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MARKOV AND NON-MARKOV HEREDITARY PROCESSES IN ASEXUAL AND RANDOM MATING SEXUAL POPULATIONS

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Abstract. In this paper, we numerically investigate the Hereditary processes in the sexual and asexual mating. The genetic diffusion models of the two cases are described by partial differential equations. The solutions of these equations are considered as the conditional probability of finding the specific genes at a certain generation with a certain frequency. We also investigate the effects of the selection and mutations as well as the dependence on the memory on the sexual random mating and on the self proliferation of the asexual case. The effects of the wild-type mutation rate and mutator mutation rate per genome on the asexual proliferation is also numerically discussed. To do so, the common finite difference rules (FDM) will be utilized. The convergence of the discrete approximate solutions on the short and long run, i.e. the dependence on the memory, are also discussed and numerically investigated. The reversibility property for both models is theoretically and numerically discussed. Finally, a concrete conclusion will be given to summarize our numerical results and their relations to the real life problems.

1. Introduction

The evolution of a population of a certain species is affected by whether the individual organisms are reproduced sexually or asexually. In asexual reproduction, an exact genetic copy of the parent organism is produced (a clone). Unlike sexual reproduction, Asexual reproduction only introduces genetic variation into the population if a random mutation in the organism's DNA is is passed on to the offspring.

Sexual reproduction involves the fusion of the nuclei of a male and female sex cell during fertilisation. The offspring inherit a mixture of alleles from both parents. Sexual reproduction has some advantages such as, it produces genetic variation in the offspring, the species can adapt to new environments due to variation, which gives them a survival advantage and the diseases are less likely to affect all the individuals in a population.

In asexual reproduction the population can increase rapidly when the conditions are favourable, only one parent is needed. This process is more time and energy efficient as it does not require a mate and it is faster than sexual reproduction. The disadvantages of asexual reproduction are given as, it does not lead to genetic variation in a population and the disease may affect all the individuals in of the next population.

Bacteria, such as E. coli, are reproduced asexually. An advantage of this is that they can produce many bacteria very quickly. A disadvantage is that all of the bacteria are genetically identical. If an antibiotic was put on the bacteria, then all of them would die. The only way for variation to be introduced into the population is by random mutation.

According to Kimura and Crow [1], asexual population has two favourable mutants can be incorporated into the population only if one occurs in a descendant of the individual in which the other occurred, while in a sexual population both mutants can be incorporated through recombination.

Later, Kimura [2] considered evolutionary factors which influence mutation rates through natural selection and discussed the mechanisms by which spontaneous mutation rates are adjusted in the course of evolution.

The rates of evolution in the asexual and the sexual population were compared by J. Maynard Smith [3]. It is shown that, if the genetic variance of the population is generated by mutation in a uniform environment, sexual reproduction confers no advantage. But if the genetic variance has arisen because of the selection that has favoured different genotypes in different environments, then sexual reproduction will accelerate

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adaptation to a new environment. These conclusions differ sharply from those reached by Crow and Kimura [1]. The difference arises because those authors treated mutations as unique events, whereas they are here treated as recurrent events.

The rate of evolutionary adaptation of sexual and asexual populations is compared in [4]. If N is the population size, l the number of loci at which at any time favourable mutations are possible but have not yet occurred, u the mutation rate per locus and s the selective advantage per locus, sexual reproduction will not accelerate evolution if $12\,Nu \ll \ln 20Ns$, or, very approximately, if $N \ll 110u$. For larger populations sex does accelerate evolution, by a factor very approximately equal to l.

In 1997, Taddei et al [5] considered the following question: whether high mutation rates m play an important role in adaptive evolution in models of large, asexual, clonal populations?. The answer is: by adapting to a new environment, the strong mutator genes (such as those that increase mutation rates by 1, 000-fold) can accelerate adaptation, even if the mutator gene remains at a very low frequency (for example, 10^{-5}. Less potent mutators can become fixed in a fraction of finite populations.

The main aim of this paper is to study the approximate solutions of the partial differential equations that mathematically model the sexual and asexual genetic hereditary processes. The need for extending the studied classical partial differential equations to time fractional differential equations is also studied. To find the approximate solutions of these models, the standard finite difference methods and the Grünwald-Letnikov scheme are implemented. Therefore, the paper is arranged as follows: The introduction is given in section 1. In section 2, a short survey of the classical genetic diffusion processes with drift in sexual and Asexual population are presented to examine the theory of the Markov processes. In section 3 the approximate solutions of the studied classical partial differential equations are given. In section 4, We study the time factional differential equations as Non-Markov processes to obtain the approximate solution of the time-fractional diffusion equation of the sexual population by applying the Grünwald-Letnikov scheme. In section 5, the convergence to the stationary discrete approximate solutions and the reversibility of the discussed processes. Finally, section 6 is to simulate the time evolution of the approximate solutions, the numerical comparison and the lose of number of genes is also studied.

2. A Brief History on Genetic Drift Equations of Sexual and Asexual Population

A large number of mathematical models to discuss the diffusion in genetics with drift for sexual population are given by biologists. The most famous scientist who was studying the genetic drift is Kimura [12],[13] and [14]. He supposed that the random mating population of size N, in which two allele A and a happen with respective frequencies x and $1-x$ with s being the selective advantage of A over a. He derived the partial differential equation which describes the evolution of genes across generations of sexual population and named it *genetic drift* equation that reads

$$\frac{\partial u(p,x;t)}{\partial t} = \frac{1}{2}\frac{\partial^2}{\partial x^2}\{V(x,t)u(p,x;t)\} - \frac{\partial}{\partial x}\{M(x,t)u(p,x;t)\}, \tag{1}$$

with boundary conditions

$$u(p,0;t) = u(p,1;t) = 0 \ ,$$

and initial condition

$$u(p,x;0) = \delta(x-p) \ ,$$

where p is the initial frequency of the gene, $M(x,t) = sx(1-x)$ is the average rate of change in x per generation, $V(x,t) = \frac{1}{2N_e}x(1-x)$ is the variance under the mutation impact and $N_e(t)$ is the effective population size at t. For more detail about the equation (1), see [15].

In 1965, Kimura and Crow [1] were the first scientists made a comparison between sexual and asexual populations. They was interested in doing experimental data on chloramphenicol resistance in bacteria. After that, Kimura [2] discuss how spontaneous mutation rates influence the evolution of Asexual population. Through many years later, many researchers had talk about different types of mutation rates experimentally but no one tries to simulate the diffusion in genetic of Asexual population.

C. Scott Wylie et al. [8] introduced the idea being the mutator is an allele that increases the mutation rate throughout the genome by disrupting some aspect of DNA replication or repair. They assume that the fixation of mutator alleles is limited by (i) competition with mutations in wild-type backgrounds, (ii) additional deleterious mutational load, and (iii) random genetic drift. They used a multiple-locus model (i.e. the partial differential equation that contain population size N, beneficial and deleterious mutation rates, and the strength of mutations s) and employed both simulation and analytic methods to investigate the effects of these three factors on the fixation probability P_{fix} of an initially rare mutator. Their simulations were also shown that effect (i) is typically small for strong-effect mutators. After C. Scott Wylie et al. [8], no researchers go through simulation of the genetic diffusion of Asexual population.

The main parameters of adaptation in the asexual reproduction are: the size of the adapting population and the height and steepness of the adaptive peak characterizing adaptation. By simulation approach, Tenaillon et. al [6] studied the effect of these parameters on the selection of mutators in asexual populations, assuming additive fitness. they show that the larger the population size, the more likely the fixation of mutator alleles.

Travis and et. al [7] examines how mutator dynamics vary according to the frequency of environmental fluctuations. They demonstrate that as each beneficial mutation provides a greater gain in fitness, mutators achieve higher densities in more rapidly fluctuating environments. They also show that mutators of intermediate strength reach higher densities than very weak or strong mutators. The partial differential equation that mathematically models the genetic diffusion of the asexual reproduction reads

$$\begin{aligned}\frac{\partial u(p,x;t)}{\partial t} &= \frac{1}{N}\frac{\partial^2}{\partial x^2}\{x(1-x)u(p,x;t)\} \\ &+ (\mu_+ - \mu_-)[1-\alpha(1-s)]\frac{\partial}{\partial x}\{x(1-x)u(p,x;t)\} \\ &- N\alpha s[x\mu_+ + (1-x)\mu_-]u(p,x;t)\ ,\end{aligned} \tag{2}$$

where N is the total population size, μ_+ is the mutator mutation rate per genome, μ_- is the wild-type mutation rate per genome, α is the fraction of beneficial mutations and s is the selection coefficient of mutation. Each of the three lines in equation (2) has a straightforward physical interpretation. The first line represents *random genetic drift*. The second line represents the mutational load of the mutator. The final line represents the *decay* of probability from the open interval $x \in (0,1)$ due to beneficial mutations. The values of the above mutation rates are given in two cases: the 1^{st} one is $\mu_- = 0$ and $\mu_+ = 0.01$, while the 2^{nd} case is $\mu_+ = 0.01$ and $r \equiv \frac{\mu_+}{\mu_-}$ where r is the mutator strength. According to Travis et al [7], they found that mutators of intermediate strength reach higher densities than very weak or strong mutators, i.e. $r = 10$ is the best to give high density. When mutator strength is low, mutators never reach a high density and thus have little impact on the evolution of the population to a changing environment. Intermediate strength mutators can reach high density. These mutators cause enough genetic variability to enable the population to adapt to a change in the environment. Hitch-hiking on the well-adapted alleles, they reach a high density within the population. At higher mutator strengths, mutators do not reach such a high density in which genes can have a large impact on the adaptation of an organism to a novel environment even though they may remain at a low density.

Â

3. Discretization of the Classical Genetic Drift Equation of the Sexual Population

In this section, we use the common standard finite-difference method (FDM) to find the approximate solution of genetic drift equation (1) that represents hereditary process of genes in a sexual population. The independent variables (x,t) are discretized by the following grid points

$$x_j = jh,\ h > 0,\ t_n = n\tau,\ \tau > 0,\ ,$$

where $j \in (1,R)$, $h = 1/R$, $R \in \mathbb{N}$ and $n \in \mathbb{N}_0$. Take the $y_j^{(n)}$ as the clump vector to approximate the function $u(x,t)$ being the solution of equation (1). $u(x,t)$ represents the conditional probability function of finding the allele A with frequency x at the generation time t, see [16],[17] asÂ

$$y_j(t_n) \approx \int_{x_j-h/2}^{x_j+h/2} u(x,t_n)dx \approx hu(x_j,t_n)\ ,$$

DefineÂ the $y^{(n)}$ vector by

$$y^{(n)} = \{y_0^{(n)}, y_1^{(n)}, \cdots, y_{R-1}^{(n)}, y_R^{(n)}\}^T\ ,$$

where $y^{(n)} = y(t_n)$ is a discrete column probability vector of finding the A allele at the t_n generation with the frequency x_j. It is better to choose the initial value $y^{(0)}$ such as $\sum_{j=0}^{R} y_j^{(0)} = 1$. $y_j^{(n)}$ must satisfy also that $\sum_{j=0}^{R} y_j^{(n)} = 1\ \ \forall n \in \mathbb{N}_0$, i.e. there is no loss of any of the conditional probability $u(x,t)$ at any generation.

Apply the standard finite difference method at equation (1), to get

$$\begin{aligned} y_j^{(n+1)} &= \{(\frac{\mu}{4N_e}+\frac{\mu s}{2R})h(j-1)(1-(j-1)h)\}y_{j-1}^{(n)}+ \\ &\{1-\frac{2\mu jh(1-jh)}{4N_e}\}y_j^{(n)}+ \\ &\{(\frac{\mu}{4N_e}-\frac{\mu s}{2R})h(j+1)(1-(j+1)h)\}y_{j+1}^{(n)}+O(h^2+\tau)\ , \end{aligned} \tag{3}$$

where $R=\frac{1}{h}$, $\mu=\frac{\tau}{h^2}$.

In order to preserve the positivity in the equation (3), it is required that

$$0<\mu\leq\frac{2N_e}{\max[jh(1-jh)]}=8N_e\ . \tag{4}$$

See [15] for more detail about the stability of the approximate solution nand the description of $y_j^{(n+1)}$. Equation (3), is written in matrix form as:

$$y^{(n+1)}=P^T.y^{(n)}\ ,\ 1\leq j\leq R-1\ , \tag{5}$$

where $P=(p_{ij})$ is $R-1\times R-1$ matrix with $i,j=1,\ldots,R-1$ which take the form:

$$P_{ij}=\begin{cases} P_{ij}^{(1)}=\mu h(j-1)(1-h(j-1))(\frac{(1.0}{4N_e}+\frac{s}{2R}), & i=j-1,\ j=1,\cdots,R-1 \\ P_{ij}^{(2)}=1-\frac{2\mu jh(1-jh)}{4N_e} & i=j,\ j=1,\cdots,R-1 \\ P_{ij}^{(3)}=(j+1)h(1-(j+1)h)(\frac{1}{4N_e}-\frac{s}{2R}), & i=j+1,\ j=1,\cdots,R-1\ . \end{cases} \tag{6}$$

It is more appropriate for the numerical calculation to write the matrix P in the form

$$P=(I+\mu H)\ , \tag{7}$$

where I is the unit matrix and $H=\{H_{ij}\},i,j\in,[R-1]$ is a square matrix whose rows have the property that the summation over each one of them is equal zero as

$$H_{ij}=\begin{cases} H_{ij}^{(1)}=h(j-1)(1-h(j-1))(\frac{(1.0}{4N_e}+\frac{s}{2R}), & i=j-1,\ j=1,\cdots,R-1 \\ H_{ij}^{(2)}=\frac{-2jh(1-jh)}{4N_e} & j=i,\ j=1\cdots,R-1 \\ H_{ij}^{(3)}=(j+1)h(1-(j+1)h)(\frac{1}{4N_e}-\frac{s}{2R}), & i=j+1,\ j=1,\cdots,R-1\ . \end{cases} \tag{8}$$

The matrix P is a stochastic matrix and with the aid of the scaling relation condition (4), one has diagonally dominant non-negative matrix and M-Matrix and that ensures the stability of the approximate solutions, see [18] and [19] for more details about the positive stable matrices. For purpose of computing, it is recommended to take the transpose of each side of (5), to rewrite equation (5) by using equation (7) as

$$z^{(n+1)}=z^{(n)}.(I+\mu H)\ , \tag{9}$$

where $z^{(n)}=(y_j^{(n)})^T$. The matrix H has the property that the summation over all its rows is zero. In the the numerical results section, we give the time evolution of this genetic diffusion case over many generations and plot $\sum_{j=1}^{R-1}y_j^{(n)}\ ,\forall n\in\mathbb{N}_0$ to show that, for this Markov case, there is lose of unity as $n>>1$. In the the numerical results section, we give the time evolution of this genetic diffusion case over many generations and plot $\sum_{j=1}^{R-1}y_j^{(n)}\ ,\forall n\in\mathbb{N}_0$ to show that, for this Markov case, there is lose of unity as $n>>1$.

3.1. **The Discretization of the Classical Genetic Drift Equation of the Asexual Population.** By applying the previous FDM to the diffusion in genetic equation (2) of Asexual colony of bacteria, one gets the discretized solution

$$\begin{aligned} y_j^{(n+1)} &= \{(\frac{\mu}{N}-\frac{\mu(\mu_+-\mu_-)[1-\alpha(1-s)]}{2R})h(j-1)(1-(j-1)h)\}y_{j-1}^{(n)} \\ &+\{1-\frac{2\mu jh(1-jh)}{N}-\frac{c\alpha\mu}{R^2}\{\mu_+jh+\mu_-(1-jh)\}\}y_j^{(n)} \\ &+\{(\frac{\mu}{N}+\frac{\mu(\mu_+-\mu_-)[1-\alpha(1-s)]}{2R})h(j+1)(1-(j+1)h)\}y_{j+1}^{(n)}+O(h^2+\tau)\ , \end{aligned} \tag{10}$$

where $R=\frac{1}{h}$, $\mu=\frac{\tau}{h^2}$.

The preservation of the non-negativity and the stability of the approximate solution defined in equation (10) requires that

$$\begin{aligned} 0 < \mu &\leq \frac{NR^2}{\max[2R^2jh(1-jh) + Nc\alpha\{jh\mu_+ + (1-jh)\mu_-\}]} \\ &= \frac{4NR^2}{2R^2 + N(\mu_+ + \mu_-)} \approx 2N, \;\; N(\mu_+ + \mu_-) < 1 \; . \end{aligned} \tag{11}$$

The matrix form of the equation (10) can be written as:

$$y^{(n+1)} = P^T.y^{(n)} \, , \, 1 \leq j \leq R-1 \, , \tag{12}$$

where a $P = (p_{ij})$ is $R-1 \times R-1$ matrix with $i, j = 1, \ldots, R-1$ which take the form:

$$P_{ij} = \begin{cases} P_{ij}^{(1)} = (\frac{\mu}{N} - \frac{\mu(\mu_+ - \mu_-)[1-\alpha(1-s)]}{2R})h(j-1)(1-(j-1)h), & i = j-1, \, j = 1, \cdots, R-1 \\ P_{ij}^{(2)} = 1 - \frac{2\mu jh(1-jh)}{N} - \frac{c\alpha\mu}{R^2}\{\mu_+ jh + \mu_-(1-jh)\}, & i = j, \, j = 1, \cdots, R-1 \\ P_{ij}^{(3)} = (\frac{\mu}{N} + \frac{\mu(\mu_+ - \mu_-)[1-\alpha(1-s)]}{2R})h(j+1)(1-(j+1)h), & i = j+1, \, j = 1, \cdots, R-1 \; . \end{cases} \tag{13}$$

Again the matrix P is a stochastic matrix as the summation over all its rows is 1 and is also a diagonally dominant non-negative matrix if and only if the value of μ is controlled by the condition (11). One can rewrite the matrix equation (12) as the matrix equation (9) by defining the matrix H as

$$H_{ij} = \begin{cases} H_{ij}^{(1)} = (\frac{1}{N} - \frac{(\mu_+ - \mu_-)[1-\alpha(1-s)]}{2R})h(j-1)(1-(j-1)h), & i = j-1, \, j = 1, \cdots, R-1 \\ H_{ij}^{(2)} = \frac{-2jh(1-jh)}{N} - \frac{c\alpha}{R^2}\{\mu_+ jh + \mu_-(1-jh)\}, & j = i, \, j = 1 \cdots, R-1 \\ H_{ij}^{(3)} = (\frac{1}{N} + \frac{(\mu_+ - \mu_-)[1-\alpha(1-s)]}{2R})h(j+1)(1-(j+1)h), & i = j+1, \, j = 1, \cdots, R-1 \; . \end{cases} \tag{14}$$

The matrix H has the property that the summation over all its rows is zero. Take the transpose of each side of the matrix equation (12) and use equation (14), you can rewrite this system of equation in the form of the matrix equation (9). The time evolution of this genetic diffusion with drift over many generations are simulated and compared for different values of generations, in section 6.

4. Extending to the Time-Fractional Genetic Drift Equation of Sexual Population and its Approximate Solution

In the Markov mechanism, it does not matter if the parents inherit their characters (genes) from the grandparentsÂ or the ancestorsÂ of the same family. The Markov processes have an exponential waiting time and the exponential distribution has a memoryless property. All Â the Â research papers that discussed the sexual genetic populationÂ Â have explained that some of the genes are lost during the succeeding generations. Unlike Asexual population. All the old research papers, in this field, discussed the classical partial differential equations that contains the term $\frac{\partial u(x,t)}{\partial t}$, where $u(x,t)$ is the solution of the partial differential equation (1) or equation(2). As one see in the last section the discrete forward in time scheme of $\frac{\partial u(x,t)}{\partial t}$ reads

$$\frac{\partial u(x,t)}{\partial t} = \frac{y_j^{(n+1)} - y_j^{(n)}}{\tau} \; . \tag{15}$$

That means the discrete conditional probability of finding the allele A at the next generation with frequency $0 < x < 1$ depends only the current generation. This has no sense as a child may be born with green eye while both his father and mother have black eyes. This child inherits his eye colour from his grand parents $y_j^{(n-1)}$, or his grand grand parents $y_j^{(n-2)}$, and so on. This is explained as the hereditary process of the child characteristics depends on the past or in other words the process has a memory. So it is not important to use the Non-Markov case to keep the memory property of genes inside the family. Only the Caputo time-fractional operator can reflect the dependence on the memory. So far, replace the first order time derivative operator $\frac{\partial}{\partial t}$ on the genetic drift equation (1) by the Caputo time fractional operator of order $0 < \beta < 1$ that reads

$${}_{t}^{}D_{*}^{\beta} f(t) = \begin{cases} \frac{1}{\Gamma(m-\beta)}\{\int_0^t f^{(m)}(\tau)K_\beta(t-\tau)d\tau\} & \text{for } \; m-1 < \beta < m \; , \\ \frac{d^m}{dt^m}f(t) & \text{for } \; \beta = m \; , \end{cases} \tag{16}$$

with

$$K_\beta(t-\tau) = \frac{(t-\tau)^{\beta+1-m}}{\Gamma(m-\beta)} \; ,$$

which is called the memory function [9]. To find out the effect of the memory on many physical , biological, etc. processes, see [10]. See also [11] for more details of Caputo time-fractional operator and its relationship

with Riemann-Liouville Integral operators.Â Then the time-fractional genetic drift of the random sexual mating reads

$$\underset{t\ *}{D}^{\beta} u(x,t) = \frac{1}{4N_e(t)}\frac{\partial^2}{\partial x^2}\{x(1-x)u\} - s\frac{\partial}{\partial x}\{x(1-x)u\}\ , 0<\beta<1\,, 0<x<1\ . \tag{17}$$

The asexual reproduction that occurs in a colony of bacteria that mathematically modelled in equation (2) does not required to be extended to the time-fractional version. The reason comes from the fact that the offspring in this case has only one parent and they inherit all their characteristics from him. The most important factors as described in equation(2) are the mutators, especially the wild-mutator and the sharp change on the environment that may cause bad or good effects on the number of the offspring.

4.1. **Discretization of the Time–Fractional Genetic Drift Equation of a Sexual Population.** The Caputo-time fractional operator [9] specified in the equation (16), is discretized by using the Grúnwald-Letnikov scheme and has been effectively implemented by many researchers to describe many bilogical, physical, chemical and etc.., see for examples [11], [17], [20] and [21]. The discretization of the Caputo time–fractional operator $\underset{t\ *}{D}^{\beta} u(x,t)$ by the Grünwald–Letnikov scheme reads

$$\underset{\tau\ *}{D}^{\beta} y_j(t_{n+1}) = \tau^{-\beta}\sum_{k=0}^{n+1}(-1)^k\binom{\beta}{k}(y_j^{(n+1-k)} - y_j^{(0)}),\ \ 0<\beta<1\ \forall n\in N_0\ . \tag{18}$$

Substitute from Grünwald–Letnikov scheme (18) in equation (17) and use the common finite difference rules, to get

$$\begin{aligned} y_j^{(n+1)} &= b_n y_j^0 + \sum_{m=2}^{n} c_m y_j^{n+1-m} + \\ &(\beta - \frac{2\mu jh(1-jh)}{4N_e})y_j^n + \frac{\mu s}{2R})h(j-1)(1-(j-1)h)\}y_{j-1}^{(n)} + \\ &\{(\frac{\mu}{4N_e} - \frac{\mu s}{2R})h(j+1)(1-(j+1)h)\}y_{j+1}^{(n)} + O(\tau^{\beta}, h^2)\ , \end{aligned} \tag{19}$$

where R and μ are defined in the previous section. we need to recall the coefficients $b_n = \sum m = 0^n(-1)^m\binom{\beta}{m}$, and $c_m = (-1)^{m+1}\binom{\beta}{m}$, introduced by Gorenflo et.Â al [20], to simplify the above equation,Â in order to fulfil the relationÂ below

$$b_n + \sum_{m=1}^{n} c_m = 1\ .$$

The condition required to maintain the equation's non-negativity (19) is provided by the scaling relation.

$$0<\mu\leq 800\beta\ .$$

By using the same previously used vector $z^{(n)}$, equation (19) can be written in the matrix form

$$z^{(n+1)} = b_n z^{(0)} + \sum_{m=2}^{n} c_m z^{(n+1-m)} + z^{(n)}.Q\ , \tag{20}$$

where, the matrix $Q = q_{ij}$, $i,j\in(1,R-1)$ has the form

$$Q_{ij} = \begin{cases} Q_{ij}^{(1)} = \mu h(j-1)(1-h(j-1))(\frac{(1}{4N_e} + \frac{s}{2R}), & i=j-1,\ j=1,\cdots,R-1 \\ Q_{ij}^{(2)} = \beta - 2\mu\frac{jh(1-jh)}{4N_e} & i=j,\ j=1,\cdots,R-1 \\ Q_{ij}^{(3)} = (j+1)h(1-(j+1)h)(\frac{1}{4N_e} - \frac{s}{2R}),, & i=j+1,\ j=1,\cdots,R-1\ . \end{cases} \tag{21}$$

The matrix Q is not a stochastic matrix as the sum over all its rows is β and $0<\beta<1$. The matrix Q with the condition imposed on μ is still positive dominant matrix and that ensures the stability of the discrete approximate solution. For more details about the approximate solution of the time–fractional genetic drift equation of A sexual population using FDM, see [15].

In the section of numerical results, we give a comparison of the approximate solution of the Markov case ($\beta=1$) and the Non-Markov case ($0<\beta<1$) for different numbers of generations and different values of β.

5. The Convergence of the Stationary Discrete Approximate Solutions of the Diffusion Equations

The continuous stationary solution of the partial differential equation is getting by taking the limit as $t\to\infty$. In other words put $\frac{\partial^{\beta}u(x,t)}{\partial t^{\beta}} = 0\ , 0<\beta\leq 1$, and omitting all the terms depending on t. Applying this method here, i.e. on the equations (1, 2 and 17, leads us to hypergeometric functions.

Till now researchers recommend the more general methodÂ used to calculate the discrete stationary solutions of the mentioned equations. Any stochastic matrixÂ P of a Markov process, is known to fulfill the identity that by takingÂ the limit of P^nÂ as $n \to \infty$ is Π, see Ross[22]. The column vector Π also is a vector of probability, so it follows that $\sum_{i=0}^{n} \Pi_i = 1$ andÂ is called the vector of the transitionÂ probability. Stochastic matrices are the matrices of the classical genetic diffusion with drift of sexual and asexual population, givenÂ inÂ equations (6) and (13) respectively. The matrix Q of the time fractional genetic drift, defined in (21) are not stochastic matrix. But Abdel-Rehim [17] proved that the matrices of the time-fractional diffusion still have stationary probability vector satisfies $\sum_{j=1}^{R-1} \Pi_j = \beta$ and $0 < \beta < 1$.

This approximate stationary convergent solution is found by ignoring time dependence t on the discrete schemes. To do so, at the classical discrete schemes (3) and (10),Â replace all $y_{j\pm k}^{(n+1)}$ by $y_{j\pm k}^{(n)}$, $k = 0, 1, 2$. For the time-fractional scheme (19), one has to replace all $y^{(0)}$, $y^{(1)}$, $\cdots$, $y^{(n+1-m)}$, and $y^{(n+1)}$ by simply $y^{(n)}$. Then the discrete scheme of the genetic equations (3) and (19) for $n \to \infty$, with drift, converge to $z.H = 0$, which is equivalent $H^T.y = 0$, where the matrix H is defined in equation (9). Apply the same procedure with the classical discrete scheme of the genetic drift equation of asexual population (10) to get the simple matrix equation $H^T.y$, where H is defined at equation (14). That means the classical and non-classical cases tend to the same stationary discrete solutions.

The H^T matrices, specified separately at the respective equations (9,10), each have an eigenvector y^* of eigenvalue zero.Â Constitute the $\bar{y} = cy^*$ vector with $c = 1/\sum_{j=1}^{R-1} y_j^*$ as a vector whose sum of its elements is 1, exactly like the Π vector.

$$d(t_n) = \sum_{j=1}^{R-1} |y_j(t_n) - \bar{y}_j|, \ n = 1, 2, \cdots \ . \tag{22}$$

The simulation in the classical case, for the Asexual genetic diffusion with drift, shows that the d row vector approximates an exponential function

$$d(t) \approx e^{-wt} \ ,$$

where w is constant variable, and is called the convergence rate .

5.1. **The Reversibility of the Diffusion Processes.** Let $\{X_n, n \in \mathbb{N}\}$ be a two-sided extension of a positive recurrent Markov is said to be reversible if $X(t_1), X(t_2), \cdots, X(t_n)$ has the same distribution as $X(\tau - t_1), X(\tau - t_2), \cdots, X(\tau - t_n)$. That means satisfying the Markov chain with the P transition matrix and the Π stationary distribution, see Kelly [23],

$$\begin{aligned} P_{ij}^{(n)} &= P\{X_1^{(n)} = j | X_0^{(n)} = i\} = P\{X_0^* = j | X_1^* = i\} \\ &= P\{X_1^* = i | X_0^* = j\} P(X_0^* = j)/P(X_1^* = i) \\ &= \frac{\Pi_j}{\Pi_i} P_{ji}^{(n)} \ . \end{aligned} \tag{23}$$

Multiply both sides of the equation (23) by the factor Π_i, we have

$$\Pi_i P_{ij}^{(n)} = \Pi_j P_{ji}^{(n)} \ , \tag{24}$$

where equationÂ (24)Â is called the time–reversibility equation, see [17], [22], [23], and [24]. The numerical result shows that the time–reversibility equation satisfies the following condition for Markov case, i.e. forÂ the classical case

$$\sum_{j=1}^{R-1} \bar{y_j}.P = \sum_{j=1}^{R-1} P^T.\bar{y_j} = 1 \ , \tag{25}$$

where $\bar{y_j}$ is the numerically obtained stationary probability row vector, see section (5) and for the time-fractional genetic diffusion with drift of sexual population, the condition of reversibility is

$$\sum_{j=1}^{R-1} \bar{y}.Q = \sum_{j=1}^{R-1} Q^T.\bar{y} = \beta \ , 0 < \beta < 1 \ . \tag{26}$$

The matrices Q are defined at equations (21). That means the Non-Markov process is also reversible but with β percent and not 100% as the Markov process. MATHEMATICA software is used to prove these conditions. Once more, the expected time between any two returns can simply be calculated from the equation

$$m_j = \frac{1}{\Pi_j} \ 1 < j < R - 1 \ . \tag{27}$$

In the following section, we give the numerical results for all the studied quantities.

6. Numerical Results

In this numerical result, the boundary conditions for the *genetic drift* models in sexual and asexual population (1) and (2) will be fixed, i.e. $u(0,p,t) = u(1,p,t) = 0\ \forall t \leq T$, where T is the generations number. The solutions are singular at the points $x = 0$ and $x = 1$ and need to be handled separately, see [25]. The initial condition is $u(x,p,0) = \delta(x-p)$, where p is the gene frequency and $0 < x < 1$. The initial condition for $p = 1/2$ is represented by the column vector $y^{(0)} = \{0,\cdots,1,\cdots,0\}$. Let $x = jh$ with $1 < j < R$, $h = 1/R$ and let $t = n\tau,\ 0 < t \leq T$, $\tau = \mu h^2$ and the steps number for one generation $n = 1/\tau$.

These given numerical results correspond to $N_e = 100$, $R = 100$, $s = 0.02$, $\mu = 250$, $\tau = 2.5 \times 10^{-2}$, $T = 50$ for the final number of generations, i.e. for long memory, and $n = 40$ for the number of steps for a single generation. The simulation of the approximate solutions are given for $\beta = 0.75, 0.6$, with number of steps for one generation $n = 137$ for $\beta = 0.75$, $n = 468$ for $\beta = 0.6$.

The 1st group of figures [1 , 2] describes the time evolution of the classical and the non-classical approximate solution of the *genetic drift* (1),(17) equations, respectively, for different β and different generation values up to the generation of 50th.

The two plots of Figure [3] represent the comparison of the *genetic drift* equations (1) and (17) for short and long memory for different values of β and different generation number.

The 3rd group of figures [4] describes the time evolution of the classical solution of the Asexual *genetic drift* (2) equation for different generation values up to the generation of 50th. These numerical results correspond to $\mu_- = 0$, $\mu_+ = 0.01$, $N = 100$, $R = 100$, $s = 0.02$, $\alpha = 0.5$, $\mu = 50$, $\tau = 5 \times 10^{-3}$, $T = 50$ for the final number of generations, i.e. for long memory, and $n = 200$ for the number of steps for a single generation.

The 4th group of figures [5] describes the effect of the wild-type mutation on the genetic diffusion of Asexual population (2) as $\mu_+ = 0.1$ and $\mu_- = 0.01$ for different generation values up to the 50th generation.

It was found that, figures [4 , 5] are similar as $\mu_- = 0$ and $\mu_+ = 0.01, 0.1$, respectively.

The group of figures [6] describes the comparison between the time evolution of the classical approximate solution of the *genetic drift* (1) equation with $\mu_+ = 0.01$ and $\mu_- = 0, 0.001$, and the same numerical results mentioned in the second group.

The 6th group of figures [7] also describes the comparison between the time evolution of the classical approximate solution of the *genetic drift* (1) equation with $\mu_+ = 0.1$ and $\mu_- = 0, 0.01$.

Figures [8] represent the total summation of the approximate solution of the models (1) and (17) for different values of β and the model (2) for $\mu_- = 0$ and $\mu_+ = 0.01, 0.1$, respectively.

The 8th [9] group of figures show the discrete stationary solution of classical *genetic drift* equations (1) and (2) respectively, in sexual and asexual population. Taking into account that the discrete stationary solution of classical *genetic drift* equation of the Asexual population (2) is within $\mu_- = 0$ and $\mu_+ = 0.01, 0.1$, respectively.

The last group of figures [12] represent the convergence of the discrete scheme of the models (1), (17) for sexual population and the model (2) for Asexual colony of bacteria within $\mu_- = 0$ and $\mu_+ = 0.01, 0.1$, respectively.

Notice that, figures [8 – 12], left, correspond to the values $\mu_+ = 0.01, \mu- = 0.001$ and $\mu_+ = 0.1, \mu- = 0.01$, respectively. This proves that the values of the mutators are the most effect factors on the hereditary process on the asexual reproduction.

Figure [8], right, represents the comparison between the total summation of the discrete solution of the Markov genetic drift as $\beta = 1$ and the Non-Markov genetic drift of the sexual mating as $\beta = 0.75$ and $\beta = 0.6$. The figure shows that there is a loose of the part of the genes while with the dependence on the memory all the numbers of allele are transformed from the zero generation to the 50^{th} generation. This numerical result proves our main point of view.

Figure [10] represents the 3D simulation of time evolution of the classical sexual genetic diffusion equation with the initial gene frequency $p = 0.1$. Figure [11] represents 3D simulation of the time evolution of the sexual time fractional genetic diffusion equation for the time–fractional $\beta = 0.75$ and with the initial gene frequency $p = 0.5$.

Finally, the discrete stationary solution of the classical sexual population (1) $\bar{y} \approx x^2$ while in the asexual colony of bacteria (2) $\bar{y} \approx x$ are shown in the numerical results section are shown in [9] .

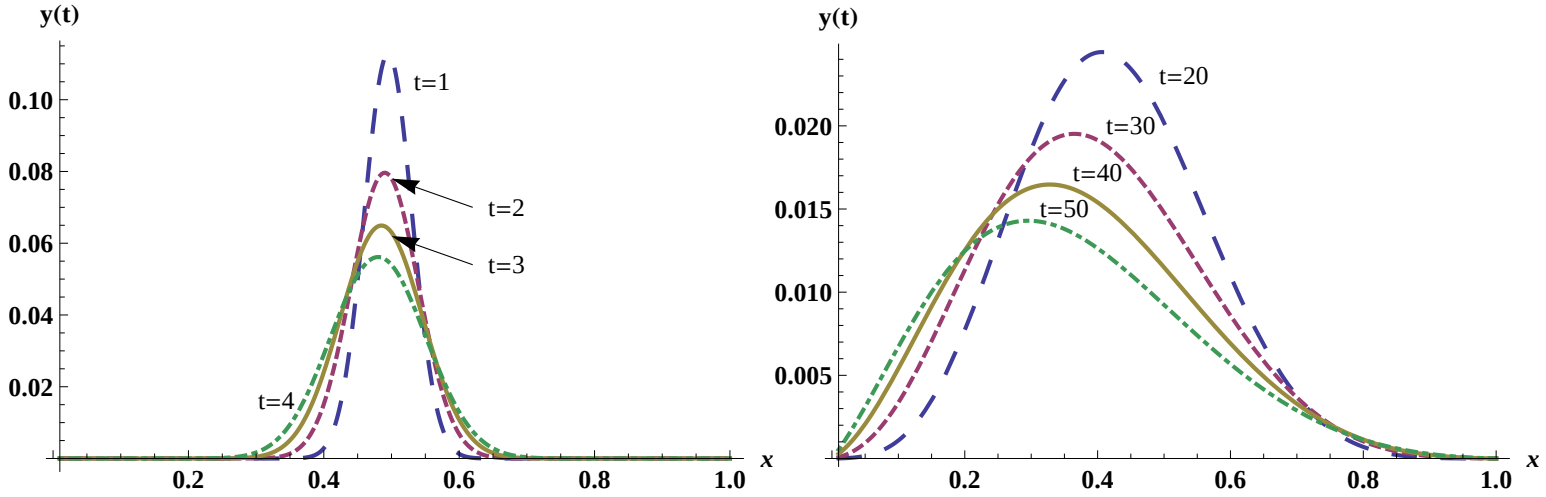

FIGURE 1. The classical approximate solution of the *genetic drift* equation (1) during different generations.

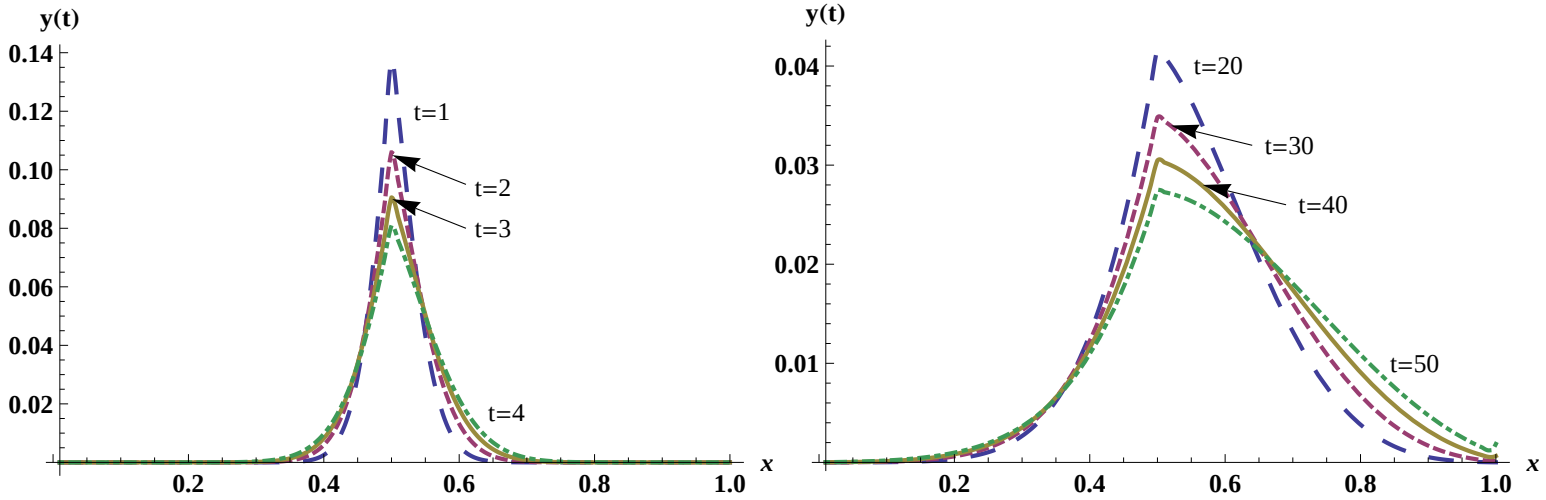

FIGURE 2. The approximate solution of the time–fractional *genetic drift* equation (17) till the 50^{th} generation for $\beta = 0.75$.

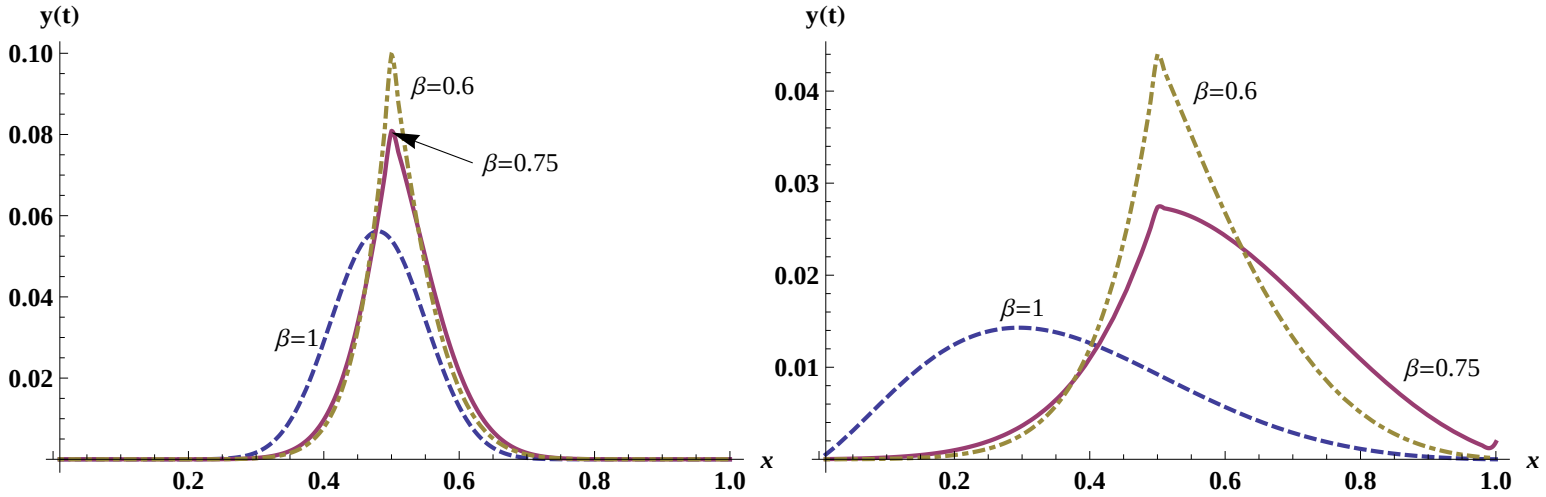

FIGURE 3. Comparison between the classical and the non–classical *genetic drift* equations (1),(17) for $t = 4, 50$, respectively with $y^{(0)} = \{0, \cdots, 1, \cdots, 0\}$.

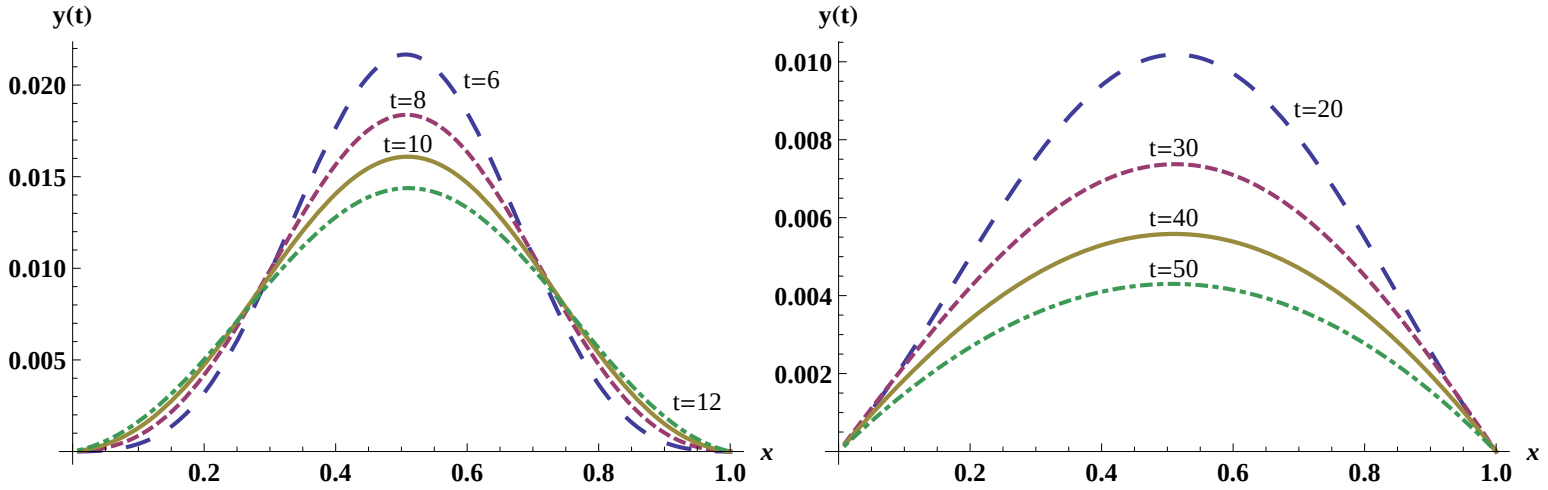

FIGURE 4. The approximate solution of the classical *genetic drift* equation (1) till the 50^{th} generation within $\mu_- = 0.001$.

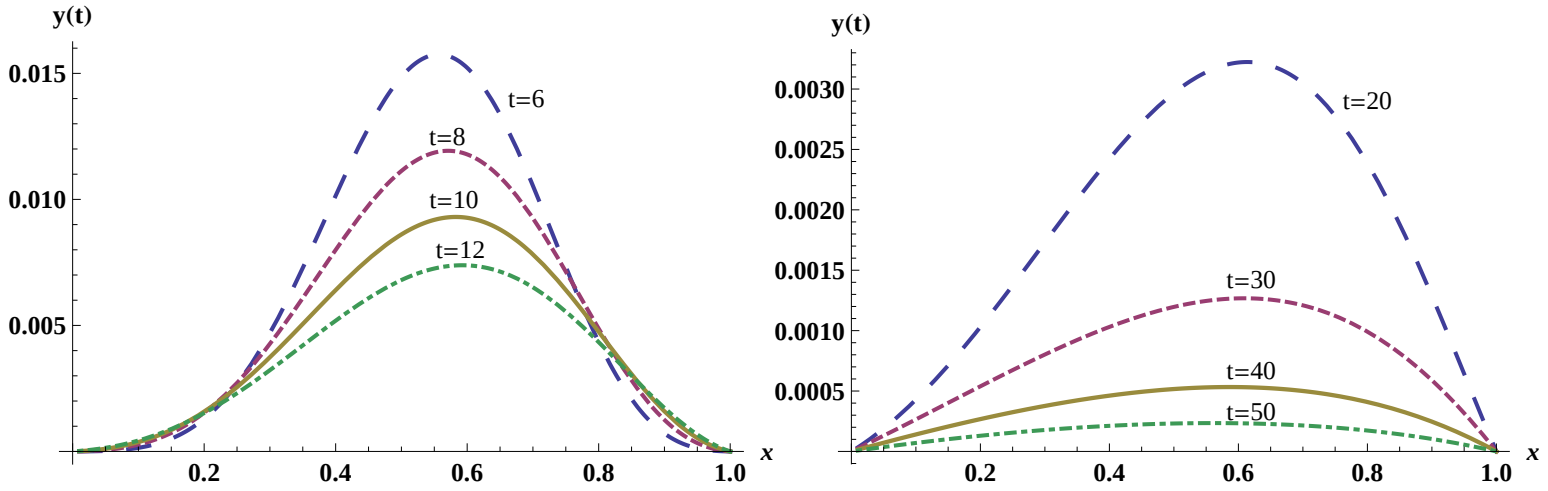

FIGURE 5. The approximate solution of the classical *genetic drift* equation (1) of asexual population for different values of generations with $\mu_- = 0.01$.

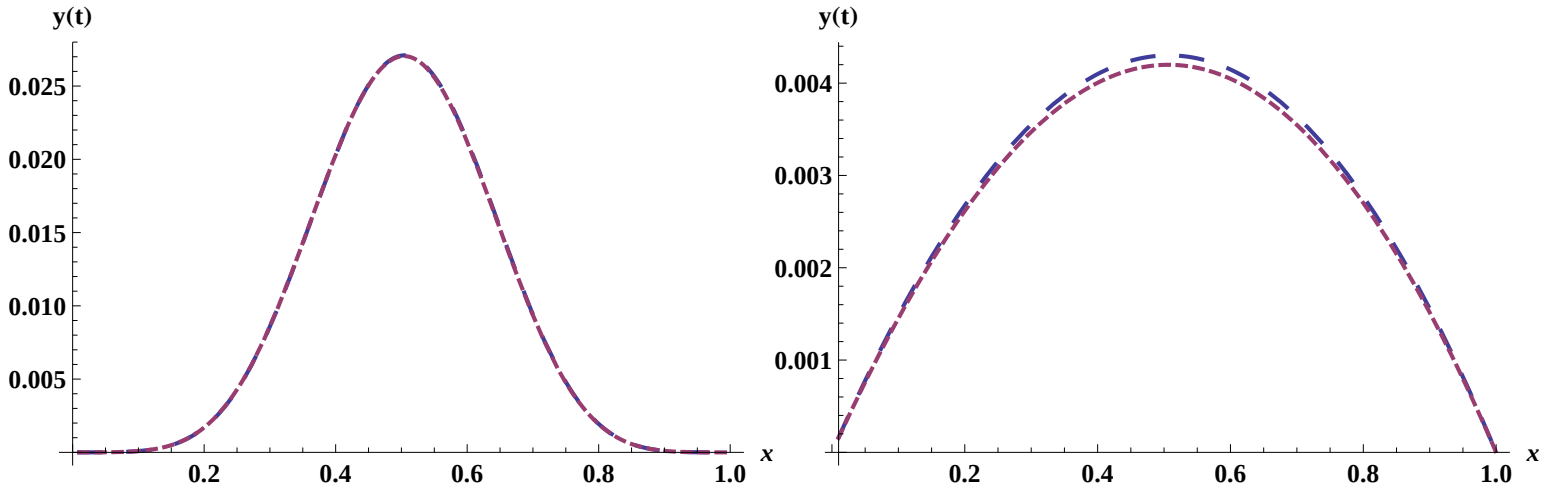

FIGURE 6. The comparison between $\mu_- = 0$ and $\mu_- = 0.001$ of the classical approximate solution of the asexual *genetic drift* (2) equation at $t = 4, 50$, respectively.

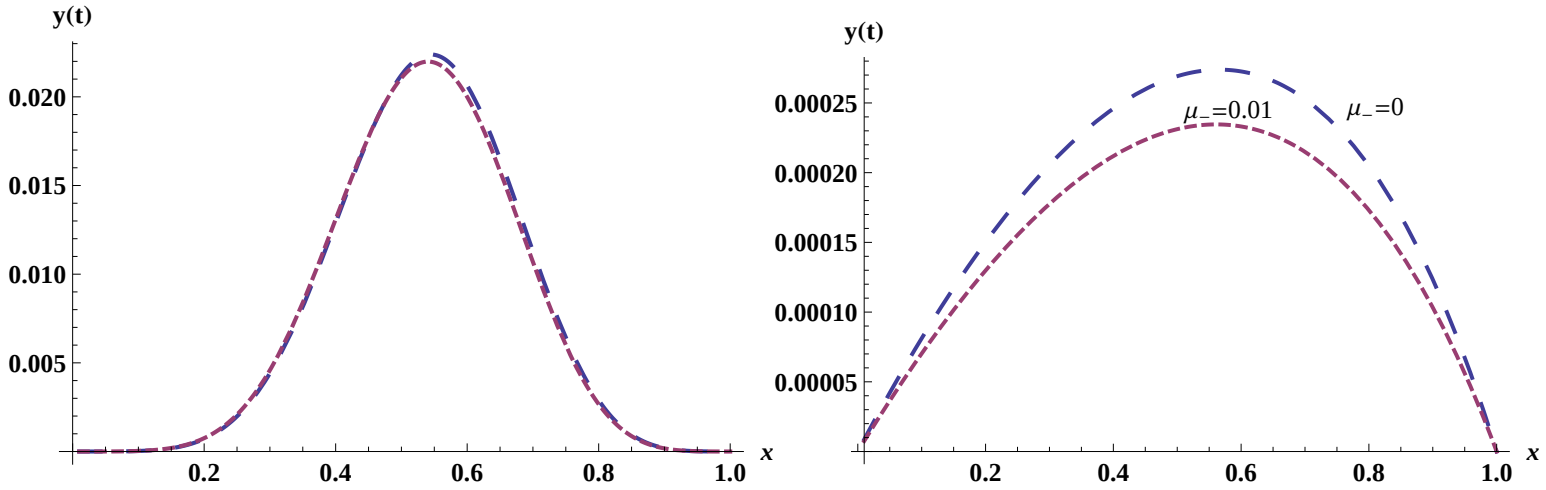

FIGURE 7. The comparison between $\mu_- = 0$ and $\mu_- = 0.01$ for the classical approximate solution of the asexual *genetic drift* (2) equation at $t = 4, 50$, respectively.

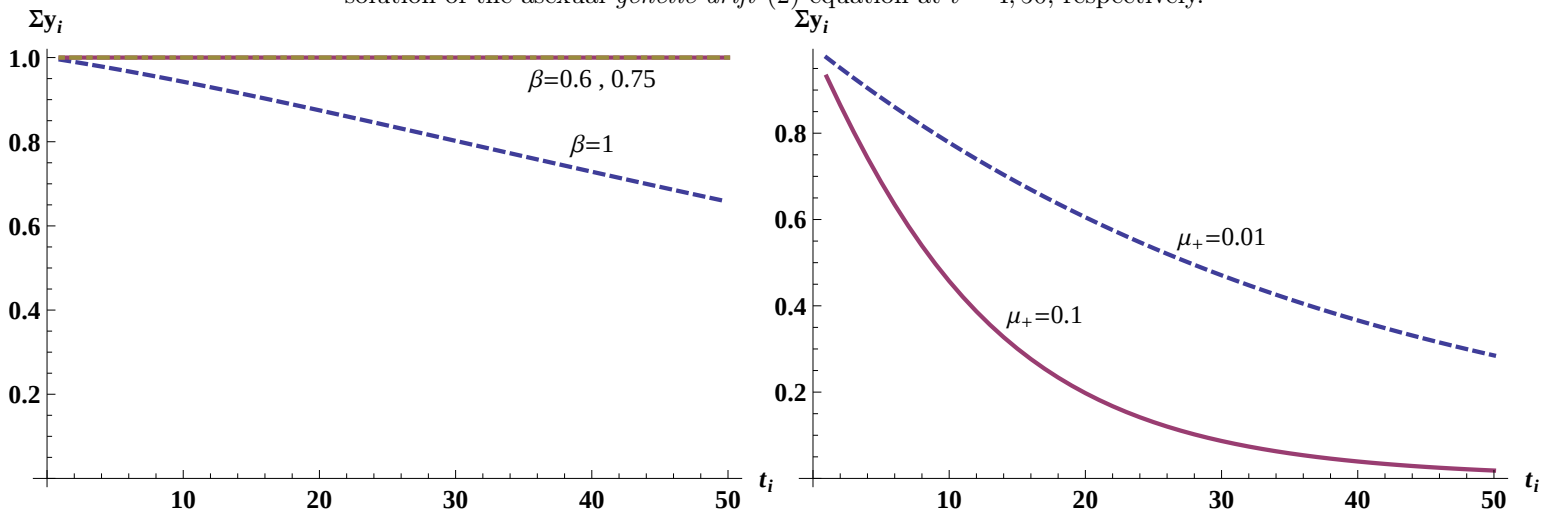

FIGURE 8. The total summation of the approximate solution between the classical and the time-fractional sexual *genetic drift* equations (1),(17) and the classical asexual *genetic drift* equation (2), respectively.

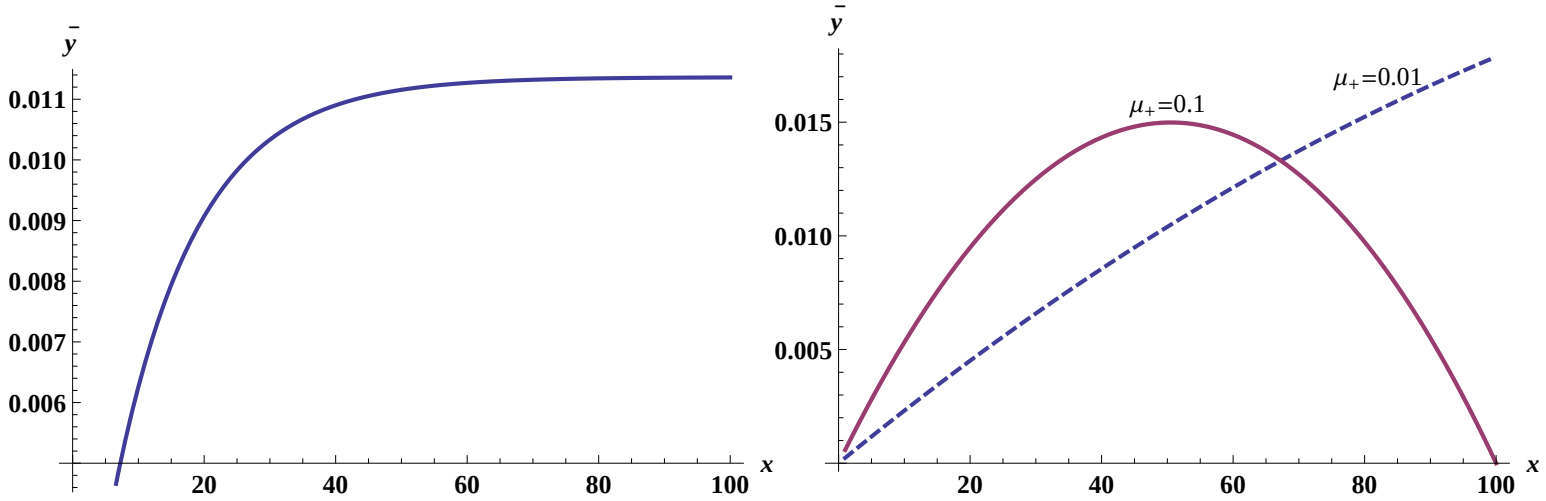

FIGURE 9. The discrete stationary solution of classical *genetic drift* equations (1) and (2) of the sexual and Asexual population, respectively.

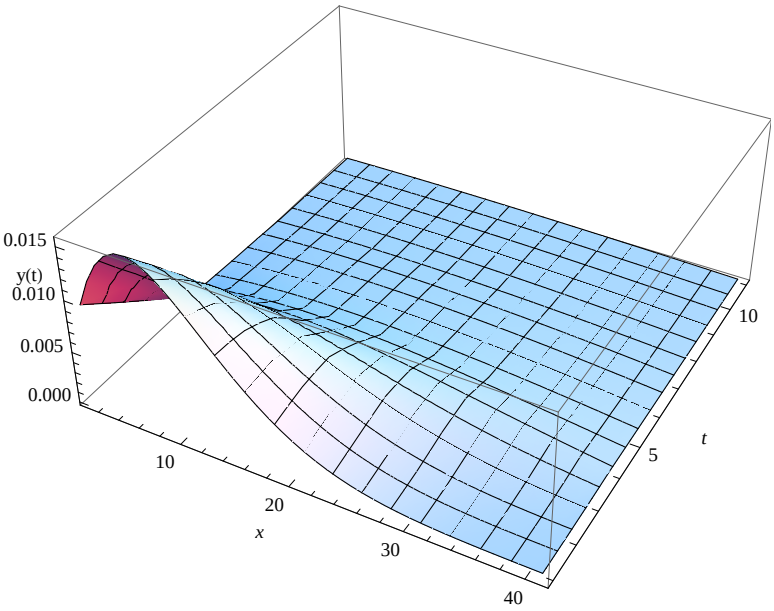

FIGURE 10. The 3rd simulation of the classical sexual Till the 10^{th} generation in genetic drift equation for the initial gene frequency $p = 0.1$.

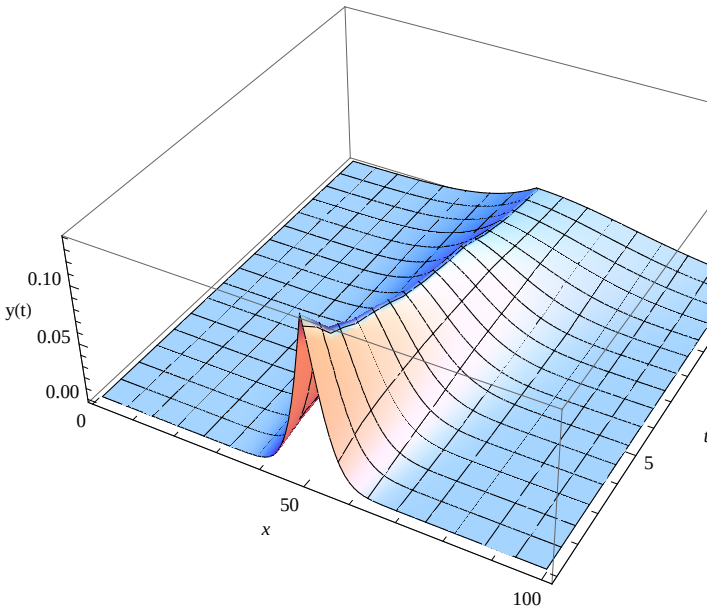

FIGURE 11. The 3rd simulation Till the 50^{th} generation of the sexual time fractional genetic diffusion equation with the gene frequency $p = 0.5$.

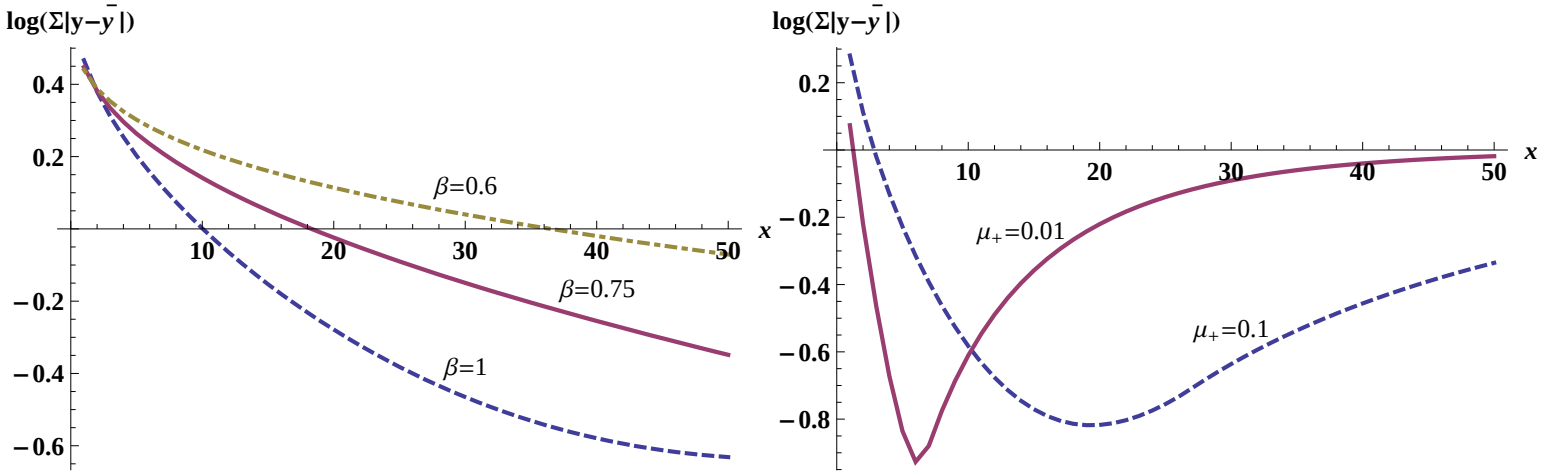

FIGURE 12. The convergence of the discrete scheme of the classical and the non–classical *genetic drift* equation (1),(17) and the classical discrete scheme of the model (2.

7. Conclusion

During sexual mating, two haploid gametes join in the process of fertilization to produce a diploid zygote and is called the Sexual reproduction. In this case, the children resemble their parents, but they are never identical to them and through generations the total number of genes will loose its unity. Therefore the extension to the Non-Markov case (the time-fractional diffusion process) must be interfere to preserve this property, i.e. the total summation of the conditional probability of finding the allele with certain frequency at a certain generation that must be one. In the Asexual reproduction, the offspring are genetically identical to each other and to their parent which causes no loose of genes property that is why there is no need to use the Non-Markov case. The numerical results shows the memory has a great effect on the closed countries i.e. countries that are interested in Endogamy such as Middle east, North Africa and Regions of South Asia. Tribes and families of Upper Egypt also interested in internal marriage to maintain relationships between tribes and the ranks of families like Arab, Al Hawara and Ashraf tribes.This phenomenon goes back thousands of years, as the Pharaohs were famous for allowing brother-sister marriage, and the most prominent of these cases is Pharaoh Tutankhamun, who was born as a result of incestuous marriage. Scientists took samples of his DNA, it was found that his parents were brother and sister. And this continued until the reign of the Ptolemies, the successors of Alexander the Great, when Cleopatra II married her brother, King Ptolemy VI, and when he passed away, Cleopatra married her second brother Ptolemy VIII, in order to preserve the purity of royal blood and limit the authority to their dynasty only. So did the Inca Empire, restricting marriage between brother and sister. While in the open countries such as USA, Germany, Britain, Russia and North Korea who prohibit that phenomenon and in Asexual population (colony of bacteria), the factors that cause genetic drift are selection and mutation.

Due to the effect of the memory on the diffusion processes, Some negative characteristics and diseases are raised on the closed countries and closed big families.

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