Population dynamics on a rugged landscape is studied analytically and numerically within a simple discrete model for evolution of \( N \) individuals in one-dimensional fitness space. We reduce the set of master equations to a single Fokker-Plank equation which allows us to describe the dynamics of the population in terms of thermo-activated Langevin diffusion of a single particle in a specific random potential. We found that the randomness in the mutation rate leads to pinning of the population and on average to a logarithmic slowdown of the evolution, resembling aging phenomenon in spin glass systems. In contrast, the randomness in the replication rate turns out to be irrelevant for evolution in the long-time limit as it is smoothed out by increasing “evolution temperature”. The analytic results are in a good agreement with numerical simulations.

Recently, a simple model was introduced to describe the evolution of a finite population of mutating species in a one-dimensional fitness space \([1,2]\). Every species is characterized by a fitness variable which controls its replication rate, and mutations move the species equiprobably up or down along this fitness axis. Analysis of this model leads to a two-staged dynamics of a population initially spread within some fitness range. At the first (fast) stage, the population forms a universal pulse-like distribution in the fitness space, which usually is accompanied by the fast growth of the mean fitness. At the second stage, the pulse propagates toward higher fitness due to mutations, new more fit mutants are constantly generated, and less fit die due to the constant population size constraint. The mean fitness grows linearly in time. The non-trivial scaling of the fitness growth with the mutation strength and the population size was found in Refs. \([1,2]\) and appears to be in a qualitative agreement with the evolution dynamics of RNA virus \([3]\).

Clearly, this model contains an implicit assumption that underlying fitness landscape in the configurational space is smooth. In reality, fitness landscapes likely exhibit substantial degree of ruggedness \([4]\), i.e. a probability to find a more (or less) fit mutant varies for different genomes. Moreover, for rugged landscapes there may be genomes which are more fit than any of their nearest neighbors in the sequence space which can be accessed as a result of a point mutation. Thus, there are local maxima of fitness. Rugged fitness landscapes are usually considered in multi-dimensional configurational spaces of individual species where the fitness is a complicated function of the detailed structure of the genome. A popular \( NK \)-model for the relation between genomic structure and fitness was introduced by Kauffman \([4]\). Many other more complicated or realistic models of rugged landscapes are described in the literature (see, e.g., \([4,5,6]\)).

Our model of one-dimensional fitness space is essentially different from these models since individual species with different internal structure but identical fitness are considered indistinguishable and therefore assigned to the same location along the fitness axis \([4]\). This model obviously cannot account for the evolution of the internal structure in the population, in particular, the famous error catastrophe which occurs at high mutation rate and leads to accumulation of “bad mutants” and perpetual loss of heritable genetic information \([8]\). Nevertheless our model of evolution in a one-dimensional fitness space is capable of reproducing important dynamic features of evolution found in more realistic models. Due to its relative simplicity, it is more amenable to analytical treatment then multi-dimensional configurational space models.

The goal of this paper is to study the dynamics of the population evolution in the framework of the model \([1,2]\) which however takes into account ruggedness of the underlying fitness landscape. Random fluctuations which are considered to be functions of one fitness variable characterizing the genotype as a whole, modify locally both replication rate as well as mutation probability. We assume that the replication rate is a sum of linear function of fitness and random fluctuations. Linear part provides the selective pressure driving the population towards higher fitness, as in smooth landscape case. Random fluctuations however provide trapping of the population near local maxima of replication rate and on average slow down the mean fitness growth. Probability of mutation up and down in fitness at a given state also has fluctuating part. We assume that these fluctuations are statistically independent of fluctuations of the replication rate. Based on master equations for underlying Markov process, we derive the Fokker-Plank equation for probability distribution of species in the population. We find that initial growth of fitness is dominated by the fluc-
tations of the replication rate, but at long-time limit these fluctuations become irrelevant. The long-time limit is completely determined by the fluctuations of the mutation rate which lead to logarithmic slowdown of the fitness growth. This phenomenon is analogous to the thermally-activated creep of a particle in quenched random (pinning) potential.

Let us first specify the model (cf. Ref. [3]). Consider a population of \( N \) individuals which can replicate according to their replication rate \( R \), and babies can mutate, thus changing their fitness relative to parents. We assume asexual replication, i.e. any individual has a chance to reproduce independently. Once a baby is born, some member of the whole population (including the new baby) which is picked at random, is eliminated to preserve the constant population size. We assume that the replication rate \( R \) is a function of only one independent variable \( x \) (fitness), which has a linearly growing component and a random component, \( R(x) = x + \xi_r(x) \) (without loss of generality we chose a unit slope). When mutation occurs, a mutant baby changes its value to \( x \pm 1 \) with probabilities \( M^\pm(x) = (1 \pm \xi_m(x))\mu/2 \), respectively. Random functions \( \xi_r \) and \( \xi_m \) are assumed to be uniformly distributed between \( \pm \Delta_{\xi,m} \) and they are “frozen”, or quenched, for a given realization.

An exact description of this Markov process involves a very large (strictly speaking, infinite) set of master equations for the probabilities of all possible population configurations. Significant simplification can be achieved in the limit of small mutation rate \( \mu \) when all population is highly localized near \( \langle x \rangle \) and only two neighboring sites \( x, x+1 \) are usually occupied simultaneously (the probability to occupy simultaneously 3 or more sites is smaller by a factor of \( \mu \)). The number of master equations then is reduced to \( N \) (see [3]). It turns out that a much simpler system of only 2 individuals exhibits similar non-trivial features as \( N \)-individual model. This system in the limit of small \( \mu \) can be described by only two equations for probability \( g \) to find a particle at site \( x \), and \( f \), the probability of finding one particle at site \( x \) and another at \( x+1 \),

\[
\begin{align*}
\partial_t f(x) &= -\mu \frac{4}{3} R(x) f(x) + \frac{1}{3} R(x) (g(x) + g(x-1)), \\
\partial_t g(x) &= \frac{4}{3} (R(x) M^+(x) f(x) + R(x+1) \times M^-(x+1) f(x+1) - \frac{1}{3} (R(x) + R(x+1)) g(x).
\end{align*}
\]

(2)

The first term in r.h.s. of (1) reflects the probability of mutation events leading to transition from the collapsed state when both particles occupy site \( x \) to either of distributed states \( (x, x+1) \) or \( (x-1, x) \). The second term describes a reverse process of replication at site \( x \) which is followed by elimination of a particle at \( x \pm 1 \) and collapse of the population to the site \( x \). Terms in the right-hand side of Eq.(2) have the same origin. For \( \mu \ll 1 \), \( \partial_t g(x) \) is small in the asymptotic limit, and \( g \) is enslaved to \( f \),

\[
g(x) = \frac{4}{3} \frac{R(x) M^+(x) f(x) + R(x+1) M^-(x+1) f(x+1)}{R(x) + R(x+1)}.
\]

Substituting \( g(x) \) into Eq.(3), taking a continuous \( x \) limit, and keeping only linear in \( \xi \) terms, we obtain a single equation for \( f \),

\[
\begin{align*}
\partial_t f(x) &= -\mu \frac{2}{3} \partial_x [(1 + 2 x \xi_r + \partial_x \xi_r) f(x)] + \left( \frac{\mu}{3} \right) \partial_x^2 [(x + \xi_r + \frac{1}{2} \xi_m) f(x)].
\end{align*}
\]

(3)

In a similar fashion but with more cumbersome algebra, an equation describing the probability distribution \( f(x,t) \) for a population of \( N \) individuals to be localized at point \( x \) at time \( t \) can be derived in a small \( \mu \) limit. In the limit of \( N \gg 1 \) it reads

\[
\partial_t f(x) = -\mu \frac{2}{3} \partial_x [(N + 2 x \xi_r + N \partial_x \xi_r) f(x)] + \left( \frac{\mu}{3} \right) \partial_x^2 [(x + \xi_r + \frac{N}{2} \xi_m) f(x)].
\]

(4)

For \( \Delta_{\xi,m} = 0 \) this equation coincides with one derived in Ref. [2] and describes the increase of average fitness of \( N \) individuals as a result of random mutations and selective pressure. In the limit of smooth landscape the average fitness \( \langle x \rangle \equiv \int f(x) dx \) grows linearly in time with the rate \( V_0 = \mu N \). A general solution to Eq.(3) in the presence of random fluctuations is not available, however one can estimate \( \langle x \rangle \) using general methods of stochastic kinetics in random media [6]. To this end, note that Eq.(3) has a form of Fokker-Plank equation for the dynamics of a single particle in the fitness space under the global bias \( N \mu/2 \) in the presence of the random quenched potential. Note that in our case the temperature itself depends on \( x \). The corresponding dynamic process is governed by the Langevin equation:

\[
\frac{dx}{dt} = \frac{N}{2} \left( 1 + \partial_x \xi_r \right) + x \xi_r + \eta(x,t),
\]

(5)

where stochastic term \( \eta(x,t) \) has the following correlator \( \langle \eta(x,t) \eta(x,t') \rangle = 2 \tau \delta(t - t') \) with local “evolution temperature” \( \tau = \frac{1}{2} (x + \xi_r + N \xi_m/2) \), and we rescaled time \( \mu t \to t \). If the stochastic term were absent, the particle would have been pinned by local minima of the quenched potential. However, in the presence of “thermal fluctuations” \( \eta(x,t) \) the system will evolve via a sequence of thermally activated jumps from one minimum to a neighboring one favored by the global positive bias (selective pressure). This type of motion is well known in the dynamics of disordered media and is usually referred to as creep [6].

We discuss the cases \( \xi_m \neq 0, \xi_r = 0 \) and \( \xi_m = 0, \xi_r \neq 0 \) separately, since they exhibit different asymptotic behavior. Let us first focus on the case \( \xi_m \neq 0, \xi_r = 0 \). It generalizes a well-known Sinai diffusion problem for the particle subject to a random force field. The particle displacement obeys ultra-slow sub-diffusion law \( \langle x \rangle \sim (\log t)^2 \) for
zero average driving \([11]\), and the power-law dependence \(\langle x \rangle \sim t^\xi\) in the driven case \([12]\). Our situation is more complicated since the evolution temperature and the magnitude of disorder depend on \(x\) explicitly. To obtain the time-dependence of \(\langle x \rangle\) we generalize the approach of Ref. \([12]\). According to the general theory of stochastic growth \([10]\) the evolution rate \(V = \langle x \rangle \propto f(\bar{U}[x])\), where \(\bar{U}\) is the typical potential barrier controlling the evolution, and \(f\) is the corresponding quasi-equilibrium distribution function, given by (we consider the limit \(x \gg N\) and therefore drop the random term in the expression for evolution temperature):

\[
f(x) \sim \exp \left( 2 \int_0^x \xi_\mu(x') dx' + (N - 1) \log(x) \right) \quad (6)
\]

Taking into account that the typical height of the potential barrier corresponding to the displacement \(x\) is \(\sqrt{\langle (\xi_\mu(x') dx')^2 \rangle} = \Delta_\mu \sqrt{x/3}\) (by assumption \(\xi_\mu\) is uniformly distributed between \(\pm \Delta_\mu\)) one finds the following estimate for the typical time to evolve over the distance \(x\),

\[
\langle t \rangle \sim V^{-1} \sim 1/f(\bar{U}[x]) \\
\sim \exp(2\Delta_\mu \sqrt{x/3} - (N - 1) \log x). \quad (7)
\]

Inverting Eq. (7) we obtain the following evolution law

\[
\langle x \rangle \sim \frac{3}{4\Delta_\mu^2} \left( \log(t + C_0) + (N - 1) \log(x) \right)^2 \quad (8)
\]

where the constant \(C_0\) is determined by the initial condition for \(\langle x \rangle\) at \(t = 0\). In the limit \(\langle x \rangle \to \infty\), the final asymptotic behavior of Eq. (8) coincides with the Sinai diffusion law \([11]\) \(\langle x \rangle \sim \sqrt{t/\Delta_\mu^2} \log t\). For large \(N\) however this regime realizes at enormously large times \(t\) and distances \(\langle x \rangle\) \((\langle x \rangle \gg N^2\) or \(t \gg e^N\)), and can be hardly observed in numerical experiment. In the intermediate asymptotic regime \(1 \ll \langle x \rangle \ll N^2\), the last term in Eq. (3) is a leading one, and taking into account that \(\log(x)\) is a slow function as compared to \(\langle x \rangle\), we obtain the following evolution law:

\[
\langle x \rangle \sim \frac{N}{\Delta_\mu^2} \log(\mu t + C_0) + x_0. \quad (9)
\]

(we restored original time units). Constants \(C_0\) and \(x_0\) themselves depend on \(N\) and \(\Delta_\mu\), and a crossover to disorder-free \((\Delta_\mu \to 0)\) linear growth of the fitness is recovered by taking the limit \(\mu t \ll C_0\) and \(C_0 \to \infty\). Our numerical results for discrete model simulations confirm analytical formula (3). Fig. 1 illustrates smooth logarithmic dependence \(\langle x(t) \rangle\) which was obtained numerically for \(N = 50\), \(\mu = 0.02\), \(\Delta_\mu = 0.7\) averaged over 50 statistically-independent configurations of quenched disorder (double angular brackets indicate computing a mean fitness of the entire population and ensemble averaging over different realizations of \(\xi\)). As expected, for large \(t\) the rate of the evolution slows down which corresponds to the aging phenomenon known in glassy dynamics \([13]\). In the Fig. 1 we also show the dynamics of the mean fitness of the population \(\langle x \rangle\) for a single run. This dependence is characterized by long periods of relatively steady fitness level (stasis) interrupted by abrupt jumps of the fitness. During these long intervals of stasis the population is trapped in regions of low probability of escape towards larger \(x\) (large negative \(\xi_\mu\)). Such an inhomogeneous pace of evolutionary changes is known in biology as a punctuated equilibrium hypothesis (see [4]).

We performed numerical experiments with our model for various \(N, \mu,\) and \(\Delta_\mu\). Fitting the results to the logarithmic dependence \(\langle x \rangle = A \log(t + t_0) + x_0\) yields a good agreement with formula (3). Figure 2 shows the dependences of \(A\Delta_\mu^2\) on \(N\) for several sets of parameters \(N, \Delta_\mu\). All the graphs collapse close to a single straight line with slope 1 in log-log coordinates, as predicted by the theory.

Now we turn to the case \(\xi_\mu = 0\) but \(\xi_\tau \neq 0\) where particle dynamics is governed by the diffusion in the random potential \(\xi_\tau\) rather than the random force field \(\vec{z} \xi_\mu\). For a statistically uniform system \((x\)-independent temperature and magnitude of the quenched disorder) with the Gaussian statistics of the disorder \(\xi_\tau\), the evolution rate \(V\) to the leading order is given by the following expression (compare with the thermally activated hopping rate from [11,4])

\[
V \sim \exp (-D/\tau^2), \quad (10)
\]

where \(D\) is the variance of the random potential and \(\tau\) is the temperature. In the present context both local variance \(D\) and the “evolution temperature” \(\tau\) depend on \(x\), however we expect that this formula holds adiabatically if the typical waiting time \(V^{-1}\) is large as compared to the relaxation time \(V_0^{-1}\). The average evolution temperature \(\langle \tau \rangle = \mu x/2\), and variance is \(D = \langle V_0^2 \xi_\tau^2 \rangle = V_0^2 \Delta_\tau^2/3\).

The evolution rate is thus determined by the following asymptotic expression

\[
V \sim V_0 \exp \left( -\frac{4V_0^2 \Delta_\tau^2}{3\mu^2 x^2} \right) = V_0 \exp \left( -\frac{N^2 \Delta_\mu^2}{3 x^2} \right) \quad (11)
\]

(here we added the pre-exponential factor \(V_0\) in order to match the results with the disorder-free case). According to Eq. (11), the evolution rate \(V\) grows monotonously with \(x\) and finally approaches the disorder-free rate \(V_0\). Therefore, in a long-time limit the quenched potential \(\xi_\tau\) becomes irrelevant because the growing with \(x\) evolution temperature smears out the pinning potential and further promote the growth of \(V\). However, for \(x\) not too large \((x \ll N\Delta_\mu)\) one can distinguish two different regimes of the (initial) evolution. If \(\Delta_\tau \gg 1\), in the absence of the thermal fluctuations a particle would be pinned
by the random potential. Due to thermal fluctuations the particle will creep toward larger x, slowly increasing the velocity from V = 0. In contrast, if Δr ∼ O(1), i.e. above the depinning threshold, the particle starts to move with the finite velocity V < V0, and after some time achieves asymptotic velocity V0. Thus, the model exhibits “thermal depinning” and ignores completely fitness fluctuations for large average fitness values. Both regimes are seen in numerical simulations of the discrete model. Shown in Fig. 3 are ⟨⟨x(t)⟩⟩ for different values of Δr. We see that for large t all lines have the same slope which coincides with the evolution rate on a smooth landscape V0 = Nμ/2 (see also inset in Fig. 3). Intermediate regime exhibits a wide range of evolution rates which depend on Δr.

In conclusion, we have shown that two types of quenched randomness in the fitness space have a different effect on the population evolution. Quenched randomness of the replication rate slows down the evolution only at the initial stage since the increase of the "evolution temperature" eventually smooths out ruggedness and evolution proceeds at a rate corresponding to the smooth landscape case. However, in the long-time limit quenched disorder of the mutation rate being amplified by the increasing replication rate, dominate the evolution temperature growth. In the limit x, t → ∞ the evolution exhibits ultra-slow logarithmic growth of averaged mean fitness ⟨⟨x⟩⟩ . Individual runs are characterized by long intervals of almost constant mean fitness ⟨x⟩ interrupted by spontaneous changes, a dynamics which is usually interpreted as punctuated equilibrium. In this work, we have made simplest ad hoc assumptions regarding the statistical properties of the fitness landscape. An important task for a future work will be to establish connections between “standard” multi-dimensional sequence space evolution models such as NK-model and one-dimensional fitness space model described here.

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[1] L. Tsimring, D. Kessler, and H. Levine, Phys. Rev. Lett. 76, 4440 (1996)
[2] D. Kessler, H. Levine, D. Ridgway and L. Tsimring, J. Stat. Phys., (1997) to appear.
[3] I.S. Novella et al., Proc. Natl. Acad. Sci. U.S.A. 92, 5841 (1995).
[4] S. Kaufmann, The Origins of Order, Oxford, New York, 1993.
[5] W. Fontana et al. Phys. Rev. E 47, 2083 (1993).
[6] S. S. Plotkin, J. Wang, and P. G. Wolynes, Phys. Rev. E 53, 6271 (1996).
[7] This is similar to assigning the population of B-cells into different affinity classes suggested by T. B. Kepler and A. S. Perelson [J. Theor. Biol., 164, 37 (1993)].
[8] M. Eigen and P. Schuster. The Hypercycle: A Principle of Natural Self-Organization, Springer, New York, 1979.
[9] G. Blatter et al., Rev. Mod. Phys. 66 1147 (1994).
[10] L. D. Landau and E. M. Lifshitz, Physical Kinetics, Oxford, New York, Pergamon Press, 1981.
[11] Y. G. Sinai, Theor. Probab. Its Appl. 27, 247 (1982).
[12] V. M. Vinokur, J. Phys. (Paris) 47, 1425 (1986).
[13] M. Alba, M. Ocio, and J. Hammann, Europhys. Lett. 2, 42 (1986).
[14] P. Le Doussal and V. M. Vinokur, Physica C 254, 63 (1995).