

# **ORIGINAL ARTICLE**

# Mathematical model for adaptive evolution of populations based on a complex domain

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Population; Evaluation: Fractional calculus

Abstract A mutation is ultimately essential for adaptive evolution in all populations. It arises all the time, but is mostly fixed by enzymes. Further, most do consider that the evolution mechanism is by a natural assortment of variations in organisms in line for random variations in their DNA, and the suggestions for this are overwhelming. The altering of the construction of a gene, causing a different form that may be communicated to succeeding generations, produced by the modification of single base units in DNA, or the deletion, insertion, or rearrangement of larger units of chromosomes or genes. This altering is called a mutation. In this paper, a mathematical model is introduced to this reality. The model describes the time and space for the evolution. The tool is based on a complex domain for the space. We show that the evolution is distributed with the hypergeometric function. The Boundedness of the evolution is imposed by utilizing the Koebe function. © 2015 Production and hosting by Elsevier B.V. on behalf of King Saud University. This is an open access

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# 1. Introduction

Consider a population evolves agreeing with the method containing mutations and natural assortment, and some of its quantitative traits are modified progressively. The question is: what is the rate of this modification? The speed of evolution is critical in constant competition of classes and is of significant

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practical prominence in relation to present day phenomena such as, adaptation of endangered species to changing environmental conditions or adaptation of pathogens to existing methods of treatment. Measurable method of evolution dates back at least to Fisher's (1930) book, which enclosed his well-known "Fundamental Theorem of Natural Selection", affirming that the rate of increase of the mean fitness of a population at any moment of time, attributed to natural selection, equals the genetic variance of fitness of that population at that moment of time. The following question is, of course, what concludes this variance in the population fitness, and how to predict it?

Though diverse epigenetic and genetic methods stand elaborated in the development plus preservation of altered tissues, the evolution of population can be determined by the relative significance of an asymmetric and the symmetric cell differentiation, cell divisions and death. A central issue in evolutionary

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genetics is to calculate whether a population accrues damaging or beneficial mutations.

The equivalent proteins and genes stay perceived to be important for instruction of various tissues. This unity and conservation of straightforward procedures entail that their mathematical models can employ crossways the spectrum of pathological and normal growth. An androgynous population accumulates damaging mutations and, consequently, its fitness will grow. On the other hand, it newly rotated out that useful mutations are additional ample than formerly supposed.

One recognizes that the technique of demonstrating such schemes remains to utilize a discrete family of ordinary differential equations labeling dynamics of cells at various maturation phases and evolution between the phases. These consequently entitled multi-compartmental models stay founded by the condition that in every lineage of cell originators there occurs a discrete sequence of maturation phases, which are consecutively crossed. Nevertheless, it is besides flattering increasingly strong that the differentiated originator's system such sequence only beneath homeostatic assumptions. A devoted cell usually arranges a continuous sequence, which may include incremental phases, the measure of which could be changeable (Biktashev, 2014; Alfaro and Carles, 2014; Britton et al., 2015; Abasi et al., 2015; Gerleea and Altrock, 2015; Chao et al., 2015; Landguth et al., 2015). As an application, cell differentiation devoid of cell divisions is detected throughout neurogenesis. Furthermore, in certain tissues such as the mammary gland, different phases of differentiation stand not well recognized.

This clarification appeals not simply to the essential biological question of whether the cell differentiation is a continuous or a discrete development and what is the amount of cell difference, nevertheless correspondingly to how to select a suitable forming method. Is the pace of maturation (commitment) verbalized by continuous divisions, or is maturation a continuous development decoupled from proliferation? The conventional interpretation in ordinary hematopoiesis appears to be opposite. To discuss these questions and to consider the impact of potential continuous transformations of the differentiation procedure, one can impose a classical method based on partial differential equations of transport category and compare this method to its discrete complement. The argument of disappearance is a multi-compartmental method of a discrete gathering of cell subpopulations, which stood newly suggested in Marciniak-Czochra et al. (2009) and Doumic et al. (2011) to study the dynamics of the hematopoietic scheme with cell proliferation and differentiation structured by a nonlinear feedback loop.

Hitherto an additional category of evolution is selection. This income that various alleles could have various susceptibilities for resampling, for instance, various amounts at which they resemble. An auxiliary element might be migration, i.e., genetic substantial is communicated among various populations for the reason that the individuals booming this substantial travel from one population to the following.

Analysis of populations has approximately continuously depended on processes founded on estimated gene identities or heterozygosities, because of these connections to variance and the binary nature of sexual reproduction and diploid inheritance. The corresponding processes and their numerous simplifications for divided populations have likewise played a central role in evolutionary biology and population genetics. This method highlights the frequent alleles by introducing in them much more weight than their population fraction, and multi-level hierarchical additive partitioning is not typically likely with heterozygosity-based measures (Landguth et al., 2015).

Investigators in numerous castigations have progressively documented that variety inside populations and compositional differentiation between populations cannot be completely categorized by a single measure. For example, ecologists have touched a consensus (Chao et al., 2014) that instead of one or a few diversity measures, it is best to practice a multifaceted diversity measure parameterized to totally describe the class abundance distributions in ecological assemblages. By analogy, moreover to measures based on heterozygosity, complementary abundance-sensitive measures that are sensitive to less frequent alleles are needed to portray a more complete picture of allele frequency distribution or differentiation among populations.

Mathematics is frolicking an ever more significant character in the physical and biological sciences. The technique followed in most texts on these topics (e.g., electrodynamics, quantum mechanics, classical mechanics, modern physics, mathematical biology, chemical biology, etc.) is forum-getting at the problem as a differential equation that is associated with one of several special differential equations (Bessel's, Hermite's, Legendre's, Laguerre's, etc.). All the above equations have solutions in term of special functions. The most important special function is the hypergeometric function (Seaborn, 1991)

$$pF_q(a_1,\ldots,a_p;b_1,\ldots,b_q;z) = \sum_{n=0}^{\infty} \frac{(a_1)_n \dots (a_p)_n}{(b_1)_n \dots (b_q)_n} \frac{z^n}{n!},$$

where  $(x)_n$  is the Pochhammer symbol. The hypergeometric function is utilized to test, classify and analyze various types of biological process (Gurarie and King, 2014).

In this paper, a mathematical model is presented for this certainty. The method designates the time and space for the evolution. The tool is based on a complex domain for space. Therefore, we utilize some of the concepts in geometric function theory, such as univalent function. We show that the evolution is distributed with the hypergeometric function. The Boundedness of the evolution is imposed by utilizing the Koebe function. This new method allowed us to understand the fitness of the population geometrically. The changing with respect to time and space is formulated by employing the concept of fractional calculus in real as well as in a complex domain.

## 2. Material and methods

In this section, we select some recent mathematical models.

#### 2.1. Adaptive dynamics

Adaptive dynamics is essentially apprehensive with qualitative questions such as stability of evolution, the direction of evolution, steady states and speciation due to branching. On the quantitative stage, the fundamental for adaptive dynamics is the following canonical equation:

$$\frac{dM}{dt} = \kappa(M) \frac{dr(M, x)}{dx} \bigg|_{x=M},$$

where *M* is the average value of the trait at time *t*, r(M,x) measures the fitness of characters with trait value *x* in the environment of resident trait values *M* and the coefficient  $\kappa(M)$  is defined as a non-negative coefficient.

### 2.2. Stochastic model

Stochastic model is concerned about the evolution and the competitive omission principle, that at each instant of time, the selection decreases the population to a certain category, which though changes in time due to random mutations. This model is imposed and vindicated in, consuming a stochastic model, under certain asymptotic statements about the mutation rate. Faintly simplifying, the key assumption is that mutations are accordingly rare that for a given population size, there is adequate time between consecutive mutations for the whole population to change to the new trait value if it is fitter than the previous.

#### 2.3. Quantitative genetics

Quantitative genetics are establishing a number of its own methods and studied complicated problems associated with a quantitative description of evolution. One method is through the technique of moments, which reflected multilocus determination of a quantitative trait in a sexually duplicating population, and in specific offered an infinite chain of ordinary differential equations for the moments of allelic distribution.

# 2.4. Gaussian distribution

Gaussian distribution is considered. This method analyzed the speed of evolution, though far from any evolutionary stable state, based on the simplest possible expressive method. This is a deterministic integro-partial differential equation, which is analogous to numerous procedures assumed or derived elsewhere. In addition, the author provided a simple derivation of this model from, avoiding to sort non-verifiable conditions, for terror that the final outcomes may convert artifacts of any such conditions. The applied efficacy of the model is demonstrated by giving that treatment of a more faithful model through asymptotic methods

$$\frac{dM}{dt} = [r(x) - \bar{M}(t)]M + \frac{\partial}{\partial x} \left[ C(x)M + D(x)\frac{\partial M}{\partial x} \right]$$
$$\bar{M} = \int_{-\infty}^{\infty} M(t, x) dx.$$

#### 2.5. The Replicator-mutator

The Replicator-mutator is a model that considered a class of nonlocal reaction–diffusion problems. The authors made a difficult and detailed analysis of the Cauchy problem associated with

$$\partial_t M = \partial_{xx} M(x - \bar{M})M, \quad \bar{M} = \int_{-\infty}^{\infty} x M(t, x) dx$$

Indeed, they showed that it can be reduced to the heat equation, and therefore calculate its solution explicitly. This allowed to designate a selection of comparing behaviors depending on the initial data.

#### 2.6. The proposed method

Our aim is to introduce a new model of evolution based on fractional calculus in real and complex domains. Our model takes the form

$$D_t^{\alpha} M(t,z) = \kappa(M) D_z^{\beta} M(t,z), \tag{1}$$

with

$$(M(0,0)=0, \quad \alpha \in (0,1], \ \beta \in (1,2], \ t \in [0,T], \ z \in U),$$

where U is the open unit disk,  $D_t^{\alpha}$  is the Riemann–Liouville fractional differential operator of order  $0 < \alpha < 1$ ,

$$D^{\alpha}f(t) = \frac{d}{dt}\int_{a}^{t} \frac{(t-\tau)^{-\alpha}}{\Gamma(1-\alpha)}f(\tau)b\tau.$$

Corresponding to the fractional integral operator for a continuous function f(t) of order  $\alpha > 0$ ,

$$f_a^{\alpha} f(t) = \int_a^t \frac{(t-\tau)^{\alpha-1}}{\Gamma(\alpha)} f(\tau) d\tau.$$

If the above operators are defined in a complex domain for analytic function f, then they are called the Srivastava–Owa operators (Podlubny, 1999; Srivastava and Owa, 1989)

$$D_{z}^{\beta}f(z) = \frac{d}{dz} \int_{0}^{z} \frac{(z-\vartheta)^{-\beta}}{\Gamma(1-\beta)} f(\vartheta) b\vartheta, \quad z \in U.$$

And

$$I^{eta}f(z) = \int_{0}^{z} rac{(z-artheta)^{eta-1}}{\Gamma(eta)} f(artheta) dartheta.$$

Remark 2.1.

$$D_t^{\alpha} t^{\lambda} = rac{\Gamma(\lambda+1)}{\Gamma(\lambda-\alpha+1)} t^{\lambda-\alpha}, \quad \lambda > -1; \quad 0 \leqslant \alpha < 1$$

and

$$I^{\alpha}t^{\lambda} = rac{\Gamma(\lambda+1)}{\Gamma(\lambda+\alpha+1)}t^{\lambda+\alpha}, \ \lambda > -1; \ \alpha > 0.$$

We need the following result, which can be found in Ibrahim and Jalab (2013) and Ibrahim et al. (2015):

**Lemma 2.1.** Let M(t,z) be a univalent function (one to one) in the unit disk for all  $t \in [0, T], z \in U$ . Then

$$\begin{split} |D_{z}^{\beta}M(t,z)| &\leq \frac{r^{-\mu}}{\Gamma(1-\mu)} (rF((2)_{n},(1)_{n};(1-\mu)_{n};rt))', \\ 1 &< \beta = 1+\mu \leq 2 \\ \left(' := \frac{d}{dz}, \ r = |z|; \quad z \in U \setminus \{0\}\right), \end{split}$$

where the equality holds true for the Koebe function

$$f(z) = \frac{z}{(1-z)^2}, \quad z \in U.$$

Our aim is to find an approximate solution for the Eq. (1).

**Theorem 2.1.** Consider the initial differential Eq. (1). If  $|\kappa(M)| < \varepsilon, \varepsilon > 0, \beta = \mu + 1, \mu \in (0, 1]$  and M(t,z) is univalent in *U*, then Eq. (1) has an approximate solution to the hypergeometric function

$$M(t,r) \approx \frac{\varepsilon}{r^{\mu} \Gamma(1-\mu) \Gamma(1+\alpha)} t^{\alpha} (rF((2)_n, (1)_n, (1)_n; (1-\mu)_n, (1+\alpha)_n; rt))'.$$

**Proof.** By using the upper bound of the fractional differential operator (Lemma 2.1), we have

$$\begin{split} D_t^{\alpha} M(t,z) &\approx \frac{\varepsilon r^{1-\mu}}{\Gamma(1-\mu)} (rF((2)_n,(1)_n;(1-\mu)_n;rt))' \\ &= \frac{\varepsilon r}{r^{\mu} \Gamma(1-\mu)} \sum_{n=0}^{\infty} \frac{(2)_n (1)_n}{(1-\mu)_n} \frac{n+1}{n!} (rt)^n. \end{split}$$

Operating the above equality, by  $I^{\alpha}$  and applying some properties of the fractional calculus (Remark 2.1), yields

$$\begin{split} M(t,r) &= \frac{\varepsilon r}{r^{\mu} \Gamma(1-\mu)} \sum_{n=0}^{\infty} \frac{(2)_{n}(1)_{n}}{(1-\mu)_{n}} \frac{n+1}{n!} r^{n} \frac{\Gamma(n+1)}{\Gamma(n+1+\alpha)} t^{n+\alpha} \\ &= \frac{\varepsilon r}{r^{\mu} \Gamma(1-\mu) \Gamma(1+\alpha)} t^{2} \sum_{n=0}^{\infty} \frac{(2)_{n}(1)_{n}(1)_{n}}{(1-\mu)_{n}(1+\alpha)_{n}} \frac{n+1}{n!} (rt)^{n} \\ &= \frac{\varepsilon r}{r^{\mu} \Gamma(1-\mu) \Gamma(1+\alpha)} t^{2} (rF((2)_{n},(1)_{n},(1)_{n};(1-\mu)_{n},(1+\alpha)_{n};rt))', \end{split}$$

where ':=d/dz. Hence, the proof.  $\Box$ 



**Figure 1** The solution of Eq. (1) for various values of  $\mu$ , where  $\alpha = \mathcal{C} = t = 1$ . Mature cells evolution with time-space distribution of cell density along the maturation level.

#### 3. Results

Eq. (1) has a converged solution in a complex domain. The solution can be approximated by a hypergeometric function. This leads to a stable evolution of the population determining by changing both time and space. Founding of the self-similar result in (2) outlines of population in the trait space at selected times during initial transient following an initial point (0,0). Parameters of the system are:  $0 < \alpha \le 1$ ,  $1 < \beta \le 2$  and  $\varepsilon > 0$  (the maximum value of the non-negative coefficient of the system). Numerical simulation on the interval  $r \in [0, 1)$ with Neumann boundary conditions (simulation with Dirichlet boundary conditions or wider interval produces indistinguishable results) is explained. We tension here that the functional form (2) is not an arbitrary assumption, but an exact consequence of the evolution Eq. (1), once appropriate initial conditions are provided. These initial conditions would be some type of Gaussian special function. Though, numerical models shown in Fig. 1 propose that the general solution at arbitrary initial distributions asymptotically grows into normal as time increases, so the special class (2) must in fact be completely illustrative. We comprehended that this result can be reflected as a positive, bounded, and stable solution in the unit disk. We could employ this method on well known fractional diffusion equations likewise the fractional wave equation in a complex domain. A reminder that the hypergeometric function includes the Mittag-Leffler function (Ibrahim and Jalab, 2013; Ibrahim et al., 2015). 3-Dimensional form of solution is imposed in Fig. 2, where  $\alpha = \mu = 0.5$  and  $\alpha = 0.9$ ,  $\mu = 0.5$ , respectively. The maximal solution is in the boundary of the unit disk at t = 0.4 and  $\varepsilon = 1$ .

# 4. Discussion

To comprehend the modification of the fractional models, we derived an approximation equation for the time–space fractional differential model supposing that a continuum of different phases can be demarcated. The fractional power in both time and space is delivered by the information that the fractional differentiation is organized by intracellular biological developments, which remain definitely continuous in time, at least when be close to over a huge total of cells. Accordingly, designed for the appropriate time scaling, we have to take responsibility that commitment and maturation of cell progenitors do not progress by the separation clock (one step in the



Figure 2 The solution of Eq. (1) when  $\alpha = \mu = 0.5$  and  $\alpha = 0.9$ ,  $\mu = 0.5$ .

maturation process = one division) nonetheless is a continuous procedure and can be considered between the divisions. This statement clarifies the essential difference between the two models (fractional model and ordinary model). The organized population model (fractional formula) is certainly a boundary of the ordinary equation with the changes between compartments connected to the division of the cells. Conversely, the simulations can show precisely the identical dynamics for an appropriate choice of the maturation rate function.

# 5. Conclusion

We formulated properties of the solution to (1) for a wide class of initial conditions, which generalized (by employing the concept of fractional calculus for both time and space) and extended (by utilizing the complex domain in the unit disk) the properties of the approximate solutions. We did that in terms of the special function called the hypergeometric function, which involve so many well known functions such as the exponential function. The proposed model described in continuous phenotypic trait space and continuous time. We did not need the equilibrium point of the system. That is the model stabilized far away from evolutionary stable equilibrium. This is the first discussion of the stability of a system without its equilibrium point. The hypergeometric function was a powerful trait in the population and emerges spontaneously during the course of evolution, as stated in Theorem 2.1. This eliminates the need for any artificial closing procedures in the fractional differential equations in a complex domain. In biological standings, the growth of the Eq. (1) in the progress of evolution is owed to the absence of stabilizing selection in the simplified version of our model. Stabilizing selection can stop that growth, as illustrated by the stationary solution of Eq. (2) around a local optimum in the fitness landscape  $(T \rightarrow \infty)$ . Additionally, the action of the hypergeometric function is needed to obtain an asymptotic approximation of the model.

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