Learning Mitigates Genetic Drift

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Abstract

Genetic drift is a basic evolutionary principle describing random changes in allelic frequencies with far-reaching consequences in various topics ranging from species conservation efforts to speciation. The conventional approach is to assume that genetic drift has the same effect on all populations undergoing the same change in size, regardless of the different behavior and history of the populations. However, here we reason that a simple act of learning from experience can significantly mitigate the effects of genetic drift. We further test this notion in an agent-based model monitoring allele frequencies in a population of prey, either able or not able to learn. Our results demonstrate that learning can mitigate drift regardless if the population is haploid or diploid or whether it reproduces sexually or asexually. These findings may be of great importance not only for a basic evolutionary theory but also for all fields using the concept of genetic drift.

Main text

Genetic drift is commonly thought to depend on allele frequencies, population size, number of reproducing individuals, and not much else[1,2]. Just as in other areas in biology[3] such a simplistic approach proved to be very useful, but as we show further in this article, it is likely not entirely correct. Just as the phenotype of an individual animal changes and develops during its life, so can change its response to various potentially deadly situations. As we will show further, if such change is systematic, and if it improves the chances of a given animal to survive such experience in the future, it can also affect the loss of genetic variability caused by drift in a given population. But, why should a simple act of learning from experience decrease random fluctuations in allele frequencies? To answer this question, let us imagine a hypothetical scenario. Let us suppose that there is a species subject to predation and that every young individual of this prey species has approximately the same chance to escape a predator if it is attacked, let us say 50%. However, if a given individual survives the attack of a predator, it learns from this experience and is more cautious in the future, increasing its chances to survive future encounters of a similar kind (e.g., to 75%). Furthermore, every future encounter that the prey individual survives allows it to learn from it and slightly increases its chance to survive the next one. In other words, individuals who survive and learn from such encounters gain an advantage in comparison to those who have not yet encountered predators and thus increase their fitness, even though this advantage has random origins and is not heritable. As a consequence, alleles carried by a given lucky individual get a temporary boost to their average fitness in the population regardless of their function. However, because such an effect is random, all alleles benefit at some point, which, in the long run, protects them against the effects of genetic drift.

In this article, we test the above-described hypothesis that learning from experience mitigates genetic drift. For this purpose, we develop an agent-based model that monitors allele frequencies in a population of prey, either able or not able to learn how to avoid/escape predators more effectively from the experience of unsuccessful past predator attacks (the code in R is freely accessible at GitHub).

Results

The haploid asexual model

First, we tested the hypothesis that learning mitigates genetic drift on a simple model assuming haploid, asexually reproducing prey. The results of these simulations show that ability to learn from experience significantly reduces the loss of genetic variability caused by genetic drift (Fig 1).

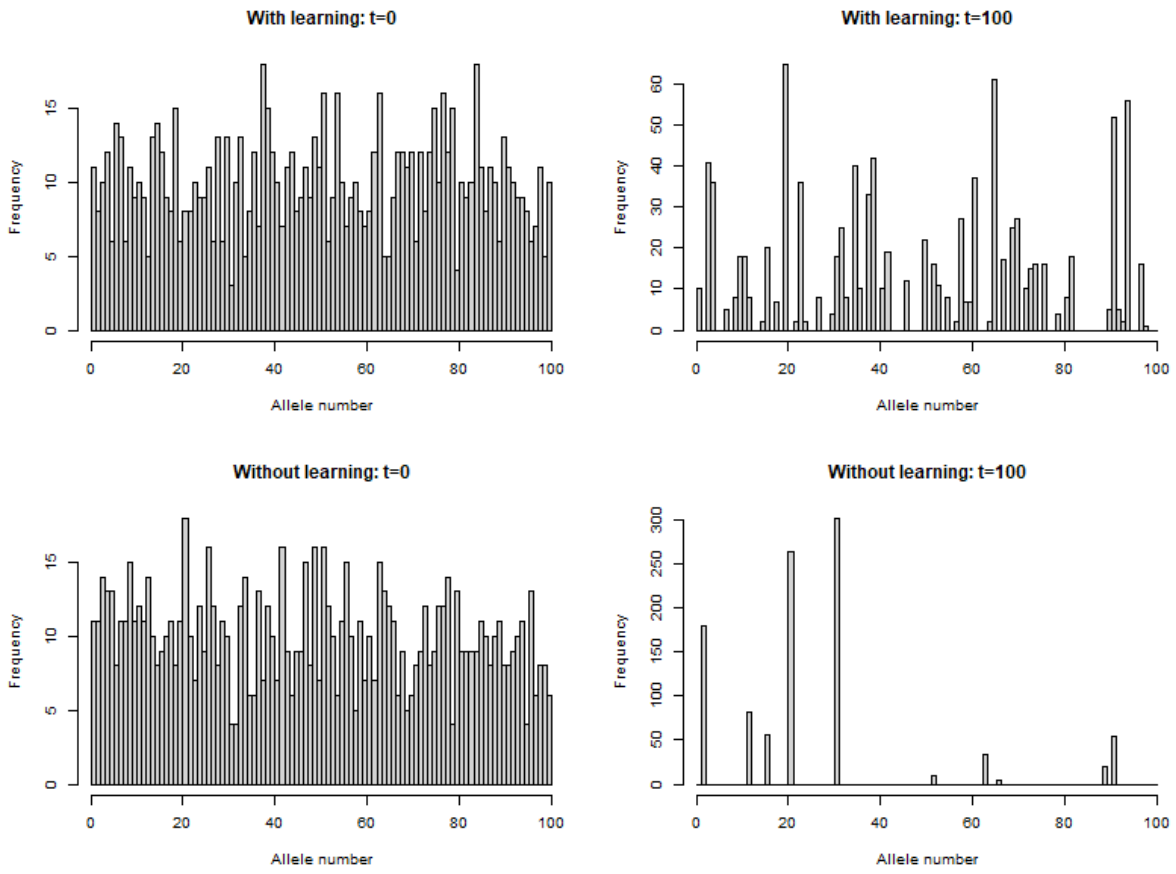

Figure 1: Learning slowdowns a loss of genetic variability caused by genetic drift. In this simulation run, prey populations consisted of 1000 individuals at the beginning of each step. Individuals killed by predators were replaced at the end of each step by a reproduction of surviving individuals (see Methods). Predators consumed 500 prey individuals per time-step in both learning and non-learning scenario. The upper bound on the probability to avoid/escape predator attack was set to 99%.

However, the size of this effect depends highly on specific circumstances. Our simulations consistently showed that the effect of learning on the genetic drift is affected by the fraction of the population killed by predators (Fig 2). Surprisingly, the relationship between the fraction of the prey population killed by predators and the effect of learning on the genetic drift is not always gradual (Fig 2 a and b). Instead, for a given simulation setting, there exists a narrow interval of values under which the learning makes only a meager difference and above which the learning greatly decreases loss of variability caused by genetic drift. Figure 2 illustrates this sudden jump by a stark contrast between simulations where 300 prey individuals were killed per time step (Fig 2 a) and simulations were 400 prey individuals killed per time step (Fig 2 b). The differences between simulations in which 500 (Fi 2 c), 600 (Fig 2 d), 700 (Fig 2 e), or 800 (Fig 2 f) prey individuals had been killed per time step were comparatively small. However, figure 2 f shows that when the intensity of predation becomes too-high, learning mitigates drift less effectively. The most likely interpretations seem to be that in such a setting, most alleles may be easily lost in one or a few time-steps, which provides less opportunity for the learning to affect the outcome.

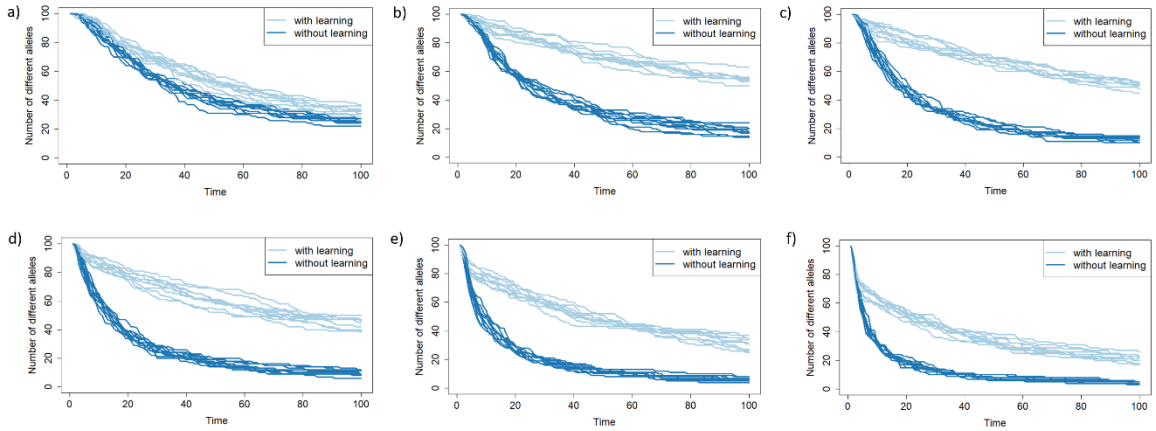

Figure 2: The intensity of predation modulates the interaction of learning and genetic drift. The figure shows the temporal dynamics of the number of alleles present in populations of prey subject to predation. All prey populations consist of 1000 individuals at the beginning of each step. Individuals killed by predators are replaced at the end of each step by a reproduction of surviving individuals (see Methods). In panels a-f, predators consume 300, 400, 500, 600, 700, 800 prey individuals per time-step, respectively. Predators kill the same number of individuals in both learning and non-learning conditions to allow for a fair comparison. The upper bound on the probability to avoid/escape predator attack is set to 99%. Each panel shows ten simulation runs for both learning and non-learning conditions.

Furthermore, the ability of the learning to conserve genetic variability highly depends on how effective learning is (Fig 3). In our simulations, the maximal limit on how effectively prey could learn to avoid or escape predators was an even more important factor than the intensity of predation itself. When the prey could learn at best to escape with 90% probability, the effect of learning was noticeable (Fig 3 a) but much smaller than when they could eventually reach 95% chance (Fig 3 b), 97,5% chance (Fig 3 c) or even 99% chance to escape (Fig 3 d).

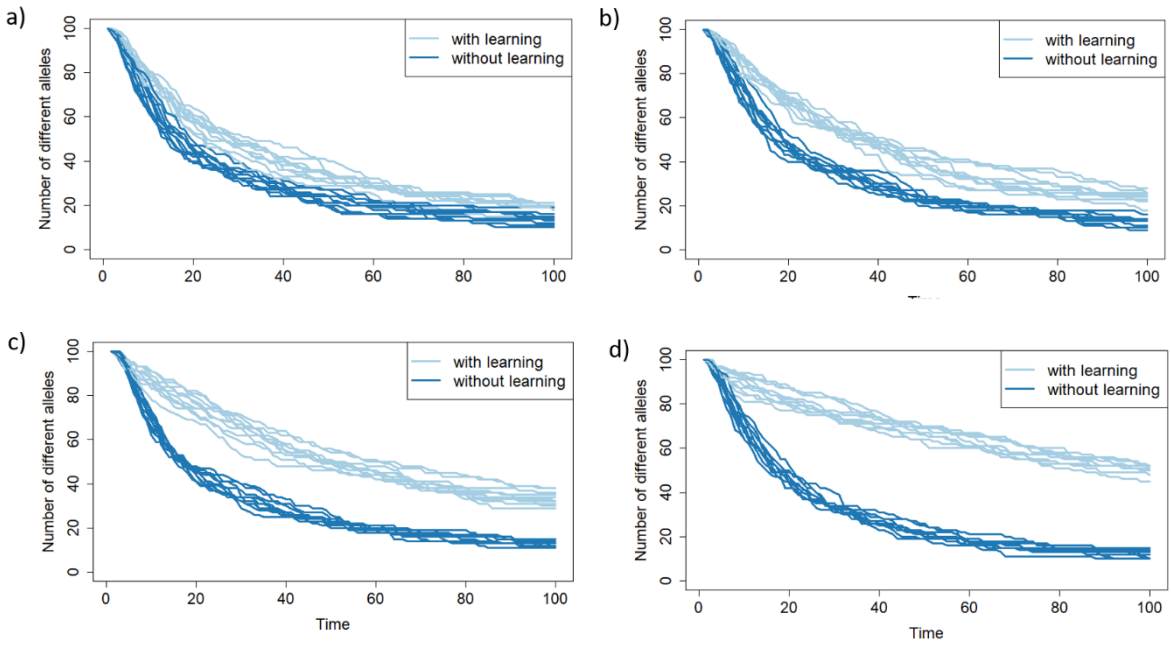

Figure 3: Effectiveness of learning dictates its effect on genetic drift. The figure shows the temporal dynamics of the number of alleles present in populations of prey subject to predation. All prey populations consist of 1000 individuals at the beginning of each step. Individuals killed by predators are replaced at the end of each step by a reproduction of surviving individuals (see Methods). Predators consume 500 prey individuals per time. Predators kill the same number of individuals in both learning and non-learning conditions to allow for a fair comparison. The upper bound on the probability to avoid/escape predator attack is set to 90% for panel a, 95% for panel b, 97,5% for panel c, and 99% for panel d. Each panel shows ten simulation runs for both learning and non-learning conditions.

The haploid sexual model

To test how is the effect of learning on the strength of genetic drift affected by the mode of reproduction, we have created an updated model in which prey individuals reproduce sexually. The results from this version of the model were qualitatively and quantitatively the same as the results from the haploid asexual version, further supporting the notion that learning from experience is an important factor affecting the strength of genetic drift.

The diploid sexual model

Finally, we created a version of the model with diploid sexually reproducing prey individuals. The results of this model show once again that learning from experience significantly reduces genetic drift (Fig 4). The main difference in comparison to the results of haploid versions of the model is that, as could be expected, genetic drift reduces genetic variability at a slower pace.

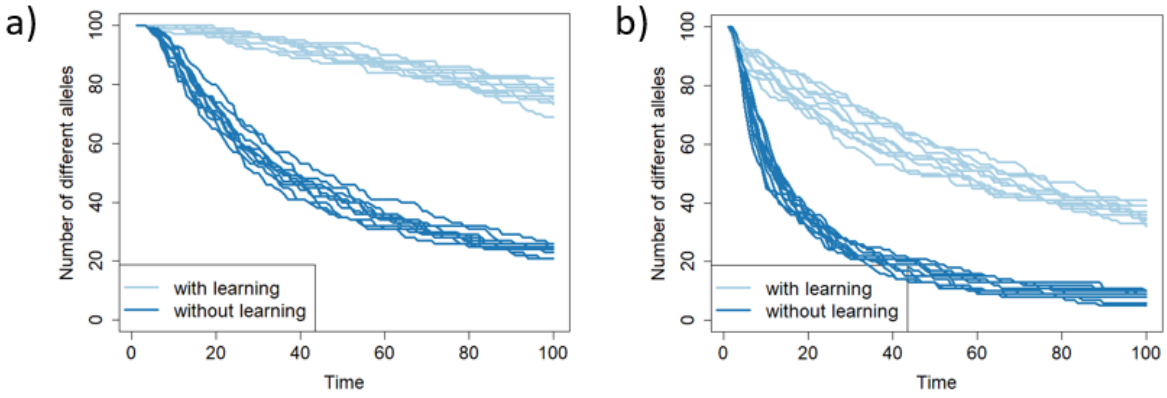

Figure 4: Effect of learning on the genetic drift in a diploid sexually reproducing population. Temporal dynamics of the number of alleles present in populations of prey subject to predation. All prey populations consist of 1000 individuals at the beginning of each step. Individuals killed by predators are replaced at the end of each step by a reproduction of surviving individuals (see Methods). In panel a, predators consume 500 prey individuals per time-step, while in panel b, predators consume 800 prey individuals per time-step. Predators kill the same number of individuals in both learning and non-learning conditions to allow for a fair comparison. The upper bound on the probability to avoid/escape predator attack is set to 99% for both panels. Each panel shows ten simulation runs for both learning and non-learning conditions.

Discussion

In this article, we have reasoned that learning from experience mitigates genetic drift. Then we developed an agent-based mathematical model to test this hypothesis. The results of our simulations confirm our hypothesis and demonstrate that learning mitigates genetic regardless of whether the affected organism is haploid or diploid or whether it reproduces sexually or asexually.

The conclusions of this article are purely theoretical. Their purpose is to point at the mathematically and logically inevitable existence of the relationship between the ability to learn from experience and genetic drift. The results presented here cannot, by themselves, tell us anything about how strong this effect is in nature. The crucial factor for determining this would be assessing how effectively animals can learn to avoid or escape a deadly danger. For most simulations in this article, we operated with an upper threshold of 90% or more. Arguably this probability may be too high in real-world settings. However, we believe that in certain conditions, it is more than plausible. For example, a study on barn owl has shown that these owls have a 90% chance of catching a stationary food item and only a 21% chance of catching moving items[4]. Furthermore, the owls were even less likely to catch an item moving towards them (18%), and most interestingly, if the food items were moving sideways, the barn owls were not able to catch them at all. Therefore, it seems feasible that the prey may learn simple strategies that would allow it to escape at least some types of predators with surprisingly high probability.

So far, in this article, we presented arguments and simulations that considered the effect of learning in the context of predation. However, for the same reasons, learning exerts the same influence on any genetic drift caused by any source of mortality (or sterility), which individuals of a given species may learn to avoid. For example, if individuals of a given species were able to learn to avoid traffic from their experience, the effect on the genetic drift would be identical with the learned ability to avoid or escape predators more efficiently. To go even further, any learned improvement in the ability to find a partner for mating, to find food and thus decrease the risk of starvation, to care for offspring and thus, increase their chance of reaching adulthood, etc., will, to some extent, help to preserve genetic variability despite the raw effects of drift.

Furthermore, the here-described relationship between learning and genetic drift also leads to some testable predictions. First, species that can learn more effectively should, on average,

better conserve genetic variability than species of less capable learners living in populations of similar size. Second but the connected prediction is that species that are "better learners" should have a lesser risk of extinction. This prediction is in good agreement with a recent study showing that bird species with greater behavioral plasticity have a smaller risk of extinction.[5] The third prediction is that causes of excess mortality, which individuals of a given species may learn to avoid, reduce genetic variability to a smaller degree than those which members of that species cannot learn to avoid or escape.

Overall, our results show that view considering genetic drift as independent on an underlying behavior of a given species is incorrect and that a simple act of learning may significantly mitigate the effects of genetic drift. Furthermore, we have shown that the level of protection against genetic drift varies in different situations, suggesting that loss of genetic variability by genetic drift is a more complex issue than it is generally believed. We hope that these findings will make existing models of evolution more precise and could prove useful in a variety of topics, including the development of effective species protection strategies, studies of the evolutionary past as well as evolutionary future.

Methods

We start with a given number of prey individuals (1000) and an initial number of unique alleles (100). The individuals are assumed haploid and each obtains a randomly chosen allele. Moreover, each individual has a probability of avoiding or escaping the predator's attack (initially 0.5). Each simulation runs for a given number of time steps (100). Predation is implicit: each time step, a given number of prey individuals are removed from the population – an individual is randomly selected, and its probability to avoid/escape predation is used to decide whether the predator's attack is successful. If yes, the individual is removed from the

population. This random selection of prey is repeated until a given number of prey are removed from the population. If the attack is not successful and prey are learning, the probability of avoiding/escaping predation of the surviving prey is increased by a given proportion (0.75); we set an upper bound on the probability to avoid/escape predation. Finally, the removed prey individuals are replaced by new ones: at the end of the time step, a surviving individual is randomly chosen (with repetition) and gives birth to one offspring, until the population is restored. Each offspring inherits the allele from its parent, and its probability of avoiding or escaping the predator's attack is set to the initial value (0.5).

The haploid sexual and diploid sexual variations of the model differ from the above description only in the way reproduction is carried out. In the model with haploid sexually reproducing individuals, following predation, mates are formed randomly and produce one offspring the allele of which is randomly taken from one of the mates. In the model with diploid sexually reproducing individuals, each individual has two alleles, randomly chosen initially from the same set of 100 alleles as considered for haploid individuals. Following predation, mates are again formed randomly and produce one offspring each allele of which is randomly taken from the respective pairs of alleles of its parents.

The above-described model was run using statistical software R, v. 4.0.1.

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Authors' Contribution

P.L. formulated the research problem, interpreted the results, designed the simulation scenarios, and co-wrote the manuscript. J.B.V. supervised the project and obtained funding. L.B. developed and coded the mathematical model, interpreted the results, designed the simulation scenarios and co-wrote the manuscript. All authors reviewed the manuscript.

Code availability

The computer code used to generate results reported in this article can be accessed at https://github.com/PeterLenart/Learning_Mitigates_Genetic_Drift.

Conflict of interest

The authors declare no conflict of interest.

References

1. Lequime, S., Fontaine, A., Gouilh, M. A., Moltini-Conclois, I. & Lambrechts, L. Genetic Drift, Purifying Selection and Vector Genotype Shape Dengue Virus Intra-host Genetic Diversity in Mosquitoes. *PLOS Genet.* **12**, e1006111 (2016).
2. Lynch, M. *et al.* Genetic drift, selection and the evolution of the mutation rate. *Nat. Rev. Genet.* **17**, 704–714 (2016).
3. Lenart, P., Scheringer, M. & Bienertova-Vasku, J. The Pathosome: A Dynamic Three-Dimensional View of Disease–Environment Interaction. *BioEssays* **41**, 1900014 (2019).
4. Shifferman, E. & Eilam, D. Movement and direction of movement of a simulated prey affect the success rate in barn owl Tyto alba attack. *J. Avian Biol.* **35**, 111–116 (2004).

5. Ducatez, S., Sol, D., Sayol, F. & Lefebvre, L. Behavioural plasticity is associated with reduced extinction risk in birds. *Nat. Ecol. Evol.* **4**, 788–793 (2020).