{1i}, \widetilde{n}_2)$ is asymptotically stable and how global this stability is, and similarly, in the phase above the Hopf bifurcation point m^{**} (preserving the notation from the three-dimensional case), how global the stability of the stable hyperbolic periodic orbit is. However, based on Corollary 3.12, the following three properties should also be satisfied with high probability (as $K \to \infty$ followed by $\varepsilon > 0$). After the end of the first phase, within an additional amount of time T, which can be chosen arbitrarily large but not depending on K, for $\beta > 0$ sufficiently small,

- the rescaled sizes of all the four subpopulations should be bounded from below β,
- the rescaled total (active+dormant+infected) host population size should stay bounded by some number strictly smaller than $\bar{n}_{1a} - \beta$ from above,

[29] When properties (A) and (B) hold with strict inequality in A apart from the corresponding equilibrium, we say that V is a *strong* Lyapunov function.

[30] Of course, solutions started from certain points of the boundary of $[0,\infty)^3$ also tend to $(\bar{n}_{1a}, 0, 0)$. From which points precisely?

- and the rescaled virus population size should be bounded by $\frac{mv\bar{n}_{1a}}{\mu_2} - \beta$.

Hence, for $\beta > 0$ let us introduce the *persistence set*

$$S_\beta := \Big\{(\widetilde{n}_{1a}, \widetilde{n}_{1d}, \widetilde{n}_{1i}, \widetilde{n}_2) \in (0,\infty)^4 \colon n_v \geq \beta, \forall v \in \{1a, 1d, 1i, 2\}, \widetilde{n}_{1a} + \widetilde{n}_{1d} + \widetilde{n}_{1i} \leq \bar{n}_{1a} - \beta, \\ \widetilde{n}_2 \leq \frac{mv\bar{n}_{1a}}{\mu_2} - \beta\Big\}. \tag{3.21}$$

Note that S_β is always well-defined and non-empty if $\beta \in (0, \bar{n}_{1a} \min\{1, \frac{mv}{\mu_2}\})$ (since we assumed that $\lambda_1 > \mu_1$). Further,

$$T_{S_\beta} := \inf\{t \geq 0 \colon (N^K_{1a,t}, N^K_{1d,t}, N^K_{1i,t}, N^K_{2,t}) \in S_\beta\} \tag{3.22}$$

is the hitting time for the persistence region S_β (it is again a stopping time for the canonical filtration). Define also

$$T^2_\varepsilon := \inf\big\{t \geq 0 \colon N_{1i,t} + N_{2,t} = \lfloor \varepsilon K \rfloor\big\}, \tag{3.23}$$

for $\varepsilon \geq 0$. Then, in particular, T^2_0 is the extinction of the total population of infected individuals and viruses.

Finally, there should be *no third phase of the invasion*, since thanks to Corollary 3.12, after a successful virus invasion no subpopulation should go extinct on the short term in the stochastic system either. Thus, on the $\log K$ scale, an entire successful invasion should take about $1/\widetilde{\lambda}$ time.

Indeed, we have the following results (see [BT23, Section 2.5]). Our first theorem states that the probability of a succesful invasion of the virus particles, i.e. of reaching the set S_β for some $\beta > 0$ before extinction of the invaders, converges to the survival probability $1 - s_2 \geq 0$ of the approximating branching process as $K \to \infty$.

Theorem 3.13 ([BT23])**.** *Assume that $\widetilde{\lambda} \neq 0$. Assume further that*

$$N^K_{1a}(0) \underset{K\to\infty}{\to} \bar{n}_{1a}$$

almost surely and

$$(N^K_{1d}(0), N^K_{1i}(0), N^K_2(0)) = (0, 0, \tfrac{1}{K}).$$

Then for all sufficiently small $\beta > 0$, we have

$$\lim_{K\to\infty} \mathbb{P}\Big(T_{S_\beta} < T^2_0\Big) = 1 - s_2. \tag{3.24}$$

The next theorem shows that in case of a macroscopic/persistent epidemic, the time until reaching the set S_β (which includes the coexistence equilibrium $(\widetilde{n}_{1a}, \widetilde{n}_{1d}, \widetilde{n}_{1i}, \widetilde{n}_2)$ of the dynamical system) behaves like $\log K/\widetilde{\lambda}$.

Theorem 3.14 ([BT23])**.** *Under the assumptions of Theorem 3.13, in case* (3.15) *holds (equivalently, $s_2 < 1$), for all sufficiently small $\beta > 0$ we have that on the event $\{T_{S_\beta} < T^2_0\}$,*

$$\lim_{K\to\infty} \frac{T_{S_\beta}}{\log K} = \frac{1}{\widetilde{\lambda}} \tag{3.25}$$

in probability.

The final theorem provides information on the time of extinction of the epidemic and implies that with high probability, the rescaled active population size stays close to its virus-free equilibrium $\bar{n}_{1a}$ and the dormant population stays small until this extinction (after which it decreases to 0 if it is not yet extinct). This theorem also holds for $\widetilde{\lambda} > 0$ where both persistence and non-persistence of the epidemic have a positive probability.

Theorem 3.15 ([BT23]). *Under the assumptions of Theorem 3.13, for all sufficiently small $\beta > 0$ we have that on the event $\{T_0^2 < T_{S_\beta}\}$,*

$$\lim_{K\to\infty} \frac{T_0^2}{\log K} = 0 \tag{3.26}$$

and

$$\mathbb{1}_{\{T_{S_\beta} > T_0^2\}} \big\| (N^K_{1a,T_0^2}, N^K_{1d,T_0^2}) - (\bar{n}_{1a}, 0) \big\| \underset{K\to\infty}{\longrightarrow} 0, \tag{3.27}$$

both in probability, where $\|\cdot\|$ is an arbitrary (but fixed) norm on $\mathbb{R}^2$.

These theorems do not tell about the fate of our rescaled stochastic population process after time T_{S_β}, and as already anticipated, we expect that the fate of the process will depend on the stability of the coexistence equilibrium and possible Hopf bifurcations. While we already sketched in Section 3.4 what we expect for $q = 0$, we will provide some additional simulations and conjectures (also for $q > 0$) below in Section 3.9.

Nevertheless, by virtue of Corollary 3.12, we can still provide an assertion on the long-term behaviour of our stochastic system. Namely, if $T_{S_\beta} < T_0^2$, then for $T > 0$ sufficiently large, with high probability, at time $T_{S_\beta} + T$ the process will again be situated in S_β. This is true because [EK86, Theorem 2.1, p. 456] guarantees that $(\mathbf{N}_{T_{S_\beta}+t})_{t\in[0,T]}$ is well-approximated by the solution $(\mathbf{n}_t)_{t\in[0,T]}$ of (3.13) given convergence of the initial conditions, and Corollary 3.12 implies that started from anywhere in S_β, $\mathbf{n}_T$ will be situated in S_β for all $T > 0$ large enough. More precisely, we have the following result, whose proof is now immediate.

Corollary 3.16 ([BT23]). *Assume that* (3.15) *holds. Then for all sufficiently small $\beta > 0$ and sufficiently large $T > 0$, we have*

$$\lim_{K\to\infty} \mathbb{P}\big(\mathbf{N}_{T_{S_\beta}+T} \in S_\beta \big| T_{S_\beta} < T_0^2\big) = 1. \tag{3.28}$$

Note that (3.28) is equivalent to the fact that the assertions

$$\lim_{K\to\infty} \mathbb{P}\big(N^K_{1a,T_{S_\beta}+T} + N^K_{1d,T_{S_\beta}+T} + N^K_{1i,T_{S_\beta}+T} \le \bar{n}_{1a} - \beta \big| T_{S_\beta} < T_0^2\big) = 1,$$

$$\lim_{K\to\infty} \mathbb{P}\Big(N^K_{2,T_{S_\beta}+T} \le \frac{m\upsilon\bar{n}_{1a}}{\mu_2} - \beta \Big| T_{S_\beta} < T_0^2\Big) = 1,$$

and

$$\lim_{K\to\infty} \mathbb{P}\big(N^K_{\upsilon,T_{S_\beta}+T} \ge \beta \big| T_{S_\beta} < T_0^2\big) = 1, \qquad \forall \upsilon \in \{1a, 1d, 1i, 2\}$$

hold. I.e., we have persistence of the epidemic on intervals starting at T_{S_β} whose length does not scale with K.

Remark 3.17 (The reproduction number, relation to stochastic epidemic models). The distinction between an initial stochastic phase, where an invader can be described by a branching process, followed by deterministic behaviour, where the whole system is well-described by a dynamical system, is of course reminiscent of stochastic and deterministic epidemic modelling. In stochastic epidemic models like the standard SIR (susceptible–infected–removed) model, the *basic reproduction number* R_0 of the epidemic is defined as the

> expected number of infections generated by one infectious individual in a large susceptible population,

cf. [AB00, Section 2.1]. Despite not treating pathogens as individuals and assuming that the population size is constant (or decreases only due to deaths caused by the infectious disease), the quantity R_0 can already be introduced in the basic SIR model the same way as in our model.

Note that we can define R_0 in our model in such a way that it still fulfills the heuristic definition of [AB00] (where we always assume that $\lambda_1 > \mu_1$). In order to obtain 'a large susceptible population', we will have to assume that K is large, since the equilibrium population size scales like $K(\bar{n}_{1a} + o(1))$ as $K \to \infty$. Then, similarly to the branching process approximation of types 1d, 1i, and 2 during the initial phase of the epidemic, we will assume that the rescaled susceptible population size is fixed as $\bar{n}_{1a}$ (ignoring also the

question of whether this number is an integer). Let us now look at an infected individual in this situation. It either recovers with probability $r/(r+v)$ or dies due to lysis, giving rise to m new virions, with probability $v/(r+v)$. Each of these new virions will eventually either degrade, which happens at rate μ_2, or successfully attack a susceptible individual. Since there are $K\bar{n}_{1a}$ susceptibles, the probability that the latter event occurs is $\frac{(1-q)D\bar{n}_{1a}}{(1-q)D\bar{n}_{1a}+\mu_2}$. The number of infected individuals emerging from attacks by these m viruses is the average number of infections generated by the originally infected individual. Thus, we obtain the expression

$$R_0 = \frac{mv(1-q)D\bar{n}_{1a}}{(r+v)((1-q)D\bar{n}_{1a}+\mu_2)}$$

for the reproduction number in our model. Note that R_0 depends on q but not on κ and μ, and in particular it is the same as for a dormancy-free epidemic with lower infectivity if we replace q by 0 and D by $(1-q)D$. This gives a rather natural interpetation of the effect of dormancy from an epidemiological point of view. Indeed, $R_0 > 1$ holds if and only if

$$(mv-(r+v))(1-q)D\bar{n}_{1a} > (r+v)\mu_2,$$

which is precisely our coexistence condition (3.15).

Note further that R_0 can also be interpreted as the average number viruses who are the 'offspring' of a single given virus, obtained via infection of a susceptible individual producing secondary viruses via lysis. We see that $R_0 > 1$ is equivalent to condition (3.15), which we interpret as the average number of 'offspring' of a given virus being at least 1. This provides a heuristic reason why $s_{1i} \neq 1$ is equivalent to $s_2 \neq 1$ (cf. Exercise 13).

3.8 A few words about the proofs in [BT23]

Similarly to Sections 1 and 2, unfortunately we will once again not be able to provide the full proof for our main convergence results. On the other hand, if we had seen the full proof of the results in Section 2.6, seeing also the one of the results of Section 3.7 would not be that interesting anymore, given that many parts of this proof again rely on techniques from [CCLLS21] and adaptations of these techniques first used in [BT20] or [BT21]. But there are also some substantial differences between the competition-induced dormancy model and the virus model. One issue is the richer and more complicated behaviour of the underlying dynamical system, even in absence of dormancy (and recovery). The other one is strongly connected to the phenomenon of contact-mediated dormancy, and we already mentioned it at the introduction of the approximating branching process*es*: The mean-matrix $\widetilde{J}$ of the full three-type branching process is not irreducible.

To adapt the techniques of [CCLLS21] and their previous adaptations, we need to work with a branching process that survives with positive probability from any initial condition with at least one positive coordinate, and this is the original branching process restricted to the infected and dormant coordinates. Regarding the first phase of the invasion, we then have the following analogue of Proposition 2.10 from the competition-induced dormancy model, where we define the stopping time

$$Q_\varepsilon = \inf\big\{t \geq 0\colon (N^K_{1a,t}, N^K_{1d,t}) \notin [\bar{n}_{1a}-\varepsilon, \bar{n}_{1a}+\varepsilon]\times[0,\varepsilon]\big\},$$

the first time when the rescaled type 1a population leaves a neighbourhood of radius ε around the equilibrium $\bar{n}_{1a}$ or the rescaled 1d population reaches size ε.

Proposition 3.18 ([BT23], partially based on methods of [CCLLS21])**.** *Assume that $\widetilde{\lambda} \neq 0$. Let $K \mapsto m^K_{1a}$ be a function from $(0,\infty)$ to $[0,\infty)$ such that $m^K_{1a} \in \frac{1}{K}\mathbb{N}_0$ and $\lim_{K\to\infty} m^K_{1a} = \bar{n}_{1a}$. Then there exists a constant $b \geq 2$ and a function $f\colon (0,\infty)\to(0,\infty)$ tending to zero as $\varepsilon \downarrow 0$ such that*

$$\limsup_{K\to\infty}\Big|\mathbb{P}\Big[T^2_\varepsilon < T^2_0 \wedge Q_{b\varepsilon}, \Big|\frac{T^2_\varepsilon}{\log K} - \frac{1}{\widetilde{\lambda}}\Big| \leq f(\varepsilon)\Big|\mathbf{N}^K_0 = \big(m^K_{1a},0,0,\frac{1}{K}\big)\Big] - (1-s_2)\Big| = o_\varepsilon(1) \tag{3.29}$$

and

$$\limsup_{K\to\infty}\Big|\mathbb{P}\Big[T^2_0 < T^2_\varepsilon \wedge Q_{b\varepsilon}\Big|\mathbf{N}^K_0 = \big(m^K_{1a},0,0,\frac{1}{K}\big)\Big] - s_2\Big| = o_\varepsilon(1). \tag{3.30}$$

λ_1	5	v	1.1
μ_1	4	μ_2	0.3
C	1	$n_{1a}(0)$	$1(=\bar{n}_{1a})$
κ	1	$n_I(0)$	0.1
q	0.1	$n_{1d}(0)$	$\pi_{1d} \cdot n_I(0)$
r	0.1	$n_{1i}(0)$	$\pi_{1i} \cdot n_I(0)$
D	0.5	σ	2

Table 3: Default choice of the parameters for the simulations of the dynamical systems (3.13) and (3.3), where $n_I(0) = n_{1d}(0) + n_{1i}(0) + n_2(0)$.

The most important change compared to Proposition 2.10 is the *change of roles in the invasion*. Now, even though naturally one would consider only type 1a as "resident" and types 1d, 1i, and 2 all as "invaders", due to the issues with the three-type branching process we will also consider type 1d as "resident", with its initial "equilibrium population size" being zero. We can then adapt the Freidlin–Wentzell type large-deviation techniques so as to guarantee that $N^K_{1d,t}$ stays close to 0 (while $N^K_{1a,t}$ stays near $\bar{n}_{1a}$) until $N_{1i,t} + N_{2,t}$ reaches εK or goes extinct. See [BT23, Section 4.3] for details.

3.9 Further simulations and conjectures related to the dynamical system

To gain an understanding of the concrete behaviour of the dynamical system (3.13) and to analyse the quantitative and qualitative effect of contact-mediated dormancy and its combination with recovery, we provide a few more simulations of the solutions for some concrete choices of the parameters, following the ones in Figure 1 for $q = 0$. The critical burst sizes m^*, m', m^{**} will have the same meaning as for the three-dimensional system (3.3), with $m^{**} = \infty$ if there is no Hopf bifurcation. We will work with the choice of parameters presented in Table 3 (abbreviating $n_I(0) := n_{1d}(0)+n_{1i}(0)+n_2(0)$), apart from those parameters that we vary in the given simulation.

Here, π_{1d}, π_{1i}, and π_2 are the dormant, infected, and virus coordinates of the coordinatewise positive ('Kesten–Stigum') left eigenvector of the mean matrix J associated to the eigenvalue $\widetilde{\lambda}$ normalized so that $\pi_{1d} + \pi_{1i} + \pi_2 = 1$ (cf. Section 2.8). Heuristically, the reason why this initial condition is natural is that for $\widetilde{\lambda} > 0$, conditional on survival of the approximating branching process $(\widehat{\mathbf{N}}(t))_{t\geq 0}$, the proportions of its dormant, infected, and virus coordinates converge to the corresponding proportions of $(\pi_{1d}, \pi_{1i}, \pi_2)$ thanks to the Kesten–Stigum theorem (Theorem 2.13).

Exercise 16. *Using the irreducibility of the mean matrix J_2, show that the assertion of the Kesten–Stigum theorem holds for the three-dimensional branching process with mean matrix $\widetilde{J}$ too (despite the lack of irreducibility $\widetilde{J}$).*

Example 3.19 (Varying r for fixed q). With the default choice of parameters apart from r, in Figure 2 we plot the transcritical bifurcation point m^*, the point m' where a pair of eigenvalues of the Jacobi matrix of (3.13) at $(\widetilde{n}_{1a}, \widetilde{n}_{1d}, \widetilde{n}_{1i}, \widetilde{n}_2)$ becomes complex, and the Hopf bifurcation point m^{**}, as functions of r. Note that the recovery-free case $r = 0$ is also included in the images (for this particular choice of q). Given that we have fixed all other parameters, m^* is a linear function of r (indeed, m^* corresponds to m in the case when we have an equality in (3.15)). Assuming that the assertions in Tables 1 and 2 hold true, we know that there is precisely one value of $m > m^*$, namely $m = m'$, where a pair of eigenvalues of the Jacobi matrix becomes complex, and for $m^{**} < \infty$ there is a unique value of $m > m'$, namely $m = m^{**}$, where these eigenvalues are purely imaginary, while for $m^{**} = \infty$ the real part of these eigenvalues remains negative for all $m > m'$. We evaluate m' and m^{**} in a discrete set of points, and we conclude that the dependency of m' on r also seems linear. As expected, there exists $r_0(q) > 0$ such that for $r > r_0(q)$, the Hopf bifurcation point m^{**} explodes and becomes infinite, i.e., $(\widetilde{n}_{1a}, \widetilde{n}_{1d}, \widetilde{n}_{1i}, \widetilde{n}_2)$ stays locally asymptotically stable for all $m > m^*$ (despite the fact that q is relatively small, while it is not necessarily small enough to deduce the lack of Hopf bifurcation for large r from the case $q = 0$ by continuity). In this case we have $r_0(q) \approx 0.69$, given that for r above

this value, as one increases m, the real parts of the two complex eigenvalues seem to converge to a strictly negative value. We know from Lemma 3.6 that such $r_0(0)$ also exists for $q = 0$ and we have $r_0(0) \leq v$. It is not included in the images, but its value is about 0.73 (so it is roughly two thirds of v).

Example 3.20 (Varying q for fixed r). With the default choice of parameters apart from q, in Figure 3 we plot m^*, m', m^{**} as functions of q. Note that the dormancy-free case $q = 0$ is also included in the images (for this particular choice of r). The equation (3.15) with an equality again gives an explicit formula for m^* as a function of q, which is finite for all $q \in [0, 1)$, monotone increasing in q, and tends to ∞ as $q \uparrow 1$. Also m' seems to only explode in the limit $q \uparrow 1$. In contrast, for $q = 0.93$, the Hopf bifurcation point m^{**} already seems to be infinite.

This provides numerical evidence that choosing the dormancy initiation probability $q \in (0, 1)$ sufficiently large eliminates the Hopf bifurcation, although recovery is relatively weak so that for $q = 0$ the Hopf bifurcation is present. It is not included in the images, but we also checked the recovery-free case $r = 0$ with otherwise unchanged parameters, and for $q = 0.97$ we also found $m^{**} = \infty$ there. In other words, dormancy can help avoid Hopf bifurcations even in the absence of recovery, albeit this may require q to be unrealistically high.

We complement our precise numerical results from Figure 3 with a schematic illustration of the critical burst sizes m^*, m', and m^{**} as functions of $q \in [0, 1)$ with all parameters but m and q fixed, for r small resp. large compared to v (in the left resp. right picture of Figure 4). Note that this illustration is based on our conjectures listed in Tables 1 and 2, while we have justified some of the properties of the curves $q \mapsto m^*(q), m'(q), m^{**}(q)$ for $q = 0$ in Section 3.4, and most of those results generalize to the case $q > 0$. The shape of the curves is not meant to be precise, but qualitatively correct, in particular we expect them to be convex (and thus lower semicontinuous) as $[0, \infty]$-valued functions.

Finally, let us comment on the case of quick resuscitation of dormant cells, that is, diverging σ.

Example 3.21 (Effects of large σ). The case of a very large σ corresponds to almost instantaneous resuscitation of dormant individuals after falling dormant. Thus, it is plausible to think that the qualitative behaviour of the active, infected, and virus coordinates of the system (3.13) behaves very similarly to the case where there is no dormancy but the parameter D of virus attacks is reduced by a factor of $1 - q$. In Figure 5 we consider a solution to (3.13) with the default choice of parameters, apart from σ which we choose as very large ($\sigma = 100$, as opposed to the default value $\sigma = 2$), also in comparison to the value of κ (being equal to 1). We see that the behaviour of this solution is very similar to the one of (3.3) with the same initial condition and with the same choice of the parameters apart from q being altered to 0 and D to $(1 - q)D$.

4 The polynomial mutation regime and its piecewise affine scaling limits

4.1 Introduction: different mutation regimes and horizontal gene transfer

Let us recall that the key features of the rare mutation regime of adaptive dynamics characterized $u_K \ll \frac{1}{K \log K}$ (plus the lower bound in (1.9)) are the following, assuming that the competitive advantage of the mutants is independent of K (the analogue of which is referred to as *strong selection* in the population-genetic context).

(I) *Clonal interference plays no substantial role*: A mutant subpopulation that eventually becomes resident sees typically no other mutant subpopulation before its fixation. If a mutant ever becomes resident, it will also fix or reach long-term coexistence with some of the former resident traits eventually.

(II) *Unique potential parent*: Most of the time there is a unique resident, and every birth with mutation originates from the current resident with high probability.

(III) *Random genetic drift plays a significant role*: The probability that a beneficial mutation becomes resident/fixes stays below one in the limit $K \to \infty$. However, stochasticity is only important at the

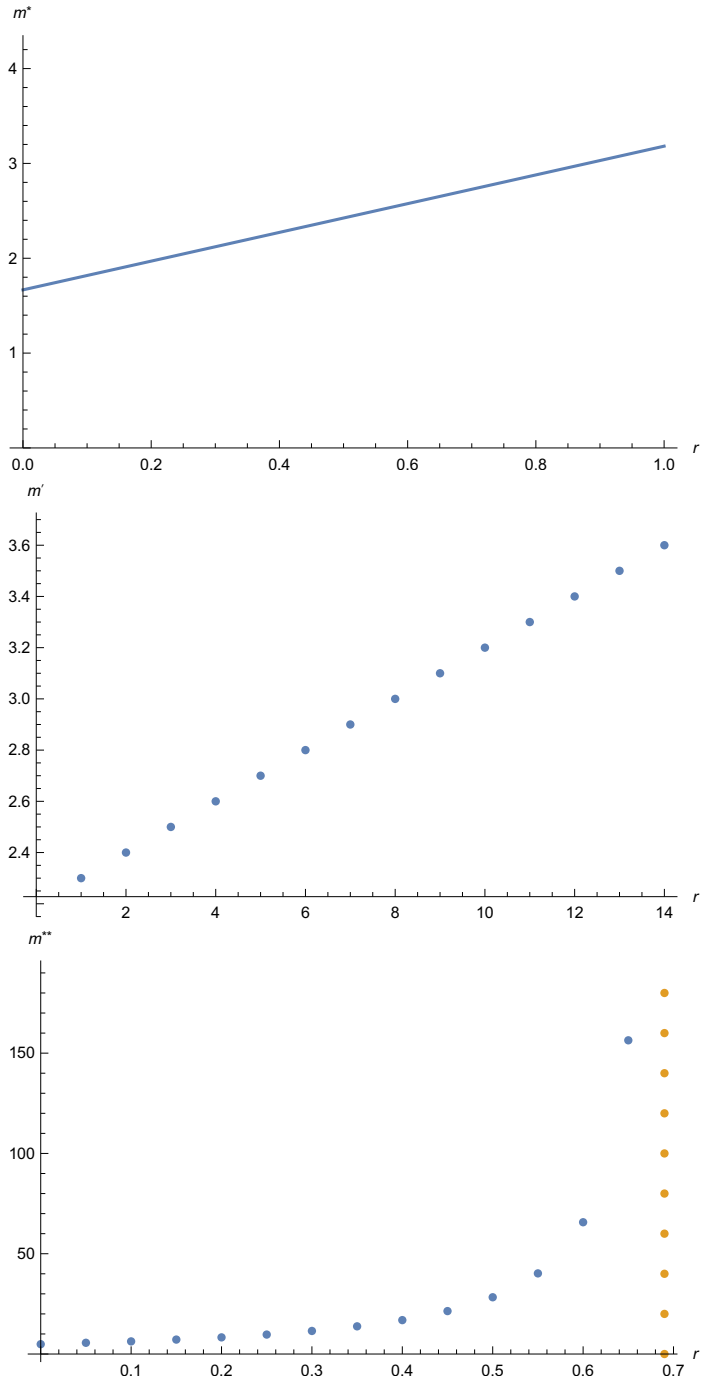


Figure 2: Values of the critical burst sizes m^*, m^{**}, m' as functions of r with all other parameters (in particular q) fixed. At $r \approx 0.69$ (orange dotted line) m^{**} explodes and becomes infinite.

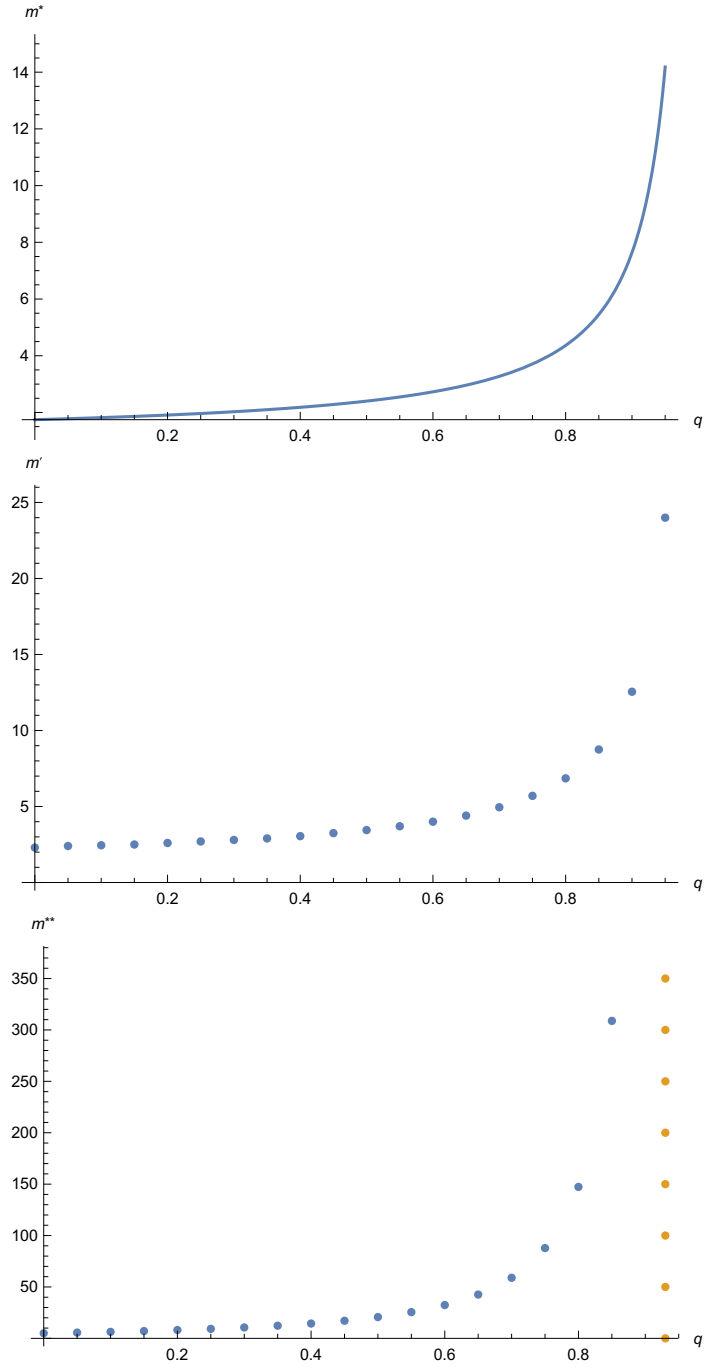
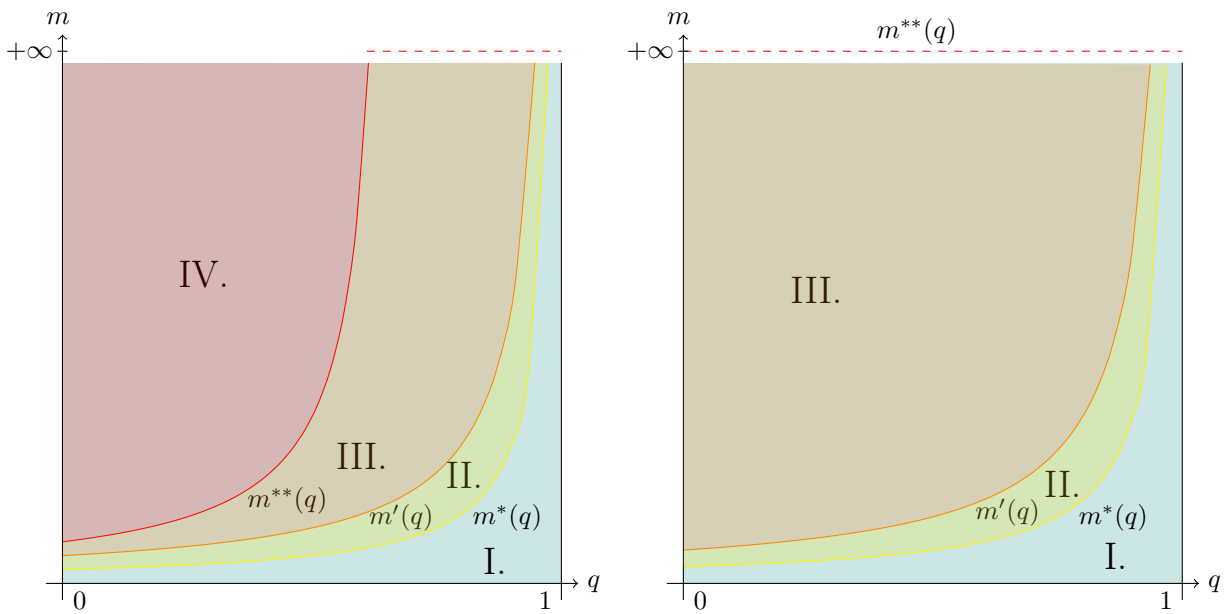


Figure 3: Values of the critical burst sizes m^*, m', m^{**} as functions of q with all other parameters (in particular r) fixed. At $q \approx 0.93$ (orange dotted line), m^{**} explodes and becomes infinite.



Region	I.	II.	III.	IV.
Characterization	$0 < m < m^*(q)$	$m^*(q) < m < m'(q)$	$m'(q) < m < m^{**}(q)$	$m > m^{**}(q)$
Stability of $(\bar{n}_{1a},0,0,0)$	stable	unstable	unstable	unstable
Existence of $(\widetilde{n}_{1a},\widetilde{n}_{1d},\widetilde{n}_{1i},\widetilde{n}_2)$	does not exist	exists	exists	exists
Stability of $(\widetilde{n}_{1a},\widetilde{n}_{1d},\widetilde{n}_{1i},\widetilde{n}_2)$	-	stable	stable	unstable
Asymptotic behaviour of positive solutions to (3.13)	eventually coord. monotone convergence to $(\bar{n}_{1a},0,0,0)$	eventually coord. monotone convergence to $(\widetilde{n}_{1a},\widetilde{n}_{1d},\widetilde{n}_{1i},\widetilde{n}_2)$	oscillatory convergence to $(\widetilde{n}_{1a},\widetilde{n}_{1d},\widetilde{n}_{1i},\widetilde{n}_2)$	periodic behaviour

Figure 4: Left: The case when r is small compared to v (e.g., $r = 0$). The Hopf bifurcation point m^{**} reaches $+\infty$ at some value $q \in (0,1)$, Right: If r is large compared to v, $m^{**}(q) = \infty$ holds for all $q \in [0,1)$, thus $(\widetilde{n}_{1a}, \widetilde{n}_{1d}, \widetilde{n}_{1i}, \widetilde{n}_2)$ is stable for all $m > m^*(q)$. In both cases, m^* only diverge as $q \uparrow 1$, and the same seems true for m'. In the coloured regions, we expect the behaviour explained in the tabular below the images.

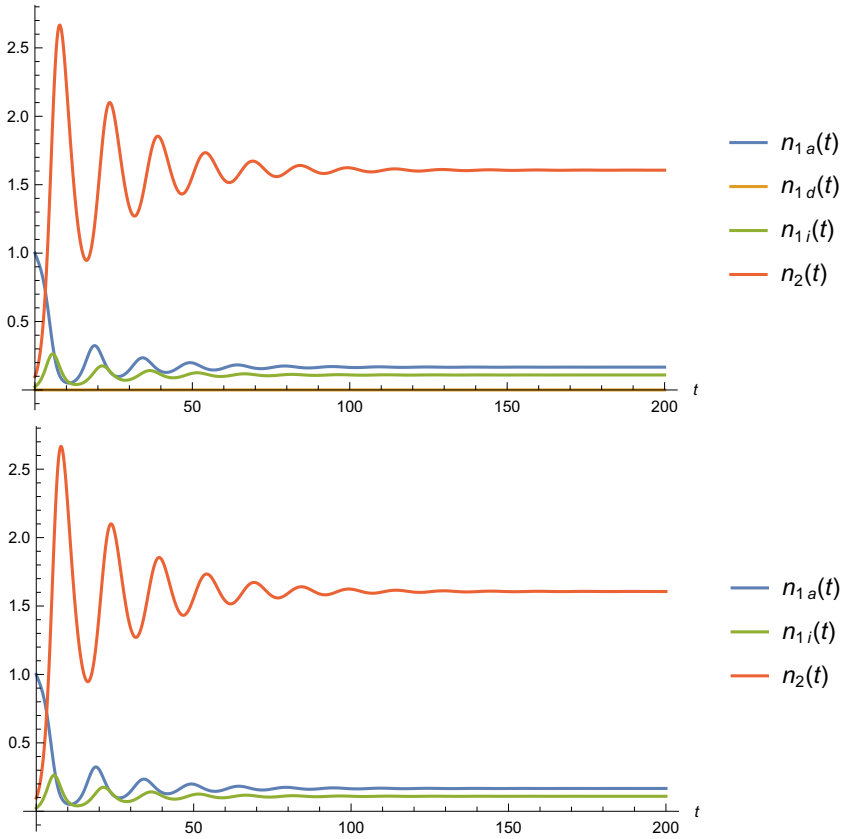


Figure 5: Solution to (3.13) with large σ ($\sigma = 100$) (top) and the one of the corresponding solution to (3.3) (bottom), for $m = 5$. In the solution to (3.3) there is no dormant coordinate, whereas the dormant coordinate of the solution to (3.13) stays very close to zero.

very beginning of Phase I of an invasion (which is followed by exponential mutant growth in the case of survival).

(IV) *No mutants of mutants*: Before a mutant subpopulation fixes, with high probability none of its individuals suffer an additional mutation.

What happens if the mutation rate is higher?

- If $u_K \asymp \frac{1}{K \log K}$, the duration of invasions of mutants surviving random genetic drift (i.e., initial fluctuations) will be of the same order $\Theta(\log K)$ as the typical waiting time between two mutations surviving drift. Hence, clonal interference will play a crucial role. Even in the case of transitive competitive relations, mutants may outcompete each other, and thus surviving drift will not imply ever becoming resident, while ever becoming resident will also not imply fixation (i.e. all individuals in the population carrying the mutation from some point in time on). Still, the "no mutants of mutants" and the "unique potential parent" assertions will remain true. The total mutation rate in the entire population still tends (slowly) to zero. Such a behaviour was conjectured in [BS19].

- The case $u_K \asymp 1/K$ was analysed in [S17]. In this model, mutations with *the same* selective advantage occur repeatedly, as well as mutations back from this mutant type to the wild type, and hence among the arising mutant families, evolution acts neutrally. The author showed that asymptotically as $K \to \infty$, there is a countably infinite family of potential parents for each mutation. However, there are still no mutants of mutants. The total mutation rate in the population is essentially constant in K.

- Assume now that $u_K \asymp K^{-\alpha}$ for some $\alpha \in (0, 1)$. Then, the mutation rate per individual still tends to zero but the total mutation rate in the population diverges. Now, if we assume a fixed countable set of traits and a fixed *mutation graph* telling which trait can mutate towards which trait, we will observe that mutant subpopulations of order K^β, $\beta > \alpha$ already give rise to mutants of mutants whose order is increasing polynomially in K, their amount is about the order $K^{\beta-\alpha}$.
  To analyse this process, we will look at the *logarithms* of subpopulation sizes. This transformation makes exponential growth/decay linear, and thus we will obtain a *piecewise affine* scaling limit as $K \to \infty$ via a suitable scaling of time and subpopulation sizes. In particular, the "continuous flow of mutations" will wipe out the effect of random genetic drift, and the scaling limit will be deterministic. Such a scaling limit was first obtained by Durrett and Mayberry [DM11] in a population-genetic model with a very similar choice of parameters (but with a constant or exponentially growing total population size).[31]

In this section we will study the lastly mentioned mutation regime, the so-called *polynomial* or *power-law* mutation regime[32], with the additional effect of *horizontal gene transfer* (HGT), following the paper by Champagnat, Méléard, and Tran [CMT21]. Horizontal gene transfer is a phenomenon where non-parental individuals of a bacterial population exchange genetic information during their lifetimes. Horizontal gene transfer is a main factor of bacterial evolution, which helps the spread of beneficial genes within the population, instead of e.g. a sexual reproduction, which is absent in their case. HGT can help e.g. the spread of a bacterial epidemic and the development of antibiotic resistances. For mathematical models involving HGT in the rare mutation regime prior to [CMT21], we refer the reader to [BCFMT16, BCFMT18].

Remark 4.1. In [BT21] we studied an extension of the competition-induced dormancy model that we saw in Section 2, where the subpopulation lacking dormancy can additionally transform individuals of the dormancy trait to ones lacking this trait via transferring additional genetic material to them. Due to the particular form of HGT studied there (which is somewhat different from the one introduced in [CMT21] that we will get to know in this section), HGT can in some cases change the direction of the evolution. Indeed, there

[31] Let us note that e.g. for $u_K \asymp 1/(K \log K)$ one can also obtain a piecewise affine scaling limit of the logarithmic population sizes, but this will be random due to the effect of drift, and also due to the sizes of mutations in case they are random.

[32] Since the probability of a mutation during birth still tends to zero in this regime, it is somewhat confusing not to call mutations rare here. Some authors therefore (rightfully) call what we and many other authors colloquially called the rare mutation regime here the regime of *very rare* mutations, see e.g. [EK23].

are some parameter regimes when instead of the fixation of the dormancy trait, the other trait coexists with it or even makes it go extinct. Stable coexistence of the two traits is in some cases possible even when the trait benefiting from HGT is not fit enough to survive when being on its own. Further, there are instances of *founder control* (a term borrowed from ecology), i.e. fixation of the initial resident and extinction of the invader regardless of their types. *Addendum to Exercise 3: Are there cases of founder control in the setting of the exercise?*

4.2 The Champagnat–Méléard–Tran model

The authors of [CMT21] studied a stochastic individual-based model for a population with individuals characterized by some trait. The population features asexual and haploid reproduction (binary fission, as before), death by age and due to logistic competition, one-sided transfer of traits between individuals. $K > 0$ will denote the carrying capacity, as usual. The trait space is a grid of mesh size $\delta > 0$ of $[0, 4]$: $\mathcal{X} = [0, 4] \cap \delta N_0 = \{0, \delta, \dots, L\delta\}$, where $L = \lfloor 4/\delta \rfloor$. The population is described by the vector

$$(N_0^K(t), \dots, N_\ell^K(t), \dots, N_L^K(t))$$

where $N_\ell^K(t)$ is the number of individuals of trait $x = \ell\delta$ at time $t \geq 0$. We define the total population size N_t^K as

$$N_t^K = \sum_{\ell=0}^{L} N_\ell^K(t).$$

The population process then evolves as a continuous-time Markov chain with the following transition rates.

- An individual with trait $x = \ell\delta$ gives birth to another individual with birth $b(x) = 4 - x$. With probability
$$p_K = K^{-\alpha} \qquad \text{with } \alpha \in (0, 1),$$
a mutation occurs and the offspring carries the mutant trait $(\ell+1)\delta$. With probability $1-p_K = 1-K^{-\alpha}$, the new individual inherits the ancestral trait.

- An individual with trait x transfers its trait to a given individual of trait y in a population of total size N at rate
$$\tau(x, y, N) = \frac{\tau}{N} \mathbb{1}_{\{x>y\}},$$
for some parameter $\tau > 0$.

- An individual with trait $x = \ell\delta$ in a population of total size N dies with natural death rate $d_K(x, N) = 1 + \frac{CN}{K}$, where $C > 0$.

Remark 4.2 (Interpretation of the trait value). The trait x may be interpreted in the biological setting as a phenotypic value quantifying e.g. the pathogenic strength of bacteria or their antibiotic resistance. Such a setting was first studied in [BCFMT18] (in the regime of rare mutations). However, direct advantages of a higher trait do not appear in the model. The trait x may be assumed to be related to the quantity of plasmids held by a bacterium. At a HGT event, the donor transfers plasmids to the recipient, thus increasing its trait. This explains why an individual can only be a recipient if the donor has a higher trait value. The recipient receives the donor trait, which is called *conjugation* in the biological setting (see [CMT21, Section 1] and the references therein). Reproduction favours small values of x, a reason for which could be that the copying of plasmids and their maintenance requires additional resources, which could otherwise be used e.g. for better reproduction. This explains why reproduction favours small values of x. Finally, mutation also increases the value of the trait, which is beneficial from the point of view of HGT but disadvantageous from the point of view of reproduction.

Remark 4.3 (Unfit traits and evolutionary suicide). Traits $x > 3$ are not fit enough to survive when being on their own since they satisfy $b(x) - d(x) < 0$. We will see that there are scenarios when such a trait can become the trait with the largest population of the system, so that the total population starts decaying exponentially. If no other trait can take back the lead from this trait, the entire population will become extinct, which we will refer to as *evolutionary suicide*. Such a phenomenon was also observed in [BCFMT18], whereas in the setting of [BT21] (without mutations, cf. Remark 4.1) it cannot occur.

Remark 4.4. Because of the factor $1/K$, competition is governed only by traits with population size of order K. Therefore, density-dependence of the death rate disappears when the total population size is negligible with respect to K.

We need to consider in the sequel two different situations: Either there is a unique trait x with population size of order K, which we will call the *resident* trait, or the total population size is $o(K)$, mostly consisting of one trait. In the latter case, the trait with the largest population size will be called the *dominant* trait.

When the trait x is the (unique) resident trait, we know from Section 1 (cf. [EK86]) that as $K \to \infty$, the total population size divided by K can be approximated on compact time intervals by $(n(t))_{t\geq 0}$, which is the unique solution to the ODE

$$\dot{n}(t) = n(t)(3 - x - Cn(t)),$$

which has the unique nonnegative and stable equilibrium

$$\bar{n}(x) = \frac{(3-x)\vee 0}{C}.$$

The notion of *invasion fitness* that we know from Section 1 can be adapted to our setting. The invasion fitness of a mutant individual of trait y in a resident population of trait x is given by

$$S(y;x) = b(y) - d_K(y, K\bar{n}(x)) + \tau \mathbb{1}_{\{x<y\}} - \tau \mathbb{1}_{\{x>y\}} = x - y - \tau \operatorname{sgn}(y-x). \tag{4.1}$$

Indeed, the total transfer rate from x to y is given by $\frac{K\bar{n}(x)\tau}{K\bar{n}(x)+1}\mathbb{1}_{\{y>x\}} \sim \tau \mathbb{1}_{\{y>x\}}$ when $K \to \infty$, and similarly from y to x. Note that $S(x;x) = 0$, and for all $x, y \in \mathcal{X}$, $S(y;x) = -S(x;y)$. This implies in particular that there is no long-term coexistence of two resident traits. We also define the fitness of an individual of trait y in a population of negligable size ($o(K)$) with dominant trait x. As already mentioned, in this case the density-dependency vanishes, and since $N_x^K(t)/N_t^K \approx 1$, the fitness is now

$$\widehat{S}(y;x) = 3 - y + \tau \mathbb{1}_{\{x<y\}} - \tau \mathbb{1}_{\{x>y\}}. \tag{4.2}$$

The study of [CMT21] of the evolutionary dynamics of the model is based on a fine analysis of the order of magnitude of size, as power of K, of each subpopulation corresponding to the different trait compartments. These powers of K evolve on the time scale $\log K$, as we already saw in the case of branching processes in Section 1.9, but we will also cite a finer result in this direction later (see Lemma 4.12). We define $\beta_\ell^K(t)$ for $0 \leq \ell \leq L$ such that

$$N_\ell^K(t \log K) = K^{\beta_\ell^K(t)-1}, \qquad \text{i.e.} \qquad \beta_\ell^K(t) = \frac{\log\big(1 + N_\ell^K(t\log K)\big)}{\log K}. \tag{4.3}$$

We assume that the trait $x = 0$ is initially resident, with density (i.e. population size normalized by K) equal to $3/C$. The initial condition is

$$N^K(0) = \Big(\lfloor \frac{3K}{C} \rfloor, \lfloor K^{1-\alpha} \rfloor, \dots, \lfloor K^{1-\ell\alpha} \rfloor, \dots, \lfloor K^{1-\lfloor 1/\alpha \rfloor \alpha} \rfloor, 0, \dots, 0\Big).$$

Due to mutations, this is close (on the logarithmic scale) to the population state reached instantaneously on the time scale $\log K$ when the initial population is only composed of $\lfloor \frac{3K}{C} \rfloor$ individuals with trait 0.[33] With this initial condition, we have

$$\beta_\ell^K(0) \underset{K\to\infty}{\longrightarrow} (1 - \ell\alpha)\mathbb{1}_{\{0\leq \ell \leq 1/\alpha\}}. \tag{4.4}$$

[33] For a precise formulation and proof of the corresponding assertion, see [CMT21, Lemma B.4].

The main result of [CMT21], Theorem 4.5 below, provides the asymptotic dynamics of the system of functions $\beta_\ell^K(t)$ when $K \to \infty$. This limit is a system of deterministic, continuous, piecewise affine functions, which can be described along successive phases determined by their resident or dominant traits. When the resident/dominant trait changes, the fitnesses governing the slopes are modified. Moreover, inside each phase, other *kinks*, i.e. changes of slopes are possible due to a delicate balance between mutations, transfer, and growth of subpopulations. The aim of [CMT21] was to cover all the possible cases: local extinctions of single traits[34], re-emergence of subpopulations, changes of slopes due to mutation and selection (competition), dynamics when the total population size is $o(K)$, extinction of the total population... The paper provided from the asymptotic dynamics of $\beta^K(t)$ explicit criteria for the occurrence of the different evolutionary outcomes (see Theorem 4.11 below) and provided a detailed study of the case of three traits. We now present the main results on the paper and then we provide an outline of its proof.

4.3 The main convergence result of [CMT21]

The first result characterizes the asymptotic dynamics of $(\beta^K(t))_{t\geq 0} = (\beta_0^K(t), \dots, \beta_L^K(t))_{t\geq 0}$ (when $K \to \infty$) by a succession of deterministic time intervals $[s_{k-1}, s_k]$, $k \geq 1$, called phases and delimited by changes of resident or dominant traits. The latter are unique except at times s_k and denoted by $\ell_k^*\delta$, $k \geq 1$. The asymptotic result holds until a possibly infinite stopping time T_0, which guarantees that there is neither ambiguity on the resident/dominant traits (Point (a) below) nor on the extinct subpopulations at the phase boundaries (Point (c) below). See Figure 6 for simulations of the limiting process for three different choices of the parameters.

Theorem 4.5 ([CMT21])**.** *Assume that $\alpha \in (0,1)$, $\delta \in (0,4)$, $3/\delta \notin \mathbb{N}$, $\frac{\tau+3}{\delta} \notin \mathbb{N}$, and (4.4) holds.*

(i) For all $T > 0$, the sequence $(\beta^K(t))_{t\in[0,T\wedge T_0]}$ converges in probability in $\mathbb{D}([0, T\wedge T_0], [0,1]^L)$ to a deterministic piecewise affine continuous function $\beta = (\beta_\ell)_{0\leq\ell\leq[L]} = (\beta(t))_{t\geq 0} = (\beta_1(t), \dots, \beta_L(t))_{t\in[0,T\wedge T_0]}$, such that $\beta_\ell(0) = (1-\ell\alpha)\mathbb{1}_{\{0\leq\ell\leq 1/\alpha\}}$. The functions β and $T-0$ are parameterized by α, δ, and τ, defined as follows.

(ii) There exists an increasing nonnegative sequence $(s_k)_{k\geq 0}$ and a sequence $(\ell_k^*)_{k\geq 1}$ in $\{0, \dots, L\}$ defined inductively as follows: $s_0 = 0$, $\ell_1^* = 0$, and for all $k \geq 1$, assuming that $s_{k-1} < T_0$ and ℓ_k^* have been constructed and that $\beta(s_{k-1}) \neq 0$, we can construct $s_k > s_{k-1}$ as follows:

$$s_k = \inf\{t > s_{k-1} \colon \exists \ell \neq \ell_k^* \colon \beta_\ell(t) = \beta_{\ell_k^*}(t)\}.$$

We then decide whether we continue the induction after time s_k (i.e. $T_0 > s_k$) or not as follows:

(a) If $\beta_{\ell_k^*}(s_k) > 0$, we set

$$\ell_{k+1}^* = \arg\max_{\ell\neq\ell_k^*} \beta_\ell(s_k)$$

if the arg max *is unique, or otherwise we set $T_0 = s_k$ and we stop the induction;*

(b) if $\beta_{\ell_k^*}(s_k) = 0$, we set $s_{k+1} = T_0 = \infty$ and $\beta(t) = 0$ for all $t \geq s_k$;

(c) if in one of the previous cases, we have for some $\ell \neq \ell_k^*$ that $\beta_\ell(s_k) = 0$ and $\beta_\ell(s_k - \varepsilon) > 0$ for all $\varepsilon > 0$ small enough, then we also set $T_0 = s_k$ and stop the induction; otherwise, the induction proceeds to the next step.

(iii) In (ii), the functions β_ℓ are defined, for all $t \in [s_{k-1}, s_k]$, by

$$\beta_0(t) = \Big[\mathbb{1}_{\{\beta_0(s_{k-1})>0\}}\Big(\beta_0(s_{k-1}) + \int_{s_{k-1}}^t \widetilde{S}_{s,k}(0; \ell_k^*\delta)\mathrm{d}s\Big)\Big] \vee 0 \tag{4.5}$$

[34] Traits $\ell\delta$ for $\ell > 0$ may go extinct and get "resurrected" via incoming mutations from their left neighbour $(\ell-1)\delta$; this is what we mean by local extinctions. In contrast, if trait 0 ever goes extinct, it will be lost forever.

and, for all $\ell \in \{1, L\}$,

$$\beta_\ell(t) = \Big(\beta_\ell(s_{k-1}) + \int_{t_{\ell-1,k}}^{t} \widetilde{S}_{s,k}(\ell\delta; \ell_k^*\delta)\mathrm{d}s\Big) \vee (\beta_{\ell-1}(t) - \alpha) \vee 0, \tag{4.6}$$

where, for all traits $x, y \in \mathcal{X}$,

$$\widetilde{S}_{t,k}(y; x) = \mathbb{1}_{\{\beta_{\ell_k^*}(t)=1\}} S(y; x) + \mathbb{1}_{\{\beta_{\ell_k^*}(t)<1\}} \widehat{S}(y; x) \tag{4.7}$$

and

$$t_{\ell-1,k} = \begin{cases} \inf\{t \geq s_{k-1} \colon \beta_{\ell-1}(t) = \alpha\}, & \text{if } \beta_\ell(s_{k-1}) = 0, \\ s_{k-1}, & \text{otherwise.} \end{cases}$$

In addition, for all ℓ *and* $a < b < T_0$ *such that the time interval* $[a, b]$ *is included in the interior of the zero set of* β_ℓ*, we have*

$$\lim_{K\to\infty} \mathbb{P}\big(N_\ell^K(t \log K) = 0, \forall t \in [a, b]\big) = 1.$$

Remark 4.6. 1. It follows from the definition of s_k and ℓ_{k+1}^* that $\max_\ell \beta_\ell(t) = \beta_{\ell_k^*}(t)$ for all $t \in [s_{k-1}, s_k)$.

2. In (4.7), when $\beta_{\ell_k^*}(t) = 1$ for $t \in (s_{k-1}, s_k)$, there is a single resident trait $\ell_k^*\delta$ with population size of order K and the function S defined in (4.1) is used. In the case where $\beta_{\ell_k^*}(t) < 1$, there is a single dominant trait, the total population size is of order $o(K)$, and the fitness function is $\widehat{S}$ defined in (4.2). During each phase, the function $\widetilde{S}_{t,k}$ is actually constant, equal to S or $\widehat{S}$ as above, expect when a dominant population becomes resident in the same phase. In the first case, for all $t \in [s_{k-1}, s_k)$, Equations (4.5) and (4.6) take the simpler form

$$\beta_0(t) = \begin{cases} \big[\mathbb{1}_{\{\beta_0(s_{k-1})>0\}}\big(\beta_0(s_{k-1}) + S(\ell\delta; \ell_k^*\delta)(t - s_{k-1})\big)\big] \vee 0, & \text{if } \beta_{\ell_k^*}(s_{k-1}) = 1, \\ \big[\mathbb{1}_{\{\beta_0(s_{k-1})>0\}}\big(\beta_0(s_{k-1}) + \widehat{S}(\ell\delta; \ell_k^*\delta)(t - s_{k-1})\big)\big] \vee 0 & \text{if } \beta_{\ell_k^*}(s_{k-1}) < 1 \end{cases}$$

and for all $\ell \in [L]$,

$$\beta_\ell(t) = \begin{cases} \big(\beta_\ell(s_{k-1}) + S(\ell\delta; \ell_k^*\delta)(t - t_{\ell-1,k})_+\big) \vee (\beta_{\ell-1}(t) - \alpha) \vee 0 & \text{if } \beta_{\ell_k^*}(s_{k-1}) = 1, \\ \big(\beta_\ell(s_{k-1}) + \widehat{S}(\ell\delta; \ell_k^*\delta)(t - t_{\ell-1,k})_+\big) \vee (\beta_{\ell-1}(t) - \alpha) \vee 0 & \text{if } \beta_{\ell_k^*}(s_{k-1}) < 1. \end{cases}$$

Otherwise, $\widetilde{S}_{t,k}$ switches from $\widetilde{S}$ to S at the first time when $\max_{\ell'} \beta_{\ell'}(t) = \beta_{\ell_k^*}(t) = 1$. Therefore, since $S(\ell_k^*\delta, \ell_k^*\delta) = 0$, we obtain in all cases

$$\beta_{\ell_k^*}(t) = \begin{cases} 1, & \text{if } \beta_{\ell_k^*}(s_{k-1}) = 1, \\ \Big[\big(\beta_{\ell_k^*}(s_{k-1}) + \widehat{S}(\ell_k^*\delta; \ell_k^*\delta)(t - s_{k-1})\big) \wedge 1\Big] \vee 0, & \text{if } \beta_{\ell_k^*}(s_{k-1}) < 1. \end{cases}$$

3. It follows from the previous formula that $\max_\ell \beta_\ell(t) \leq 1$ for all $t \in [0, T_0)$ (and even for $t = T_0$ if $T_0 < \infty$).

4. When $\beta_\ell(s_{k-1}) = 0$, the time $t_{\ell-1,k}$ corresponds to the first time when the incoming mutation rate in subpopulation $\ell\delta$ (coming from subpopulation $(\ell-1)\delta$) becomes significant. Therefore, for $\ell = 0$ there is no such time defined.

5. Note that Theorem 4.5 keeps track of populations of size K^β for $0 < \beta \leq 1$, but not of populations of smaller order, which go fast to extinction on the time scale $\log K$.

It is instructive to look at [CMT21, Section 3], where the process $\beta(t)$ is studied in detail in the case of three traits $0, \delta, 2\delta$. There, one can see examples of:

1. In case $\tau < \delta$, there is *no evolution*: Neither δ nor 2δ is advantageous compared to 0, and therefore trait 0 will stay resident forever.

2. For $\tau > \delta$, there will be multiple phases. Possible scenarios are:

   (a) fixation of one of the traits, i.e. one trait stays resident forever,

   (b) *evolutionary cyclic behaviour*, i.e. after some time, $\beta(t)$ becomes periodic (and not constant) in each coordinate. See e.g. Figure 6 (a). There are cases when this occurs in such a way that there is always a resident trait, but it is also possible that there are (possibly also infinitely often repeated) phases where there is only a dominant trait, and in that case, trait δ may go extinct and then arise again thanks to incoming mutations from trait 0 (see Figure 6 (c)),

   (c) evolutionary suicide (see e.g. Figure 6 (b)).

Exercise 17. *What are the possible evolutionary outcomes in the case of two traits ($L = 1$)? At most how many times can the resident change? Is cyclic behaviour possible? Is evolutionary suicide possible?*

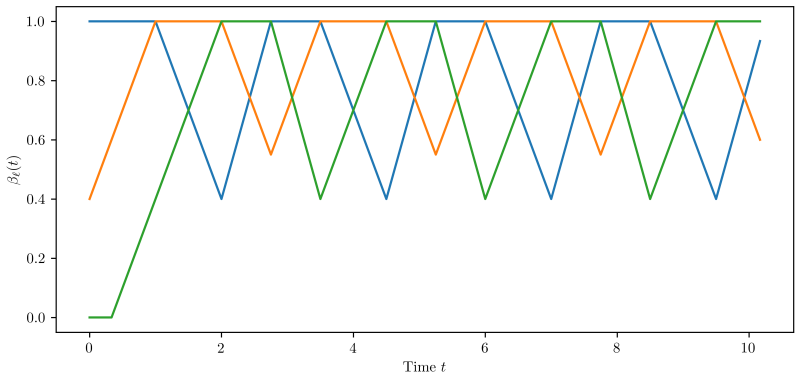
With a larger number of traits, it was also observed in [CMT21] numerically that the system may show cyclic but not periodic behaviour (see [CMT21, Figure 2.1 (c)]).

Remark 4.7. Evolutionary cyclic behaviour implies infinitely many resident changes in $\beta(t)$ given finitely many traits. As already pointed out in Remark 1.11, such a behaviour is impossible if the mutation rate per individual is $o(1/K \log K)$, but it may occur when it is weakly asymptotically equivalent to $1/K \log K$ (cf. the "rock–paper-scissors cycle" in [BS17] and the speculations of [BS19] about the case of multiple mutations, which were mentioned in Remark 1.11). What is interesting here is that the cyclic behaviour is not due to asymmetric competition (like in [BS17, BS19]) but to an interplay of the advantage of higher traits due to horizontal gene transfer and incoming mutations and the one of lower traits due to better reproduction. Symmetric competition with additional horizontal gene transfer is in fact not very far from asymmetric competition, and in different settings it may also lead to a coexistence between different traits (see e.g. [BT21]).

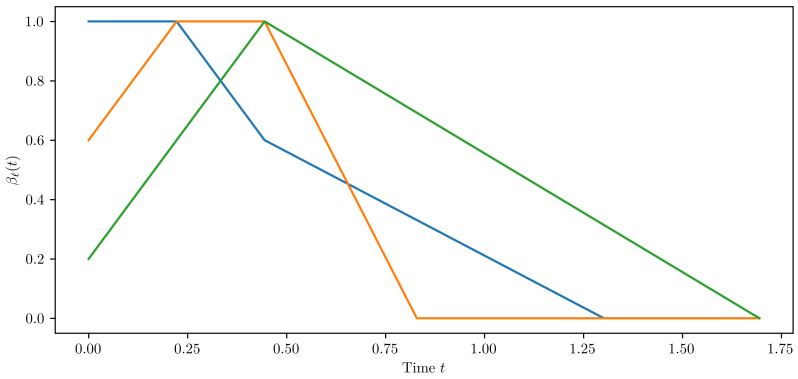
It is not clear whether $T_0 = +\infty$ holds for almost all parameters α, δ, and τ. However, the authors of [CMT21] performed simulations of the limiting process (see [CMT21, Appendix D] for a description of the algorithmic construction of the slopes $\beta_\ell(t)$), and they did not encounter any case when $T_0 < \infty$ in the simulations. In Theorem 4.11 below, we will see that T_0 is larger than the time of extinction or of first *re-emergence* (i.e. returning to residency, see below for a precise definition) of a trait.

Remark 4.8. Coquille, Kraut, and Smadi [CKS21] studied a model similar to the one of [CMT21], on a general *mutation graph*, i.e. with a general finite trait space as vertex set and the possible mutation directions between the traits as the set of (directed) edges. Their model did not contain horizontal gene transfer, which excludes evolutionary suicide and even the existence of phases where there is no resident trait, but in chance, they also considered asymmetric competition, which gives rise to coexistence between multiple traits (which does not occur in the original model of [CMT21]). In [CKS21, Theorem 3.6] they observed an interesting example where the induction goes through over all $k \in \mathbb{N}$ because the stopping conditions in Theorem 4.5, part (ii) cases (a) and (c) do not occur, and the resident traits follow each other (eventually) cyclically, but the times of resident changes s_k accumulate towards a *finite* time T_0. That is, in the scaling limit, the whole history of the population happens within a finite time interval. Certainly, this is not possible for the original individual-based model, and thus the convergence to $\beta(t)$ is not valid for $t \geq T_0$.

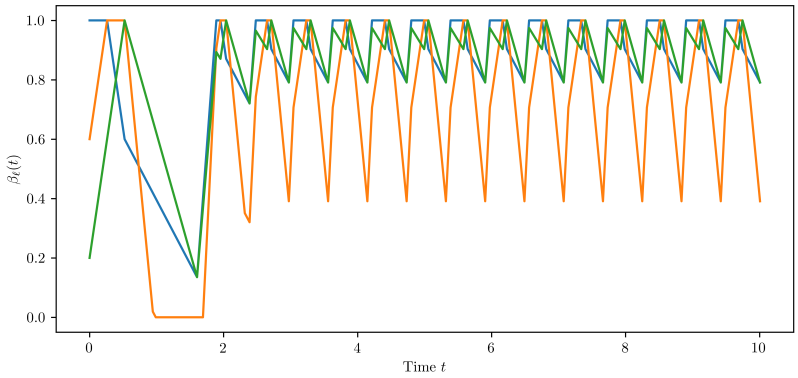
It is still generally believed that in the model of [CMT21], such an accumulation should not occur. However, it does occur in an extension of the model where competition-induced dormancy is incorporated along the lines of Section 2 studied in the paper [BPT23]. In one example ([BPT23, Example 3.2]) it was shown rigorously that the times of resident changes accumulate towards a point $T_0 < \infty$. In fact, the fact that time points where the slopes change may have a finite accumulation was already observed in the model of [DM11], see Lemma 1 of that paper. Moreover, [BPT23, Example 3.7] shows (at least numerically) that it is also possible that T_0 is an accumulation point of times of changes of *dominant* traits



(a)



(b)



(c)

Figure 6: The processes $\beta_0(t)$ (blue), $\beta_1(t)$ (orange), and $\beta_2(t)$ (green) for different choices of the parameters with three traits ($L = 2$). (a): $\delta = 1.4, \alpha = 0.6, \tau = 2$. We see a periodic behaviour showing re-emergences of all traits, in particular also of trait 0, the fittest one. (b): $\delta = 1.9, \alpha = 0.4, \tau = 3.7$. The population is directly driven to evolutionary suicide. (c): same choice of parameters as in the middle, apart from the fact that τ now equals 3.43. When trait $2\delta = 3.8 > 3$ becomes dominant, the population size is of order $o(K)$. We see a re-emergence of trait 0 after a phase of apparent macroscopic extinction (i.e. a total population size of $o(K)$). Although trait δ goes extinct while 2δ is dominant, it is recreated by mutations from trait 0. The choices of parameters (a), (c) with corresponding images first appeared in [CMT21]. Here we reproduced them via a code written by Tobias Paul, which was also used for creating the simulations in his master's thesis and the related paper [BPT23].

and $\max_\ell \limsup_{t\uparrow T_0} \beta_\ell(t) < 1$. In this case, the total population may be considered as unfit since it is not able to maintain a size of order K at least periodically.

In the model of [BPT23], there are also choices of parameters (see e.g. [BPT23, Example 3.3]) where T_0 seems infinite, but the behaviour of the system does not only look non-periodic (like in [CMT21, Figure 2.1 (c)] in the original model), but it is not even clear if the consecution of residents ever becomes cyclic.

4.4 Analytical and biological properties of the piecewise affine limiting process

The next theorem characterizes β as solution of a dynamical system.

Theorem 4.9 ([CMT21]). *Under the assumptions of Theorem 4.5, we set*

$$\ell^*(t) = \sum_{k\geq 1} \ell_k^* \mathbb{1}_{[s_{k-1},s_k]}(t) \text{ and } \widetilde{S}_t(y;x) = \mathbb{1}_{\{\beta_{\ell^*(t)}=1\}} S(y;x) + \mathbb{1}_{\{\beta_{\ell*(t)}<1\}} \widehat{S}(y;x).$$

The function $\beta(t)$ is right-differentiable on $[0,T_0)$ and satisfies

$$\dot{\beta}_\ell(t) = \Sigma_\ell(t) \mathbb{1}_{\{\beta_\ell(t)>0 \text{ or } (\beta_\ell(t)=0 \text{ and } \beta_{\ell-1}(t)=\alpha)\}}, \tag{4.8}$$

where Σ_ℓ is defined recursively by $\Sigma_0(t) = \widetilde{S}_t(0;\delta\ell^*(t))$ and $\forall l \geq 1$

$$\Sigma_\ell(t) = \begin{cases} \widetilde{S}_t(\ell\delta;\ell^*(t)\delta) \vee \Sigma_{\ell-1}(t), & \text{if } \beta_\ell(t) = \beta_{\ell-1}(t) - \alpha, \\ \widetilde{S}_t(\ell\delta;\ell^*(t)\delta), & \text{if } \beta_\ell(t) > \beta_{\ell-1}(t) - \alpha. \end{cases}$$

Remark 4.10. One may wonder if the system of ODEs (4.8) actually characterizes the function β. For this we first need to characterize $t \mapsto \ell^*(t)$ as an explicit function of $\beta(t)$. One would like to define it as $\ell^*(t) = \arg\max_{0\leq\ell\leq L} \beta_\ell(t)$ and take it right-continuous. This is correct if there is a single $\arg\max$. Otherwise, there are by definition of T_0 two choices ℓ and ℓ' and there is a single admissible choice in the sense that the corresponding affine solution to (4.8) on $[t,t+\varepsilon]$ satisfies $\ell^*(s) = \arg\max_{0\leq\ell\leq L}$ for $\varepsilon > 0$ small enough. Indeed, if $\max_{0\leq\ell\leq L} \beta_\ell(t) = 1$, since $S(\ell'\delta,\ell\delta) = -S(\ell\delta,\ell'\delta)$, one of the two fitnesses is positive, for example $S(\ell\delta;\ell'\delta)$. If one takes the wrong choice $\ell^*(t) = \ell'$, then $\Sigma_\ell(t) = S(\ell\delta;\ell'\delta) > 0$, hence the solution to (4.8) gives $\beta_\ell(s) > 1$ for $s > t$ locally. If $\max_{0\leq\ell\leq L} \beta_\ell(t) < 1$, a similar argument with $\widehat{S}$ consists in choosing the trait with higher invasion fitness. Therefore, (4.8) can be expressed as an autonomous ODE system and there is a unique admissible solution.

Exercise 18. *Verify the statement of the penultimate sentence of Remark 4.10.*

By re-emergence of a trait $\ell\delta$, we mean that $\beta_\ell(s) = 1$ on some time interval $[t_1,t_2]$ with non-empty interior, then $\beta_\ell(s) < 1$ on some non-empty interval (t_2,t_3), and then $\beta_\ell(s) = 1$ again on some interval $[t_3,t_4]$ with non-empty interior. We would like to predict the evolutionary outcome depending on the parameters α,δ,τ. According to the case of three traits ($L = 2$) studied in [CMT21, Section 3], there are so many possible situations that we are not able to fully characterize the outcomes. Therefore, [CMT21, Section 2.2] focused on the beginning of the dynamics until either global extinction or re-emergence of one trait.

We assume that $\delta < 4/3$ (so that $L \geq 3$) and only consider the case $\delta < \tau < 3$.[35] Let

$$\widetilde{k} := \lceil \frac{\tau}{\delta} \rceil \qquad \text{and} \qquad \bar{k} = \lfloor 2\frac{\tau}{\delta} \rfloor.$$

It turns out (it follows from the proof of Theorem 4.11 presented in [CMT21]) that, for the first few phases,

$$s_k = \frac{k\alpha}{\tau - \delta},$$

[35] For $\tau < \delta$, the initial resident stays resident forever, see the case of three traits in [CMT21, Section 3].

trait $k\delta$ is resident on $[s_k, s_{k+1})$ (i.e. $\beta_k(s) = 1$) and for all $s \in [s_k, s_{k+1})$,

$$\beta_\ell(s) = \begin{cases} [1 - (\ell - k)\alpha + (\tau - \delta)(s - s_k)] \vee 0, & \text{if } k < \ell \le L, \\ 1 - \frac{\alpha - (k-\ell-1)}{\tau - \delta}(\tau - \frac{k-\ell}{2}\delta) - (\tau - (k-\ell)\delta)(s - s_k), & \text{if } 0 \le l < k. \end{cases} \tag{4.9}$$

These formulas stay valid until either $\beta_0(s) = 0$ (loss of trait 0), or $\beta_0(s) = 1$ for some $s > s_1$ (re-emergence of trait 0), or $\ell_k^* \delta > 3$ (the population size becomes $o(K)$). The function $\beta_0(s)$ in (4.9) is piecewise affine and its slope becomes positive at time $s_{\widetilde{k}}$. Hence, its minimal value is equal to

$$m_0 = \beta_0(s_{\widetilde{k}}) = 1 - \frac{\alpha(\widetilde{k} - 1)}{\tau - \delta}\big(\tau - \frac{\widetilde{k}}{2}\delta\big).$$

Provided that the latter is positive, β_0 reaches 1 again in phase $[s_{\bar{k}}, s_{\bar{k}+1})$ at time

$$\bar{\tau} = s_{\bar{k}} + \frac{\alpha(\bar{k} - 1)}{\tau - \delta}\frac{\tau - \frac{\bar{k}}{2}\delta}{k\delta - \tau}.$$

Theorem 4.11 ([CMT21])**.** *Assume that $\delta < \tau < 3$, $\delta < 4/3$, and the assumptions of Theorem 4.5 hold. Then,*

(a) if $m_0 > 0$ and $\bar{k}\delta < 3$, then the first re-emerging trait is 0 and the maximal exponent is always 1 until this re-emergence time,

(b) if $m_0 < 0$, trait 0 gets lost before its re-emergence and there is global extinction of the population before the re-emergence of any trait,

(c) if $m_0 > 0$ and $\bar{k}\delta > 3$, there is re-emergence of some trait ℓ such that $\ell\delta < 3$ and, for some time t before the time of first re-emergence, $\max_{1 \le \ell \le L} \beta_\ell(t) < 1$.

Biologically, Case (b) corresponds to evolutionary suicide. In Case (a), very few individuals with trait 0 remain, and they manage to return to residency after different traits having been resident, and until their re-emergence, the entire population size remains $\Theta(K)$ (albeit possibly with a much smaller prefactor of K than $\bar{n}(0)$). In Case (c), trait 0 itself may be lost, and there is an intermediate time interval where the total population size is $o(K)$, and re-emergence occurs after subpopulations with too large traits become small enough. In the latter two cases, one can expect successive re-emergences. However, it is not known if there exists a limit cycle for the dynamics. In particular, there are also cases when a trait different from 0 stays resident forever (see [CMT21, Section 3]). Cases (a), (b), (c) correspond to the examples (a), (b), (c) in Figure 6, respectively.

Heuristically, using the approximation that $\widetilde{k} \approx \tau/\delta$, we obtain that $m_0 \approx 1 - \frac{\alpha\tau}{2\delta}$. Hence, we have $m_0 > 0$ (re-emergence) provided τ is less than approximately $2\delta/\alpha$ and extinction otherwise. Transfer rates higher than $2\delta/\alpha$ favour extinction because the population is pushed to higher trait values. Small values of δ or high values of α give more time for extinction of the small subpopulations. Note that, for $m_0 > 0$, the condition $\bar{k}\delta < 3$ is roughly $\tau < 3/2$. Hence, for transfer rates smaller than $3/2$, 0 re-emerges first, while otherwise, other traits may re-emerge first and the population size will be $o(K)$ on some time interval.

4.5 Main ideas of the proof of Theorem 4.5

We start from the stochastic birth-and-death process $(N_0^K(t), \dots, N_L^K(t))_{t \ge 0}$ with mutation, competition, and transfer. Our goal is to study the limit behaviour of the vector-valued function $(\beta_0^K(t), \dots, \beta_L^K(t))_{t \ge 0}$ defined in (4.3).

Theorem 4.5 was obtained in [CMT21] by a fine comparison of the size of each subpopulation defined by a given trait value with carefully chosen branching processes with immigration. The stochastic dynamics consists in a succession of phases $[\sigma_k^K \log K, \theta_k^K \log K]$ followed by intermediate steps $[\theta_k^K \log K, \sigma_{k+1}^K \log K]$. They proved that θ_k^K converge in probability to s_k for $k \ge 1$. In each phase there is a single resident or

dominant subpopulation. When another trait reaches a comparable size, the intermediate step starts and ends after the replacement of the resident or dominant trait. In the limit, intermediate steps vanish on the time scale $\log K$.

To control of the exponents $\beta_\ell^K(t)$, the authors proceeded by a double induction, first on the phases, and inside each phase, on the traits $\ell\delta$, from $\ell = 0$ to $\ell = L$. The exponents are approximately piecewise affine. Changes of slopes (kinks) may happen when a new trait emerges, when a trait dies out or when the dynamics of a trait $\ell \geq 1$ becomes driven by incoming mutations from the trait $\ell - 1$. The authors used asymptotic results on branching processes with immigration to control the non-dominant subpopulations within the phases. During intermediate steps, two subpopulations are of maximal order. The authors used comparisons with dynamical systems and logistic branching processes for these phases.

Let us now describe in more detail how the stopping times θ_k^K are constructed. In each phase, this time may have two different definitions depending on whether the future emerging trait is dominant or resident. For this, it is convenient to consider θ_*^K and $\widetilde{\theta}_*^K$ where the index is omitted. Consider a phase, starting at time $\sigma_*^K \log K$ with the largest subpopulation of trait $\ell^*\delta$. Two cases may occur: Either $\ell^*\delta$ is a resident trait with population size close to its equilibrium size $(3 - \ell^*\delta)K/C$, or $\ell^*\delta$ is a dominant trait with population size $o(K)$.

In the first case, for the k-th step, we have $\sigma_*^K = \sigma_k^K \to s_{k-1}$ in probability when $K \to \infty$ and $\ell^* = \ell_K^*$. Other population sizes are negligible with respect to K. Given parameters $\varepsilon_* > 0$ and given $m > 0$ to be fixed later, we introduce

$$\theta_*^K = \inf\Big\{t \geq \sigma_*^K \colon N_{\ell^*}^K(t\log K) \notin \big[(\frac{3-\ell^*\delta}{C} - 3\varepsilon_*)K, (\frac{3-\ell^*\delta}{C} + 3\varepsilon_*)K\big] \text{ or } \sum_{\ell\neq\ell^*} N_\ell^K(t\log K) \geq m\varepsilon_* K\Big\}.$$

Roughly speaking, this is the first time when the trait ℓ^* population size rescaled by K leaves a small neighbourhood of its equilibrium or the total population size of all other traits reaches K times a significant factor $m\varepsilon^*$.

On the time interval $[\sigma_*^K \log K, \theta_*^K \log K]$, we proceed by induction on the traits. For $\ell \in [L]$, having proved the convergence of $\beta_0^K(t), \dots, \beta_{\ell-1}^K(t)$ to $\beta_0(t), \dots, \beta_{\ell-1}(t)$, we will bound the population size $N_\ell^K(t)$ for $\ell \neq \ell^*$ from below and above by branching processes with immigration on each interval where $\beta_{\ell-1}$ is affine. Either $\theta_*^K \to \infty$ when $K \to \infty$ with probability tending to 1 and in the limit there is no further change of the resident population. Or, θ_*^K has a finite limit s_* in probability ($s_* = s_k$ for the k-th phase). In this case, we will show that at time θ_*^K, only two traits have sizes of order K and are then competing with each other, or $s_* = T_0$ and we stop the analysis. In the first case, there is a transition step, with duration of order 1, leading with high probability to the replacement of the resident population by the new trait.

In the second case, for the k-th phase, the time σ_*^K is chosen such that $\sigma_*^K = \sigma_k^K \to s_{k-1} + s$ for some small $s > 0$ in probability when $K \to \infty$. We proceed similarly as before, replacing θ_*^K with

$$\widetilde{\theta}_*^K = \inf\Big\{t \geq \sigma_*^K \colon \beta_{\ell^*}^K(t) \notin \big[\beta_{\ell^*}(t) - \varepsilon_*, \beta_{\ell^*}(t) + \varepsilon_*\big] \text{ or } \sum_{\ell\neq\ell^*} N_\ell^K(t\log K) \geq m\varepsilon_* N_{\ell^*}^K(t\log K)\Big\}.$$

The interpretation of the second condition is clear: When the total population size of all other traits $\ell \neq \ell^*$ in total becomes significant compared to the size $N_{\ell_*}^K(t\log K)$ of the dominant population, t must be close to a time of change of dominant trait for the limiting process β. The first condition means that $\beta_{\ell^*}^K(t)$ leaves a small neighbourhood of $\beta_{\ell^*}(t)$. Given the convergence assertion of Theorem 4.5 that we want to prove, it is untypical that this occurs if ε is not too small. As long as it has not occurred, $\beta_{\ell^*}^K(t)$ is well-approximated by $\beta_{\ell^*}(t)$.

The full proof of Theorem 4.5 can be found in [CMT21, Section 5]. It is too long and technical to present it here, but it is a well-written proof that the reader may be interested to see. In the rest of Section 4, we will present some results on branching processes under a logarithmic scaling together with their proofs, to provide a flavour of the arguments. For similar (and for the proof of the theorem also essential) results on branching processes with immigration and logistic branching processes, we refer the reader to the appendix of [CMT21]. Although from the point of view of the proof of Theorem 4.5 these are just auxiliary results,

they can be adapted in many other population-dynamic and population-genetic model as well. Their proofs are also of independent interest; they use the weaponry of stochastic analysis.

4.6 Branching process in continuous time: scaling to a line

The following auxiliary result, [CMT21, Lemma A.1] is one of the important ingredients of the proof of Theorem 4.5. Here, we consider a single population $Z^K = (Z_t^K)_{t\geq 0}$ following a linear birth-and-death process, i.e. a branching process, with individual birth rate $b \geq 0$, individual death rate $d \geq 0$ and initial value $Z_0^K = \lfloor K^\beta - 1 \rfloor \in \mathbb{N}_0$. According to this initial condition, the population is initially extinct when $\beta = 0$, and $Z_0^K \sim K^\beta$ when $K \to \infty$ if $\beta > 0$. We denote the law of Z^K by $\mathrm{BP}(b, d, \beta)$. In the sequel, we denote the *net growth rate* of this process by $r = b - d$.

Lemma 4.12 ([CMT21])**.** *Let Z^K follow the law $\mathrm{BP}(b, d, \beta)$ where $b, d \geq 0$ and $\beta > 0$. Then for any $T > 0$, the process $(\frac{\log(1+Z^K_{s\log K})}{\log K})_{s\geq 0}$ converges in probability in $L^\infty([0,T],\mathbb{R})$ to $((\beta + rs) \vee 0)_{s\geq 0}$ when K tends to infinity. In addition, if $b < d$, then for all $t > \beta/r$,*

$$\lim_{K\to\infty} \mathbb{P}(Z^K_{t\log K} = 0) = 1. \tag{4.10}$$

Proof. Some of the ideas of this proof originate from [DM11].

Step 1 (Construction of a martingale)**.** We have

$$Z_t^K = K^\beta + M_t^K + \int_0^t r Z_s^K \mathrm{d}s,$$

where M^K is a square integrable martingale with predictable quadratic variation[36] $\langle M^K\rangle_t = \int_0^t (b+d) Z_s^K \mathrm{d}s$. Taking the expectation, $\mathbb{E}(Z_t^K)$ solves the linear ODE $\mathbb{E}(Z_t^K) = K^\beta + \int_0^t r\mathbb{E}(Z_s^K)\mathrm{d}s$ (written in integral form), which yields that

$$\mathbb{E}(Z_t^K) = K^\beta \mathrm{e}^{rt}.$$

Now, we want to use Itô's formula for semimartingales $X = (X_t)_{t\geq 0}$ and $Y = (Y_t)_{t\geq 0}$, which in differential form reads

$$\mathrm{d}(XY)_t = X_t \mathrm{d}Y_t + Y_t \mathrm{d}X_t + \langle X, Y\rangle_t,$$

with $t \mapsto \langle X, Y\rangle_t$ being their cross-covariation. In our case, this means

$$\mathrm{d}(\mathrm{e}^{-rt} Z_t^K) = \mathrm{e}^{-rt}\mathrm{d}Z_t^K - \mathrm{e}^{-rt} r Z_t^K \mathrm{d}t = \mathrm{e}^{-rt}\mathrm{d}M_t^K + \mathrm{e}^{-rt} r Z_t^K \mathrm{d}t - r Z_t^K \mathrm{e}^{-rt}\mathrm{d}t = \mathrm{e}^{-rt}\mathrm{d}M_t^K$$

(the cross-covariation vanishes because the deterministic process $t \mapsto \mathrm{e}^{-rt}$ is of bounded variation). Taking our initial condition into account, we obtain $1 + \mathrm{e}^{-rt} Z_t^K = 1 + K^\beta + \widehat{M}_t^K$, where $\widehat{M}_t^K = \int_0^t \mathrm{e}^{-rs}\mathrm{d}M_s^K$ is a square integrable martingale with quadratic variation process

$$\langle \widehat{M}_t^K\rangle = \int_0^t \mathrm{e}^{-2rs}(b+d) Z_s^K \mathrm{d}s.$$

Using Doob's maximal inequality, for any $0 < \eta < \beta$,

$$\begin{aligned}\mathbb{P}\Big(\sup_{t\leq T\log K} \big|\mathrm{e}^{-rt} Z_t^K - K^\beta\big| \geq K^\eta\Big) &= \mathbb{P}\Big(\sup_{t\leq T\log K} |\widehat{M}_t^K| \geq K^\eta\Big) \leq 4K^{-2\eta}\mathbb{E}\Big(\langle M^K\rangle_{T\log K}\Big) \\ &\leq 4K^{\beta-2\eta}(b+d)\cdot \begin{cases} \frac{1}{r}(1 - K^{-rT}) & \text{if } b \neq d, \\ T\log K & \text{if } b = d. \end{cases}\end{aligned}$$

[36] See the footnote in Section 2.8 for a remark on the notion of predictable quadratic variation.

Step 2 (Case $r > 0$)**.** Fix $T > 0$ and $\eta = 2\beta/3$. On the event

$$\Omega_1^K = \Big\{ \sup_{t \le T \log K} \big| \mathrm{e}^{-rt} Z_t^K - K^\beta \big| \le K^{2\beta/3} \Big\},$$

whose probability tends to 1, we have

$$\begin{aligned}
&\sup_{t\le T} \Big| \frac{\log\Big(1 + Z^K_{t\log K}\Big)}{\log K} - (\beta + rt) \Big| \\
&\quad = \sup_{t\le T} \Big| \frac{\log\Big(1 + Z^K_{t\log K}\Big)}{\log K} - \frac{\log\big(1 + K^{\beta+rt}\big)}{\log K} + \frac{\log\big(1 + K^{\beta+rt}\big)}{\log K} - \frac{\log\big(K^{\beta+rt}\big)}{\log K} \Big| \\
&\quad \le \sup_{t\le T} \Big[\frac{1}{\log K} \Big| \log\left(\frac{1 + Z^K_{t\log K}}{1 + K^{\beta+rt}} \right) \Big| + \frac{\log\big(1 + K^{-\beta-rt}\big)}{\log K} \Big] \\
&\quad \le \sup_{t\le T} \Big[\frac{1}{\log K} \Big| \log\left(\frac{1 + Z^K_{t\log K} \vee K^{\beta+rt}}{1 + Z^K_{t\log K} \wedge K^{\beta+rt}} \right) \Big| + \frac{K^{-\beta-rt}}{\log K} \Big] \\
&\quad \le \sup_{t\le T} \Big[\frac{1}{\log K} \Big| \log\left(1 + \frac{|Z^K_{t\log K} - K^{\beta+rt}|}{1 + Z^K_{t\log K} \wedge K^{\beta+rt}} \right) \Big| + \frac{K^{-\beta-rt}}{\log K} \Big] \\
&\quad \le \sup_{t\le T} \Big[\frac{1}{\log K} \Big| \frac{|Z^K_{t\log K} - K^{\beta+rt}|}{1 + K^{\beta+rt} - K^{\frac{2\beta}{3}+rt}} \Big| + \frac{K^{-\beta-rt}}{\log K} \Big] \\
&\quad \le \frac{K^{-2\beta/3}}{\log K} \sup_{t\le T} \frac{K^{rt}}{K^{\beta+rt} - K^{2\beta/3+rt}} + K^{-\beta} \log K, \\
&\quad \le \frac{2K^{-\beta/3} + K^{-\beta}}{\log K},
\end{aligned}$$

where the last inequality holds (only) for K large enough, and in the third and fifth step we used that $\log(1+\varepsilon) < \varepsilon$ for $\varepsilon > 0$ small enough. A simple adaptation of the previous argument gives the same condition for $b = d$.

Exercise 19. *Prove the latter statement.*

Step 3 (Case $r < 0$)**.** Since the function $t \mapsto \beta + rt$ vanishes at time $\beta/|r|$, we need to consider three phases. We fix $\varepsilon \in (0, \beta)$ and set $T_\varepsilon = \frac{\beta-\varepsilon}{r}$ and $\eta = \beta - \varepsilon/3$. First we prove that before time $T_\varepsilon \log K$, the population size remains large enough to use the same argument as in Step 2 (to show that the convergence of the rescaled logarithmic frequency process to the respective line). Second, the population goes extinct with high probability between times $T_\varepsilon \log K$ and $(T_\varepsilon + 2\varepsilon/|r|) \log K$, and third, in order to obtain the convergence in the L^∞ norm, we prove that the supremum of the process $(\frac{\log\left(1+Z^K_{t\log K}\right)}{\log K})_t$ on the entire time interval under consideration remains of the order $K^{\mathrm{polynomial}(\varepsilon)}$. Since ε can be chosen arbitrarily small, the result follows.

Step 3(i). On the set

$$\Omega_2^K = \Big\{ \sup_{t \le T_\varepsilon \log K} |\mathrm{e}^{-rt} Z_t^K - K^\beta| \le K^{-\beta-\varepsilon/3} \Big\}$$

whose probability tends to 1, using arguments analogous to Step 2, we have

$$\begin{aligned}
\sup_{t\le T_\varepsilon} \Big| \frac{\log\Big(1 + Z^K_{t\log K}\Big)}{\log K} - (\beta + rt) \Big| &\le \sup_{t\le T_\varepsilon} \Big[\frac{1}{\log K} \frac{|Z^K_{t\log K} - K^{\beta+rt}|}{1 + (K^{\beta+rt} - K^{\frac{2\beta}{3}+rt})_+} + \frac{K^{-\beta-rt}}{\log K} \Big] \\
&\le \frac{K^{\beta-\varepsilon/3}}{\log K} \sup_{t\le T_\varepsilon} \frac{K^{rt}}{1 + (K^{\beta+rt} - K^{\frac{2\beta}{3}+rt})_+} + \frac{K^{-\varepsilon}}{\log K} \le \frac{2K^{-\varepsilon/3} + K^{-\varepsilon}}{\log K},
\end{aligned}$$

with the last inequality again holding (only) for K large enough. The right-hand side also converges to zero when $K \to \infty$.

Step 3(ii). It follows from the previous step that, with probability converging to 1 as $K \to \infty$, $Z^K_{T_\varepsilon \log K} \leq 2K^\varepsilon$. Now, it was shown in [M16, Section 5.4.5, p. 180] that the extinction time T_{ext} of a BP$(b, d, 1)$ satisfies

$$\mathbb{P}(T_{\text{ext}} > t) = \frac{r\mathrm{e}^{rt}}{b\mathrm{e}^{rt} - d}, \qquad r \geq 0.$$

Hence, for a BP$(b, d, 2K^\varepsilon)$ branching process, thanks to the branching property, we have

$$\mathbb{P}(T_{\text{ext}} > t) = 1 - \Big(1 - \frac{r\mathrm{e}^{rt}}{b\mathrm{e}^{rt} - d}\Big)^{2K^\varepsilon}.$$

Thus, for $t = \frac{2\varepsilon}{|r|} \log K$,

$$\mathbb{P}\Big(T_{\text{ext}} > \frac{2\varepsilon}{|r|} \log K\Big) \sim 2\frac{|r|}{d} K^{-\varepsilon}.$$

Since this tends to 0 when $K \to \infty$, we have completed the proof of (4.10).

Step 3(iii). Since the last two steps were true for any value of $\varepsilon > 0$, in order to complete the proof, it is enough to check that

$$\sup_{s \in [T_\varepsilon, T_\varepsilon + 2\varepsilon/|r|]} \frac{\log\Big(1 + Z^K_{s \log K}\Big)}{\log K} \leq \frac{2d}{|r|}\varepsilon.$$

For this, we observe that the maximal sizes of families stemming from each individual alive at time $T_\varepsilon \log K$ on the time interval $[T_\varepsilon \log K, (T_\varepsilon + 2\varepsilon/|r|) \log K]$ are bounded by the sum of sizes at time $2\varepsilon \log K/|r|$ of $Z^K_{T_\varepsilon \log K}$ many independent Yule processes with birth rate b, i.e., i.i.d. geometric random variables G_i with expectation $K^{b\varepsilon/|r|}$.[37] Hence, with probability tending to 1,

$$\sup_{t \in [T_\varepsilon, T_\varepsilon + 2\varepsilon/|r|]} Z^K_{t \log K} \leq \sum_{i=1}^{2K^\varepsilon} G_i \leq K^{\frac{2d}{|r|}\varepsilon}.$$

Exercise 20 (easy)**.** *The last inequality stands without further explanation in [CMT21, Appendix A]. Fill the gaps of the proof and show (using the footnote on Yule processes below) that this inequality is indeed true. Which known inequality is implicitly used here?*

This finishes the proof.

□

[37] Such a Yule process is defined as a BP$(b, 0, 1)$, i.e., a binary pure birth process with individual birth rate b, started with one single individual at time zero. It is a classical result that at time $t > 0$, the number of individuals of such a Yule process follows a geometric distribution with expectation e^{bt}.

A Proof of Lemma 1.4

Proof of Lemma 1.4. By Theorem 1.14 part (a), it suffices to prove that

$$\sup_{K\geq 1}\sup_{t\geq 0}\mathbb{E}\big((Z_t^K)^p\big)<\infty,$$

where $\mathcal{L}(Z^K)=\mathbb{P}^K(2\bar{b},0,\underline{\alpha},z_0^K)$ when $\sup_{K\geq 1}\mathbb{E}((z_0^K)^p)<\infty$. Let us define $v_t^k=\mathbb{P}(Z_t^K=k/K)$. Then we have

$$\begin{aligned}\frac{\mathrm{d}}{\mathrm{d}t}\mathbb{E}\big((Z_t^K)^p\big)&=\sum_{k\geq 1}\big(\frac{k}{K}\big)^p\frac{\mathrm{d}v_t^k}{\mathrm{d}t}\\&=\frac{1}{K^p}\sum_{k\geq 1}k^p\big[2\bar{b}(k-1)v_t^{k-1}+\underline{\alpha}\frac{k}{K}\big(2\bar{b}+\underline{\alpha}\frac{k}{K}\big)v_t^k\big]\\&=\frac{1}{K^p}\sum_{k\geq 1}\Big[2\bar{b}\Big(\Big(1+\frac{1}{k}\Big)^p-1\Big)+\underline{\alpha}\frac{k}{K}\Big(\Big(1-\frac{1}{k}\Big)^p-1\Big]k^{p+1}v_t^k.\end{aligned}$$

Now, for $k/K>4\bar{b}/\alpha$, the quantity inside the square brackets on the right-hand side can be bounded from above by $-2\bar{b}[3-2(1-1/k)^p-(1+1/k)^p]$, which is asymptotically equivalent to $-2\bar{b}p/k$ as $k\to\infty$. Therefore, there exists a constant $k_0\in\mathbb{N}$ that can be assumed bigger than $4\bar{b}/\alpha$ such that, for any $k\geq k_0$, $-2\bar{b}[3-2(1-1/k)^p-(1+1/k)^p]\leq-\bar{b}p/k$, thanks to Taylor's formula. Then, using the fact that $(1+x)^p-1\leq x(2^p-1)$ for any $x\in[0,1]$, we can write

$$\frac{\mathrm{d}}{\mathrm{d}t}\mathbb{E}\big((Z_t^K)^p\big)\leq\sum_{k=1}^{Kk_0-1}2\bar{b}(2^p-1)\big(\frac{k}{K}\big)^pv_t^k-\sum_{k\geq Kk_0}\bar{b}p\big(\frac{k}{K}\big)^pv_t^k\leq 2\bar{b}(2^p-1)k_0^p+\bar{b}pk_0^p-\bar{b}p\mathbb{E}((Z_t^K)^p).$$

Writing $C=(2(2^p-1)+p)k_0^p/p$, this differential inequality has the solution

$$\mathbb{E}\big((Z_t^K)^p\big)\leq C+\mathbb{E}((z_0^K)^p-C)\mathrm{e}^{-\bar{b}pt},$$

which gives the desired boundedness of the p-th moments.

□

B The function L in the definition of the rate function in Section 1.8

This part of the proof of Theorem 1.17 was not spelt out in [C06], but there is an explanation for a somewhat more general case in [BPT23, Appendix, Section B.1], which we follow here. For $y,z\in\mathbb{R}^2$ we define the function $L(y,z)=\sup_{\alpha\in\mathbb{R}^2}\left(\langle\alpha,z\rangle-H(y,\alpha)\right)$ with

$$H(y,\alpha)=\int_{\mathbb{R}^2}\left(\exp(\langle\alpha,x\rangle)-1\right)\nu_y(\mathrm{d}x),$$

where the measures ν_y, $y\in\mathbb{R}$, are defined as follows:

$$\nu_x(1)=p(x),\qquad\nu_x(-1)=q(x).$$

Now, calculating the gradient of the function in the supremum with respect to α shows that $L(y,z)=0$ if and only if $z=p(y)-q(y)$, as claimed in Section 1.8.

C Proof of Lemma 2.19

Proof of Lemma 2.19. Since (π_{2a},π_{2d}) is a left eigenvector of J corresponding to the eigenvalue $\widetilde{\lambda}$, we have

$$(\lambda_2-\lambda_1)+\sigma\frac{\pi_{2d}}{\pi_{2a}}=\widetilde{\lambda}=p(\lambda_1-\mu)\frac{\pi_{2a}}{\pi_{2d}}-(\kappa\mu+\sigma).$$

Hence, since $\widetilde{\lambda} > 0$, given that $\varepsilon > 0$ is small enough, we obtain

$$\frac{\pi_{2d}}{\pi_{2a}} = \frac{\widetilde{\lambda} - \lambda_2 + \lambda_1}{\sigma} = \frac{\widetilde{\lambda} - \lambda_2 + \mu + \alpha\Big(\frac{\lambda_1 - \mu}{\alpha}\Big)}{\sigma} > \frac{-\lambda_2 + \mu + \alpha\Big(\frac{\lambda_1 - \mu}{\alpha} + 3\sqrt{\varepsilon}\Big)}{\sigma} \geq \frac{\mu - \lambda_2 + \alpha(n_1 + n_{2a})}{\sigma}$$

and

$$\frac{\pi_{2d}}{\pi_{2a}} = \frac{p\alpha\Big(\frac{\lambda_1 - \mu}{\alpha}\Big)}{\widetilde{\lambda} + \kappa\mu + \sigma} < \frac{p\alpha\Big(\frac{\lambda_1 - \mu}{\alpha} - 2\varepsilon\Big)}{\kappa\mu + \sigma} \leq \frac{p\alpha(n_1 + n_{2a})}{\kappa\mu + \sigma},$$

as asserted. □

D Proof of Proposition 2.16

Proof of Proposition 2.16 from [BT20]. If $\pi_{2a} - \delta < \frac{N^K_{2a,T^2_\varepsilon}}{N^K_{2a,T^2_\varepsilon} + N^K_{2d,T^2_\varepsilon}} < \pi_{2a} + \delta$, then there is nothing to show. Let us assume that

$$\frac{N^K_{2a,T^2_\varepsilon}}{N^K_{2a,T^2_\varepsilon} + N^K_{2d,T^2_\varepsilon}} \leq \pi_{2a} - \delta,$$

the symmetric case $\frac{N_{2a,T^2_\varepsilon}}{N_{2a,T^2_\varepsilon} + N_{2d,T^2_\varepsilon}} \geq \pi_{2a} + \delta$ can be treated similarly. Let us introduce the event

$$\widetilde{A}_\varepsilon := \{T^2_{\sqrt{\varepsilon}} < T^2_0 \wedge R_{2\varepsilon}\}$$

on which we conditioned in (2.30). Our first goal is to show that for $\varepsilon > 0$ small, with high probability, once the total mutant population size reaches εK, for sufficiently large $C > 0$ it will not decrease to a level lower than $\varepsilon K/C$ again before it reaches $\sqrt{\varepsilon}K$. To be more precise, for $C > 0$ we introduce the stopping time

$$T_{\varepsilon,\varepsilon/C} = \inf\big\{t \geq T^2_\varepsilon \colon N^K_{2,t} \leq \tfrac{\varepsilon K}{C}\big\}.$$

Then our goal is to show that if C is large enough, then $T^2_{\sqrt{\varepsilon}}$ is larger than $T^2_\varepsilon + \log\log(1/\varepsilon)$ and smaller than $T_{\varepsilon,\varepsilon/C}$. First of all, for all $\varepsilon > 0$ sufficiently small[38], [CCLLS21, Lemma A.1] implies that for C large enough,

$$\lim_{K\to\infty} \mathbb{P}\big(T_{\varepsilon,\varepsilon/C} < T^2_{\sqrt{\varepsilon}} \big| \widetilde{A}_\varepsilon\big) = 0. \tag{D.1}$$

On the other hand, note that the total size of mutant individuals is stochastically dominated from above by a Yule process with birth rate λ_2. Thus, by [CCLLS21, Lemma A.2], we have

$$\lim_{K\to\infty} \mathbb{P}\big(T^2_{\sqrt{\varepsilon}} \leq T^2_\varepsilon + \log\log(1/\varepsilon) \big| \widetilde{A}_\varepsilon\big) \leq \sqrt{\varepsilon}(\log(1/\varepsilon))^{\lambda_2}. \tag{D.2}$$

Using these, we want to show that the fraction $\frac{N^K_{2a,t}}{N^K_{2a,t} + N^K_{2d,t}}$ cannot stay below $\pi_{2a} - \delta$ on $[T^2_\varepsilon, T^2_{\sqrt{\varepsilon}}]$ with probability close to one. Let us define the following five independent Poisson random measures on $[0,\infty]^2$ with intensity measure $\mathrm{d}s\mathrm{d}\theta$:

- $P^{\mathrm{b}}_{2a}(\mathrm{d}s, \mathrm{d}\theta)$ representing the birth events of the active mutant individuals,
- $P^{\mathrm{d}}_{2a}(\mathrm{d}s, \mathrm{d}\theta)$ representing the death events of the active mutant individuals,
- $P^{\mathrm{s}}_{2a\to 2d}(\mathrm{d}s, \mathrm{d}\theta)$ representing the active→dormant switching events,

[38] Here, we skip some details that can be found in [BT20, Section 4.1]: We use that the coupling (2.28) is satisfied on $\widetilde{A}_\varepsilon$ and the branching process $(Z^{\varepsilon,-}_{2a,t}, Z^{\varepsilon,-}_{2d,t})$ is supercritical.

- $P^{\mathrm{d}}_{2d}(\mathrm{d}s,\mathrm{d}\theta)$ representing the death events of the dormant mutant individuals (for $\kappa=0$ this measure can be omitted),
- $P^{\mathrm{s}}_{2d\to 2a}(\mathrm{d}s\mathrm{d}\theta)$ representing the dormant→active switching events.

The following assertion is often very useful in the context of Poisson point processes.

Theorem D.1 (Colouring Theorem, [K93]). *Let Π be a Poisson point process on S with intensity measure μ. Let the points of Π be randomly coloured by k colours, the probability that a point receives the i-th colour being p_i (such that $p_i\geq 0$, $\sum_{i=1}^k p_i=1$), and the colours of different points of Π being independent (of one another and of the position of the points). Let Π_i the set of the points that have the i-th colour. Then $\Pi_1,\dots,\Pi_k$ are independent Poisson point processes on S, and Π_i has intensity measure $\mu_i=p_i\mu$ for all $i\in\{1,\dots,k\}$.*

The set Π_i is often called a(n independent) *thinning* of Π with survival probability p_i, see e.g. [BB09, Section 1.3.2]. The interpretation for this is that one can consider i.i.d. Bernoulli random variables $\{I^i_X\}_{X\in\Pi}$ with common survival probability p_i, and then Π_i equals $\{X\in\Pi|I^i_X=1\}$ in distribution, for all $i=1,\dots,k$.

In our case, the reason why competitive death events can be assumed as independent of active→dormant switches is that the corresponding Poisson random measures can be obtained as an *independent thinning* of a Poisson random measure with survival probability $1-p$ respectively the complementary thinning (with survival probability p), which are independent Poisson random measures according to [K93, Section 5.1]. Let

$$\widetilde{P}^{\mathrm{b}}_{2a}(\mathrm{d}s,\mathrm{d}\theta):=P^{\mathrm{b}}_{2a}(\mathrm{d}s,\mathrm{d}\theta)-\mathrm{d}s\mathrm{d}\theta,\dots,\widetilde{P}^{\mathrm{s}}_{2d\to 2a}(\mathrm{d}s,\mathrm{d}\theta):=P^{\mathrm{s}}_{2d\to 2a}(\mathrm{d}s,\mathrm{d}\theta)-\mathrm{d}s\mathrm{d}\theta$$

be the associated *compensated* measures (see the explanation below).

The fraction $\frac{N^K_{2a,t}}{N^K_{2a,t}+N^K_{2d,t}}$ is a semimartingale and can be decomposed as follows

$$\frac{N^K_{2a,t}}{N^K_{2a,t}+N^K_{2d,t}}=\frac{N^K_{2a,T^2_\varepsilon}}{N^K_{2a,T^2_\varepsilon}+N^K_{2d,T^2_\varepsilon}}+M_2(t)+V_2(t),\qquad t\geq T^2_\varepsilon,$$

with M_2 being a martingale and V_2 a finite-variation process such that

$$\begin{aligned}
M_2(t)=&\int_{T^2_\varepsilon}^t\int_{[0,\infty)}\mathbb{1}\{\theta\leq\lambda_2 N^K_{2a,s-}\}\frac{N^K_{2d,s-}}{N^K_{2,s-}(N^K_{2,s-}+1)}\widetilde{P}^{\mathrm{b}}_{2a}(\mathrm{d}s,\mathrm{d}\theta)\\
&-\int_{T^2_\varepsilon}^t\int_{[0,\infty)}\mathbb{1}\{\theta\leq N^K_{2a,s-}(\mu+\alpha(1-p)(N^K_{1,s-}+N^K_{2a,s-}))\}\\
&\quad\times\frac{N^K_{2d,s-}}{N^K_{2,s-}(N^K_{2,s-}-1)}\widetilde{P}^{\mathrm{d}}_{2a}(\mathrm{d}s,\mathrm{d}\theta)\\
&-\int_{T^2_\varepsilon}^t\int_{[0,\infty)}\mathbb{1}\{\theta\leq N^K_{2a,s-}(\alpha p(N^K_{1,s-}+N^K_{2a,s-}))\}\frac{1}{N^K_{2,s-}}\widetilde{P}^{\mathrm{s}}_{2a\to 2d}(\mathrm{d}s,\mathrm{d}\theta)\\
&+\int_{T^2_\varepsilon}^t\int_{[0,\infty)}\mathbb{1}\{\theta\leq\kappa\mu N^K_{2d,s-}\}\frac{N^K_{2a,s-}}{N^K_{2,s-}(N^K_{2,s-}-1)}\widetilde{P}^{\mathrm{d}}_{2d}(\mathrm{d}s,\mathrm{d}\theta)\\
&+\int_{T^2_\varepsilon}^t\int_{[0,\infty)}\mathbb{1}\{\theta\leq\sigma N^K_{2d,s-}\}\frac{1}{N^K_{2,s-}}\widetilde{P}^{\mathrm{s}}_{2d\to 2a}(\mathrm{d}s,\mathrm{d}\theta)
\end{aligned}$$

and

$$\begin{aligned}
V_2(t)=\int_{T^2_\varepsilon}^t\Big\{&\lambda_2 N^K_{2a,s}\frac{N^K_{2d,s}}{N^K_{2,s}(N^K_{2,s}+1)}\\
&-N^K_{2a,s}(\mu+\alpha(1-p)(N^K_{1,s}+N^K_{2a,s}))\frac{N^K_{2d,s}}{N^K_{2,s}(N^K_{2,s}-1)}\\
&-N^K_{2a,s}\alpha p(N^K_{1,s}+N^K_{2a,s}))\frac{1}{N^K_{2,s}}\mathrm{d}s+\kappa\mu N^K_{2d,s}\frac{N^K_{2a,s}}{N^K_{2,s}(N^K_{2,s}-1)}+\sigma N^K_{2d,s}\frac{1}{N^K_{2,s}}\Big\}\mathrm{d}s.
\end{aligned}$$

Further, the predictable quadratic variation of the martingale M_2 is given as follows

$$\begin{aligned}\langle M_2\rangle_t = &\int_{T_\varepsilon^2}^t \lambda_2 N_{2a,s}^K \frac{(N_{2d,s}^K)^2}{(N_{2,s}^K)^2(N_{2,s}^K+1)^2}\mathrm{d}s\\ &+\int_{T_\varepsilon^2}^t \mu N_{2a,s}^K(\mu+\alpha(1-p)(N_{1,s-}^K+N_{2a,s-}^K))\frac{(N_{2d,s}^K)^2}{(N_{2,s}^K)^2(N_{2,s}^K-1)^2}\\ &+N_{2a,s}^K\alpha p(N_{1,s}^K+N_{2a,s}^K)\frac{1}{(N_{2,s}^K)^2}\mathrm{d}s+\kappa\mu N_{2d,s}^K\frac{(N_{2a,s}^K)^2}{(N_{2,s}^K(N_{2,s}^K-1))^2}\\ &+\sigma N_{2d,s}^K\frac{1}{(N_{2,s}^K)^2}\mathrm{d}s.\end{aligned}$$

Although we do not want to go into formal details with stochastic integrals against processes with jumps here (see again [E19] for a great overview), the principle is again simple. In the definition of $M_2(t)$, the frequencies of events appearing in the indicators are tuned in such a way that they correspond to our process $\mathbf{N}_t^K$, and they are multiplied by a factor expressing the change of the fraction of active individuals among all trait 2 individuals after a given type of jump (exercise!). We integrate against the compensated Poisson point process, which is a martingale, and thus the stochastic integral will also be a martingale, similarly to how integrating against Brownian motion (which is a martingale itself) yields a martingale under suitable assumptions on the integrand. One necessary condition on the integrand is predictability, which is achieved via taking left limits (i.e. evaluation at time $s-$, i.e. before the jump) everywhere. The finite-variation process is similar, but here the additional randomization via the indicator including θ is missing, and we integrate against the Lebesgue measure, so that taking left limits is not necessary. A bit more precisely, we integrate the aforementioned changes of trait 2a/(2a+2d) fractions against the intensity measure of the Poisson point process rather than against the Poisson point process itself, where w.r.t. the θ-coordinate we integrate over the whole space. The predictable quadratic variation (see also the corresponding footnote in Section 2.7) looks again similar to the finite-variation process, but here the jump sizes are squared, and every term appears with a positive sign. In the case of integrals against Poisson point processes, it is true that the predictable quadratic variation is obtained via integrating the squares of jump sizes against the intensity measure.

This yields that there exists $C_0>0$ such that for all $t\geq T_\varepsilon^2$,

$$\langle M_2\rangle_t \leq C_0(t-T_\varepsilon^2)\sup_{T_\varepsilon^2\leq s\leq t}\frac{1}{N_{2,s}^K-1}.$$

This implies

$$\langle M_2\rangle_{(T_\varepsilon^2+\log\log(1/\varepsilon))\wedge T_{\varepsilon,\varepsilon/C}} \leq \frac{C_0\log\log(1/\varepsilon)}{\frac{\varepsilon K}{C}-1} \tag{D.3}$$

and

$$\begin{aligned}&\limsup_{K\to\infty}\mathbb{P}\Big(\sup_{T_\varepsilon^2\leq t\leq T_\varepsilon^2+\log\log(1/\varepsilon)}|M_2(t)|\geq\varepsilon\Big|\widetilde{A}_\varepsilon\Big)\\ &\leq\limsup_{K\to\infty}\Big(\mathbb{P}\Big(\sup_{T_\varepsilon^2\leq t\leq(T_\varepsilon^2+\log\log(1/\varepsilon))\wedge T_{\varepsilon,\varepsilon/C}}|M_2(t)|\geq\varepsilon\Big|\widetilde{A}_\varepsilon\Big)\\ &\qquad+\mathbb{P}\big(T_{\varepsilon,\varepsilon/C}<T_\varepsilon^2+\log\log(1/\varepsilon)\big|\widetilde{A}_\varepsilon\big)\Big)\\ &\leq\limsup_{K\to\infty}\frac{1}{\varepsilon^2}\mathbb{E}\Big[\langle M_2\rangle_{(T_\varepsilon^2+\log\log(1/\varepsilon))\wedge T_{\varepsilon,\varepsilon/C}}\Big|\widetilde{A}_\varepsilon\Big]+\sqrt{\varepsilon}(\log 1/\varepsilon)^{\lambda_2}\\ &=\sqrt{\varepsilon}(\log 1/\varepsilon)^{\lambda_2},\end{aligned} \tag{D.4}$$

where in the first inequality of the last line we used Doob's martingale inequality for the first term and (D.1) together with (D.2) for the second term, and the last inequality of the last line is due to (D.3).

Let us now consider the finite-variation process V_2. This can be written as

$$V_2(t) = \int_{T_\varepsilon^2}^t P\Big(\frac{N_{2a,s}^K}{N_{2,s}^K}\Big)\frac{N_{2,s}^K}{N_{2,s}^K+1} + Q^{(s)}\Big(\frac{N_{2a,s}^K}{N_{2,s}^K}\Big)\frac{N_{2,s}^K}{N_{2,s}^K-1} + R^{(s)}\Big(\frac{N_{2a,s}^K}{N_{2,s}^K}\Big)\mathrm{d}s, \tag{D.5}$$

with

$$P(x) = \lambda_2 x(1-x), \quad Q^{(s)}(x) = (\kappa\mu - \mu - \alpha(1-p)(N_{1,s}^K + N_{2a,s}^K)))x(1-x),$$
$$R^{(s)}(x) = \sigma(1-x) - p\alpha\big(N_{1,s}^K + N_{2a,s}^K\big)x.$$

For $\varepsilon > 0$ small, on $[T_\varepsilon^2, T_{\sqrt{\varepsilon}}^2]$, $Q^{(s)}$ and $R^{(s)}$ are close on $[0,1]$, respectively, to the polynomial functions Q, R given as follows

$$Q(x) = (\kappa\mu - \mu - \alpha(1-p)\bar{n}_1)x(1-x) = (\kappa\mu - \mu - (1-p)(\lambda_1 - \mu))x(1-x),$$
$$R(x) = \sigma(1-x) - p\alpha\bar{n}_1 x = \sigma(1-x) - p(\lambda_1 - \mu)x.$$

Thus, for given $\varepsilon > 0$, for all sufficiently large K, the integrand in (D.5) is close to the polynomial function

$$S(x) = (\lambda_2 + \kappa\mu - \mu - (1-p)(\lambda_1 - \mu))x(1-x) + \sigma(1-x) - p(\lambda_1 - \mu)x.$$

Since $S(0) > 0$ and $S(1) < 0$, further, S is of degree 2, the equation $\dot{x} = S(x)$ has a unique equilibrium in $(0,1)$. Now, let (π_{2a}, π_{2d}) be the left eigenvector of the matrix J defined in (2.13) corresponding to the eigenvalue $\widetilde{\lambda}$ such that $\pi_{2a} + \pi_{2d} = 1$. A direct computation implies that π_{2a} is a root of S and thus equal to this equilibrium. Thus, we can choose $\delta > 0$ and $\theta > 0$ such that $\pi_{2a} - \delta > 0$ and for all $x < \pi_{2a} - \delta$, $S(x) > \theta/2$. By continuity, this implies that for all sufficiently small $\varepsilon > 0$ and accordingly chosen sufficiently large $K > 0$, on the event $\widetilde{A}_\varepsilon$ the following relation holds for all $s \in \big[T_\varepsilon^2, T_{\sqrt{\varepsilon}}^2\big]$ and $x \in (0, \pi_{2a} - \delta)$:

$$P(x)\frac{N_{2,s}^K + 1}{N_{2,s}^K} + Q^{(s)}(x)\frac{N_{2,s}^K - 1}{N_{2,s}^K} + R^{(s)}(x) \geq \frac{\theta}{2} > 0. \tag{D.6}$$

Let us define

$$\mathfrak{t}_{2a}^{(\varepsilon)} := \inf\Big\{t \geq T_\varepsilon^2 \colon \frac{N_{2a,t}^K}{N_{2d,t}^K} \geq \pi_{2a} - \delta\Big\}.$$

From (D.4) and (D.6) we obtain that on the event $\widetilde{A}_\varepsilon$, for any $t \in [T_\varepsilon^2, (T_\varepsilon^2 + \log\log(1/\varepsilon)) \wedge \mathfrak{t}_{2a}^{(\varepsilon)}]$,

$$\pi_{2a} - \delta \geq \frac{N_{2a,t}^K}{N_{2,t}^K} \geq \frac{\theta}{2}\Big(\log\log(1/\varepsilon) \wedge (\mathfrak{t}_{2a}^{(\varepsilon)} - T_\varepsilon^2)\Big) - \varepsilon$$

with a probability higher than $1 - \sqrt{\varepsilon}(\log(1/\varepsilon))^{\lambda_2}$. Since $\frac{\theta}{2}\log\log(1/\varepsilon)$ tends to ∞ as $\varepsilon \downarrow 0$, it follows that for $\varepsilon > 0$ small, $\mathfrak{t}_{2a}^{(\varepsilon)}$ is smaller than $T_\varepsilon^2 + \log\log(1/\varepsilon)$ and thus smaller than $T_{\sqrt{\varepsilon}}^2$ with a probability close to 1 on the event $\widetilde{A}_\varepsilon$, where we also used (D.2).

Lastly, note that each jump of the process $N_{2a,t}^K/N_{2,t}^K$ is smaller than $(\varepsilon K/C + 1)^{-1}$, and hence smaller than δ for all K sufficiently large (given ε). Thus, after the time $\mathfrak{t}_{2a}^{(\varepsilon)}$, the process will be contained in the interval $[\pi_{2a} - \delta, \pi_{2a} + \delta]$ for some positive amount of time. Hence, we conclude the proposition. □

E Proof of Corollary 3.12

The following proof is an almost verbatim quote of the proof of [BT23, Corollary 2.5].

Proof of Corollary 3.12. According to the properties of the linearized variant of the system (3.13) near $(0,0,0,0)$, if $(n_{1a}(0), n_{1d}(0), n_{1i}(0), n_2(0)) \in [0,\infty)^4$ with $n_{1a}(0) > 0$, then $\liminf_{t\to\infty} n_{1a}(t) > 0$. (This follows from the fact that the Jacobi matrix at $(0,0,0,0)$ has a positive eigenvalue with eigenvector $(1,0,0,0)$.)

Next, note that if $\mathbf{n}(0) \in [0,\infty)^4$ with $n_{1a}(0) > 0$, then there are two possibilities. Either $\max\{n_{1i}(0), n_2(0)\} > 0$ and hence $n_{1d}(t), n_{1i}(t), n_2(t) > 0$ for all $t > 0$, or $\max\{n_{1i}(0), n_2(0)\} = 0$ and hence $\lim_{t\to\infty} \mathbf{n}(t) = (\bar{n}_{1a}, 0, 0, 0)$. Thanks to the invariance of ω-limit sets[39], this implies that if the ω-limit set of $(\mathbf{n}(t))_{t\geq 0}$ contains a point with a zero coordinate (which is necessarily not the type 1a coordinate), then in fact the ω-limit set contains $(\bar{n}_{1a}, 0, 0, 0)$, i.e. the solution converges to $(\bar{n}_{1a}, 0, 0, 0)$ at least along a subsequence of times. But for a coordinatewise positive initial condition, that would contradict Proposition 3.11, hence the positivity part of the proposition.

Next, let us verify that $\limsup_{t\to\infty} n_{1a}(t) + n_{1d}(t) + n_{1i}(t) < \bar{n}_{1a}$. Summing the first three lines of (3.13), we obtain

$$\dot{n}_{1a}(t) + \dot{n}_{1d}(t) + \dot{n}_{1i}(t) = n_{1a}(t)(\lambda_1 - \mu_1 - C(n_{1a}(t) + n_{1d}(t) + n_{1i}(t)) - \kappa\mu n_{1d}(t) - v n_{1i}(t).$$

Let us choose $\varepsilon > 0$ such that $\liminf_{t\to\infty} \kappa\mu n_{1d}(t) + v n_{1i}(t) > \varepsilon$. Then if for some $t > 0$ we have $n_{1a}(t) + n_{1d}(t) + n_{1i}(t) \geq \bar{n}_{1a}$, then we have

$$\frac{\mathrm{d}}{\mathrm{d}t}(n_{1a}(t) + n_{1d}(t) + n_{1i}(t)) < -\varepsilon.$$

Now, solutions of (3.13) are continuously differentiable thanks to the Picard–Lindelöf theorem, and hence we obtain that there exists $\delta > 0$ such that whenever $n_{1a}(t) + n_{1d}(t) + n_{1i}(t) \geq \bar{n}_{1a} - \delta$, we have

$$\frac{\mathrm{d}}{\mathrm{d}t}(n_{1a}(t) + n_{1d}(t) + n_{1i}(t)) < -\varepsilon/2.$$

This implies the time

$$t_{\bar{n}_{1a}-\delta} = \inf\left\{t \geq 0 \colon n_{1a}(t) + n_{1d}(t) + n_{1i}(t) < \bar{n}_{1a} - \delta\right\}$$

is finite, and for all $t > t_{\bar{n}_{1a}-\delta}$ we have $n_{1a}(t) + n_{1d}(t) + n_{1i}(t) \leq \bar{n}_{1a} - \delta < \bar{n}_{1a}$. Thus, $\limsup_{t\to\infty} n_{1a}(t) + n_{1d}(t) + n_{1i}(t) < \bar{n}_{1a}$.

Finally, the asymptotic upper bound on $n_2(t)$ as $t \to \infty$ is the analogue of [BK98, Lemma 2.3] in our model. Relying on the already proven parts of Corollary 3.12, we can now provide a short proof for it. Recall that under the assumptions of the corollary we have

$$\limsup_{t\to\infty} n_{1a}(t) + n_{1d}(t) + n_{1i}(t) < \bar{n}_{1a}, \qquad \liminf_{t\to\infty} n_j(t) > 0, \forall j \in \{1a, 1i, 2\},$$

and hence there exists $\beta > 0$ such that

$$\limsup_{t\to\infty} n_{1i}(t) \leq \bar{n}_{1a} - \beta.$$

Thus, we obtain for all t sufficiently large

$$\dot{n}_2(t) = -(1-q)D n_{1a}(t) n_2(t) + m v n_{1i}(t) - \mu_2 n_2(t) < m v(\bar{n}_{1a} - \beta) - \mu_2 n_2(t). \tag{E.1}$$

This shows that for such t, if $n_2(t) \geq \frac{m v(\bar{n}_{1a}-\beta)}{\mu_2}$, then $s \mapsto n_2(s)$ is decreasing at t. Consequently,

$$\limsup_{t\to\infty} n_2(t) \leq \frac{m v(\bar{n}_{1a} - \beta)}{\mu_2} < \frac{m v \bar{n}_{1a}}{\mu_2},$$

as wanted. □

[39]The ω-limit set is the set of points to which $\mathbf{n}(t)$ accumulates along a sequence of diverging times, and it is a well-known result that this set is *invariant*, i.e. starting from this set at time 0, the solution will stay in this set for all positive and even for all negative times.

F Declaration of exercise sheets (Frankfurt, 2024)

For the course in Frankfurt in July 2024, the exercises in these lecture notes have to be grouped into exercise sheets for official reasons. The content of the six exercise sheets is the following:

1. **Basics of Lotka–Volterra type ODEs and invasion fitnesses**: Exercises 1–4.
2. **The dynamical system corresponding to the competition-induced dormancy model**: Exercises 6–9.
3. **Stochastic aspects of the competition-induced dormancy model**: Exercises 5 and 10.
4. **The dynamical system corresponding to the virus model**: Exercises 11, 14, and 15.
5. **Stochastic aspects of the virus model**: Exercises 12, 13, and 16.
6. **The piecewise affine limiting process in the Champagnat–Méléard–Tran model and related convergence results**: Exercises 17–20.

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