Asymptotic profile in selection–mutation equations: Gauss versus Cauchy distributions

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A B S T R A C T

In this paper, we study the asymptotic (large time) behaviour of a selection–mutation–competition model for a population structured with respect to a phenotypic trait when the rate of mutation is very small. We assume that the reproduction is asexual, and that the mutations can be described by a linear integral operator. We are interested in the interplay between the time variable t and the rate ε of mutations. We show that depending on $\alpha > 0$, the limit $\varepsilon \to 0$ with $t = \varepsilon^{-\alpha}$ can lead to population number densities which are either Gaussian-like (when α is small) or Cauchy-like (when α is large).

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1. Introduction

1.1. Selection–mutation–competition models

The phenotypic diversity of a species impacts its ability to evolve. In particular, the importance of the variance of the population along a phenotypic trait is illustrated by the *fundamental theorem of natural selection* [15], and the *breeder's equation* [21]: the evolution speed of a population along a one dimensional fitness gradient (or under artificial selection) is proportional to the variance of the initial population. Recently, the phenotypic variance of populations has also come to light as an important element to describe the evolutionary dynamics of ecosystems (where many interacting species are considered) [27,4,26].

Over the last decade, the issue of *Evolutionary Rescue* emerged as an important question [3,10,17] (see also the seminal work of Luria and Delbrück [20]), and led to a new interest in the phenotypic distribution

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of populations, beyond phenotypic variance. Evolutionary Rescue is concerned with a population living in an environment that changes suddenly. The population will survive either if some individuals in the population carry an unusual trait that turns out to be successful in the new environment, or if new mutants able to survive in the new environment appear before the population goes extinct (see [22] for a discussion on the relative effect of *de novo mutations* and *standing variance* in Evolutionary Rescue). In any case, the fate of the population will not be decided by the properties of the bulk of its density, but rather by the properties of the tail of the initial distribution of the individuals, close to the favourable traits for the new environment. A first example of such problems comes from emerging diseases [16]: Animal infections sometimes are able to infect humans. This phenomenon, called zoonose, is the source of many human epidemics: HIV, SARS, Ebola, MERS-CoV, etc. A zoonose may happen if a pathogen that reaches a human has the unusual property of being adapted to this new human host. A second example comes from the emergence of microbes resistant to an antimicrobial drug that is suddenly spread in the environment of the microbe. This second phenomenon can easily be tested experimentally [3,24], and has major public health implications [9].

Most papers devoted to the genetic diversity of populations structured by a continuous phenotypic trait describe the properties of mutation–selection equilibria. It is however also interesting to describe the genetic diversity of populations that are not at equilibrium (*transient dynamics*): pathogen populations for instance are often in transient situations, either invading a new host, or being eliminated by the immune system. We refer to [18] for a review on transient dynamics in ecology. For asexual populations structured by a continuous phenotypic trait, several models exist, corresponding to different biological assumptions [11]. If the mutations are modelled by a diffusion, the steady populations (for a model close to (1), but where mutations are modelled by a Laplacian) are Gaussian distributions [19,6]. Furthermore, [1,14] have considered some transient dynamics for this model. In the model that we will consider (see (1)), the mutations are modelled by a non-local term. It was shown in [7] (see also [6]) that mutation–selection equilibria are then Cauchy profiles (under some assumptions), and this result has been extended to more general mutation kernels in [8], provided that the mutation rate is small enough. Finally, let us notice that the case of sexual populations is rather different, since recombinations by themselves can imply that a *mutation–recombination equilibrium* exists, even without selection. We refer to the infinitesimal model [5], and to [25] for some studies on the phenotypic distribution of sexual species in a context close to the one presented here for asexual populations.

In this article, we consider a population consisting of individuals structured by a quantitative phenotypic trait $x \in I$ (I open interval of $\mathbb{R}$ containing 0), and denote by $f := f(t, x) \geq 0$ its density. Here, the trait x is fully inherited by the offspring (if no mutation occurs), so that x is indeed rather a breeding value than a phenotypic trait (see [23]). We assume that individuals reproduce with a rate 1, and die at a rate

$$x^2 + \int_I f(t, y) \, \mathrm{d}y.$$

This means that individuals with trait $x = 0$ are those who are best adapted to their environment, and that the fitness decreases like a parabola around this optimal trait (this is expected in the surroundings of a trait of maximal fitness). It also means that the strength of the competition modelled by the logistic term is identical for all traits. When an individual of trait $x \in I$ gives birth, we assume that the offspring will have the trait x with probability $1 - \varepsilon$, and a different trait x' with probability $\varepsilon \in (0, 1)$. ε is then the probability that a mutation affects the phenotypic trait of the offspring. We can now define the growth rate of the population of trait x (that is the difference between the rate of *births without mutation*, minus the death rate) as

$$r_\varepsilon(t, x) = 1 - \varepsilon - x^2 - \int_I f(t, y) \, \mathrm{d}y.$$