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Letter

The hidden threat: genetic load dynamics in tetraploids and diploids

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Abstract

Polyploids are organisms with three or more chromosome sets, which often give them different physiology, morphology, and evolutionary properties. The impact of polyploidy on the genetic load dynamics, in terms of accumulation, fixation, and purging of deleterious mutations, is largely unknown. Here, we use forward-in-time genomic simulations to compare the fitness effects of genetic load components assuming diploid and tetraploid populations under different demographic scenarios. Under partial recessiveness, we show that tetraploids tend to accumulate a higher genetic load than diploids in small and large populations, with negative consequences on their fitness. Under complete recessiveness, the same pattern is observed only in small populations, but similar load effects are found in large diploid and tetraploid populations under mutation-drift equilibrium. The only scenario where tetraploids suffered less from the effects of genetic load was in bottlenecked populations under complete recessiveness. Our results highlight the importance of factors such as demography, dominance and selection coefficients, and genetic drift in shaping the different patterns of accumulation of deleterious mutations in diploids and tetraploids. While some studies have suggested that tetraploids might have a reduced genetic load because recessive mutations could be masked more efficiently, our findings align more closely with evidence that tetraploids frequently accumulate a higher genetic load than diploids. This insight is significant for assessing the role of ploidy in the conservation of endangered species.

Keywords: polyploidy, forward simulations, small populations, deleterious mutations, purifying selection, genetic drift

Introduction

Polyploidization occurs when the genome undergoes one or more rounds of duplication, resulting in the presence of multiple sets of chromosomes. Polyploidy appears through whole genome duplication (WGD), which may result from errors in cell division, spontaneous duplication events within a species, or hybridization between species (Otto, 2007; Spoelhof et al., 2020). Polyploids can be classified based on their origin or their patterns of inheritance. Autopolyploids arise from genome duplication within a species and typically show polysomic inheritance due to random chromosome pairing. Allopolyploids, formed through hybridization between species followed by WGD, often display disomic inheritance, with preferential pairing of homologous chromosomes from each parent (Meirmans & Van Tienderen, 2013; Van de Peer et al., 2017). Recent advances in sequencing entire genomes and transcriptomes have revealed hundreds of polyploidization events across the tree of life during the last 500 million years of evolution (Li et al., 2021; Van de Peer et al., 2017). Ancient WGDs are challenging to detect, as duplicated genes may diverge over time, acquiring new functions or being lost due to chromosomal rearrangements (Freeling et al., 2012). These changes can alter genome architecture and, in some cases, lead to diploid-like chromosomal pairing behavior during meiosis (Ma & Gustafson, 2005). This process, known as diploidization, refers to the functional return of a polyploid genome to a diploid state and occurs to restore normal meiotic behavior (Brooker & Schrider, 2025; Li et al., 2021). As a result, much of our current understanding of polyploidy is based on relatively recent events (Oyston et al., 2022).

Polyploidy is widespread in several plants, particularly among angiosperms and ferns (Goswami, 2013; Toda & Okamoto, 2020). It is estimated that approximately 15% of speciation events in angiosperms and 31% in ferns are directly associated with increases in ploidy level (Wood et al., 2009). Although it is considered rare in most animals, polyploidy is reported in some groups, including amphibians (Mezzasalma et al., 2023; Schmid et al., 2015) and fishes (Braasch & Postlethwait, 2012; Comber & Smith, 2004). It is well documented in sturgeons (Vasil'ev, 2009), as well as in several teleosts (Braasch & Postlethwait, 2012; Conant, 2020). Among invertebrates, cases of polyploidy have been observed in insects (Ren et al., 2020), molluscs (Hallinan & Lindberg, 2011), and crustaceans (Symonová et al., 2018).

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Polyploidization can cause harmful but also beneficial changes in organisms. The increased genetic material can make the genome more unstable. Over time, this can result in chromosomal rearrangements and changes in gene expression that may have negative effects, especially on highly conserved genes (Fox et al., 2020). At the same time, by adding a new set of chromosomes, duplicated genes might not be tied down to perform only the functions carried out by the original genes. This offers more flexibility, allowing natural selection to favor the development of new functions (Otto, 2007).

In addition, initial gene redundancy in polyploids can buffer against the effects of deleterious mutations even if, in the long term, this can also enhance the opportunity for these mutations to accumulate in the genome (Baduel et al., 2018; Otto, 2007; Ronfort, 1999). Studying the dynamic of deleterious mutations is crucial for species conservation as they can threaten population survival, particularly in small populations where genetic drift can randomly increase their frequency up to fixation and inbreeding tends to unmask recessive deleterious alleles (Eyre-Walker & Keightley, 2007; Hedrick & Garcia-Dorado, 2016; Lynch et al., 1995).

Although polyploidy has shaped the evolutionary history of many species, including economically important ones in agriculture (e.g., potatoes, wheat, corn, sugarcane, strawberries) and aquaculture (e.g., salmonids, sturgeons), the population genetic theory of polyploids has lagged behind that of diploids. In particular, the genetic load and its impact on individual fitness are poorly known, and only a handful of studies investigated this issue (Baduel et al., 2018; Monnahan et al., 2019; Otto, 2007; Otto & Whitton, 2000; Ronfort, 1999).

Broadly, genetic load is the presence of deleterious mutations in the genome. More specifically, genetic load was defined in different ways, emphasizing, for example, the processes giving rise to the load (Charlesworth & Barton, 1996; Haag & Roze, 2007; Lynch et al., 1995), or considering the load effects on fitness, genetic variation, or population persistence (Agrawal & Whitlock, 2012; Kimura et al., 1963; Lynch & Gabriel, 1990; Muller, 1950). Here, we define genetic load as the accumulation of deleterious genetic variants in individual genomes, distinguishing between realized (or expressed) load and masked (or inbreeding) load (Bertorelle et al., 2022). These load fractions jointly contribute to the total genetic load of an individual or a population but independently produce an immediate or a potentially future fitness reduction, respectively. A new deleterious mutation, if fully recessive, does not affect its carrier's fitness and contributes only to the masked load. The same mutation, however, may contribute to the realized load in the homozygous offspring of carrier crosses. Partially recessive mutations have both realized and masked effects, in different proportions depending on the dominance coefficient.

Polyploids are expected to differ from diploids in genetic load dynamics due to genetic redundancy, complex segregation, and recombination patterns during meiosis (Butruille & Boiteux, 2000; Monnahan et al., 2019). Some authors argue that polyploids benefit from being able to mask deleterious mutations with redundant wild-type alleles (Comai, 2005; Haldane, 1937; Waldman et al., 2018). Conversely, other authors contend that this advantage is transitory and eventually becomes a drawback, as it reduces the effectiveness of purifying selection in removing these alleles, allowing them to accumulate at high frequencies (Otto, 2007; Otto & Whitton, 2000). Also, it has been observed that the level of allelic dominance affects the genetic load differently in diploid and tetraploid populations in *Arabidopsis arenosa*. Under partial recessiveness, the genetic load is higher in tetraploids, while the opposite is true under complete recessiveness (Monnahan et al., 2019).

Computer simulations are a useful tool for understanding complex evolutionary processes when theoretical derivation is not possible, empirical data and estimations are difficult to obtain, and comparisons across species are unfeasible (Kyriazis et al., 2023). This is the case when the goal of the study is to understand the genetic load dynamics and fitness effects under different demographic scenarios, selection and dominance coefficients, and level of ploidy. In particular, individual-based forward genetic simulations are a flexible tool for constructing realistic demographic, genomic, and life history models.

Here, we explore how ploidy level, population size, and dominance coefficients shape the accumulation and fixation of deleterious mutations using individual-based forward genetic simulations. To measure these dynamics, we estimated the fitness effects of unconditional deleterious mutations (i.e., we do not consider rare deleterious mutations that become neutral or beneficial due to environmental changes) under different demographic and genetic scenarios. We focused on the impact of the population size (small vs. large), demographic trends (stable vs. bottlenecked populations), and allelic dominance (partial vs. complete recessiveness). Our findings are relevant for comparing populations with different ploidy levels within the same species, as well as for comparisons between species with different ploidy levels. These insights could also have practical implications in conservation genetics. Understanding how polyploidy interacts with demographic factors and mutation dominance in shaping genetic load can help, for example, in designing more effective conservation strategies specific to diploid or polyploid species. This knowledge could guide the selection of individuals for captive breeding or reintroduction programs, helping to balance the trade-offs between maintaining genetic diversity and minimizing the accumulation of deleterious mutations in endangered polyploid and diploid populations alike.

Materials and methods

The model

Individual-based forward genetic simulations were performed using SLiM v4.0 (Haller & Messer, 2022). We developed our model starting from the script provided by William Booker (https://github.com/MesserLab/SliM-Extras/blob/master/models/autotetraploid.slim) to simulate autotetraploid populations with pure tetrasomic inheritance (henceforth tetraploid) with a non-Wright–Fisher implementation that allows overlapping generations and detailed specifications of the reproduction process and the fitness function. The model simulates random mating of hermaphroditic individuals each carrying four (for tetraploids) or two (for diploids) homologous chromosomes of 200 Megabases. The callback addRecombinant was used to allow crossing-over events between any possible chromosome pairings, as occurs in autotetraploids. The populations were regulated by setting carrying capacities for each ploidy. To contain the running time of the simulation, each mating pair could produce only one offspring per generation.

Unconditionally deleterious mutations were simulated using a mutation rate of 1×10^{-9} mutations per base per generation and a recombination rate of 1×10^{-8}, values consistent with empirical data from eukaryotic organisms (Lynch, 2010; Wang & Obbard, 2023). The same parameters were used for diploid and tetraploid populations, with the only exception that diploid populations were simulated with twice the number of individuals to ensure similar chromosome copy numbers across ploidy levels.

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Each scenario was replicated 20 times to test the effects of different factors on the accumulation of genetic load and control the stochastic effects of drift and mutations.

The per-individual realized and masked loads were estimated in lethal equivalents (LE), a unit of genetic load that quantifies the cumulative effect of deleterious mutations. We calculated it by summing the products of selection coefficients and dominance coefficients for heterozygous mutations, following Bertorelle et al. (2022):

$$\text{Realized } load \text{ (individual)} = \sum_{i=1}^{L(hom)} s_i + \sum_{j=1}^{L(het)} h_j s_j,$$

$$\text{Masked } load \text{ (individual)} = \sum_{j=1}^{L(het)} (0.5 - h_j)\, s_j,$$

where *L(hom)* and *L(het)* are the homozygous and the heterozygous loci, respectively, and h and s are the dominance and selection coefficients of the deleterious mutations, respectively. For tetraploids, the dominance coefficient can take different values depending on the number of mutant alleles (see below).

The fitness of each individual depends on the additive effect at each locus and was computed as:

$$\Pi_{i=1}^{L(hom)} (1 - s_i)\, \Pi_{j=1}^{L(het)} (1 - h_j s_j).$$

In the non-Wright–Fisher model of SLiM, fitness measures the probability of individual survival, i.e., the model is under hard selection.

Fitness and genetic load were computed every 100 simulation steps (henceforth cycles) for all individuals in small populations and for 100 random individuals in large populations. Each cycle corresponds to one year, and the generation time of both ploidies was about two years. We consider that a population has reached mutation-drift equilibrium when the masked and/or the realized load remained stable throughout the simulation.

Demographic scenarios

Diploid populations

We simulated two populations regulated by different carrying capacities (K): one large (N = ~4,000) and one small (N = ~80). All simulations ran for 30,000 cycles, or until extinction. We refer to these as "stable K" simulations. A bottleneck scenario was then simulated to analyze the effect of a drastic reduction in population size on the genetic load. We first simulated a large population ($N \sim 4{,}000$) for 25,000 cycles, followed by a 10% decline in each cycle until reaching the expected collapsed size ($N \sim 80$). This small population was then simulated for 5,000 additional cycles.

Tetraploid populations

The large and small tetraploid populations simulated had carrying capacities regulating N to ~ 2,000 and ~ 40, respectively. In the bottleneck scenario, after a burn-in period of 25,000 cycles, the large population ($N \sim 2{,}000$) experienced a 10% decline in each cycle until reaching the expected collapsed size ($N \sim 40$). The post-bottleneck population was then simulated for 5,000 additional cycles.

Distribution of selection (s) and dominance (h) coefficients

Diploid populations

Since the values of s and h are often unknown, we performed a set of preliminary simulations using unconstrained uniform distribution for s (ranging from −1 to 0) and h (ranging from 0 to 0.5). We allowed natural selection in the model to act on these mutations, generating an emergent empirical joint distribution of s and h values, following Dussex et al. (2023). This preliminary simulation of $N \sim 2{,}000$ for tetraploids and $N \sim 4{,}000$ for diploids was run for 100,000 cycles and sampled the emergent distribution of s and h values. From the last burn-in cycle (100,000), the values of s and h were sampled from 1,000 different mutations for each replicate of the genetic load simulations for diploid and tetraploid populations. We refer to this method as "*hs-input*."

Tetraploid populations

To assign the dominance coefficients for the three types of heterozygous tetraploid genotypes (*AAAa*, *AAaa*, and *Aaaa*), a dosage-like model (i.e., $h_1 = h_1$; $h_2 = 2h_1$; $h_3 = 3h_1$) was implemented in the *hs-input* model. The dosage-like model has been used in other theoretical and empirical studies of polyploid species (Layman & Busch, 2018; Ozimec & Husband, 2011; Ronfort, 1999). For the burn-in simulation to generate the *hs-input* starting file, the values of s were assigned from a uniform distribution (ranging from −1 to 0), and the values of h_1 (for the genotype *AAAa*) from a uniform distribution (ranging from 0 to 0.25), from where they scale up as $h_2 = 2h_1$ for *AAaa* and $h_3 = 3h_1$ for *Aaaa*. Values of h larger than 0.5 occurred only for a tiny fraction of the simulated *Aaaa* genotypes (<0.001%) and were removed to avoid meaningless negative loads.

Partial and complete recessive deleterious mutations

Most deleterious mutations are recessive or partially recessive (Fernández et al., 2004; García-Dorado & Caballero, 2000). To independently test the effect of dominance coefficients on determining the relative amounts of genetic load in tetraploid and diploid populations, we modeled the same demographic scenarios under partial and complete recessiveness.

Results

Emergent relationship between h and s

The emergent pattern of h and s coefficients from our simulations with uniform input values generated the expected negative relationship (García-Dorado & Caballero, 2000; Grossen et al., 2020), where highly deleterious mutations tend to be more recessive than slightly deleterious mutations (Figure 1A and B). This is because mutations with a combination of large h and s coefficients are effectively removed by purifying selection, whereas mutations with smaller s coefficients can tolerate a broader range of h coefficient values. For tetraploids, the average s was −0.21, and the average h was 0.07, while for diploids the average s was −0.19 and the average h was 0.12. Under complete recessiveness ($h = 0$), the distribution of fitness effects that emerged after 100,000 cycles had an average s of −0.46 in tetraploids for N ~2,000 (Figure 1C) and an average s of −0.39 in diploids with N ~4,000 (Figure 1D). With both levels of recessiveness, tetraploids showed a higher (negative) average s than diploids, probably due to an increased opportunity of masking highly deleterious alleles.

Simulations with large population size

In all simulated replicates, both diploid and tetraploid populations persisted until cycle 30,000. Under partial recessiveness, the individual masked load in tetraploids was approximately three times larger than in diploids (2.84 vs. 0.97 LE, on average, respectively; see Figure 2A). Tetraploid individuals also had twice as much realized load than diploids (0.18 vs. 0.09 LE, respectively, Figure 2B).

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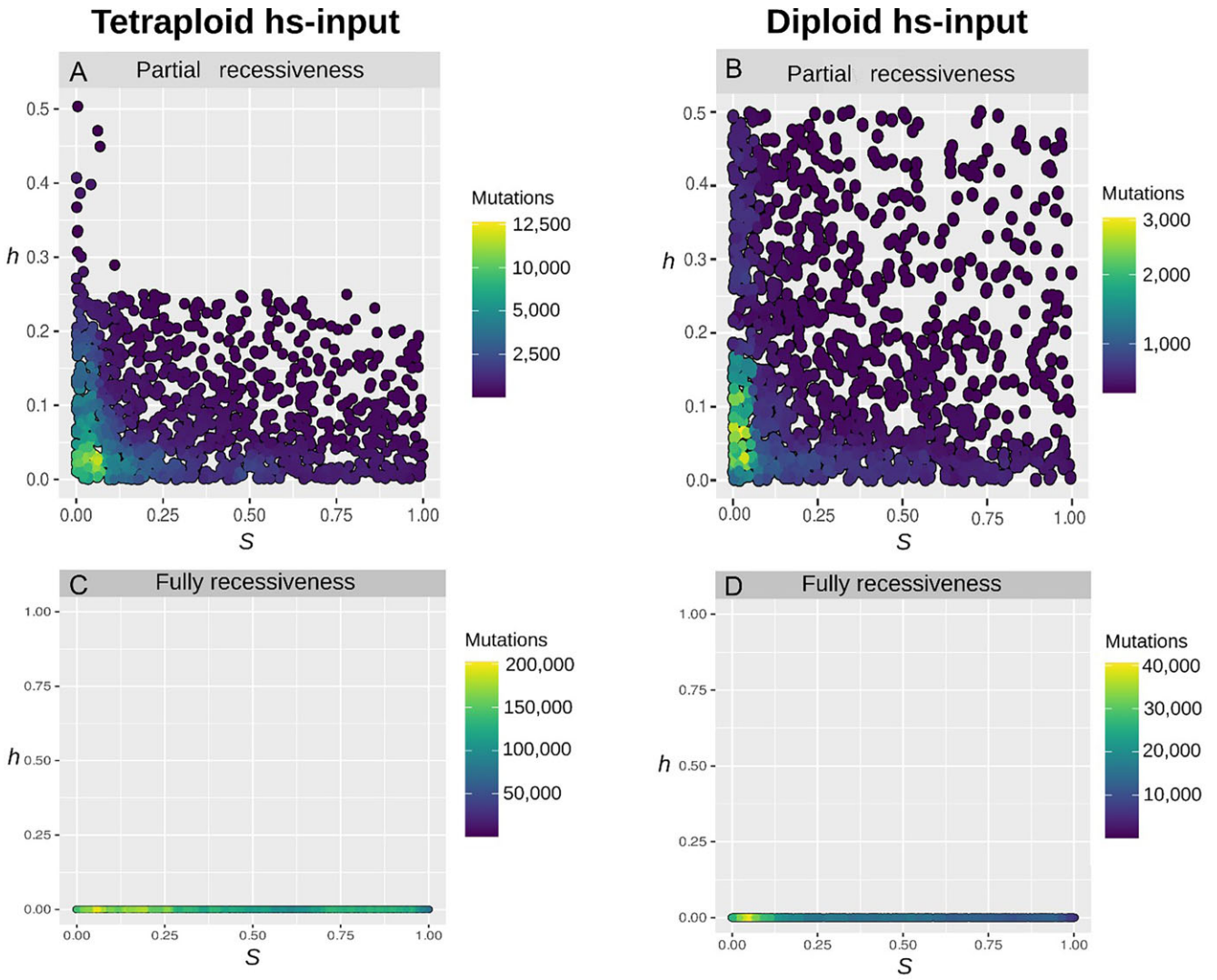


Figure 1. Emergent relationship between *h* and *s* after 100,000 burn-in cycles, each point represents a mutation. (A) partially recessive mutations of ~2,000 tetraploid individuals; (B) partially recessive mutations of ~4,000 diploid individuals; (C) complete recessive mutations of ~2,000 tetraploid individuals; (D) complete recessive mutations of ~4,000 diploid individuals.

This higher realized load in tetraploids led to a lower fitness (0.83) compared to diploids (0.92) (Figure 2C).

Under complete recessiveness, tetraploids showed again a greater masked load than diploids, with the difference being much more evident this time (144.44 and 7.25 LE, respectively, Figure 2D). The realized load was very small (< 0.05 LE) and similar in tetraploids and diploids (Figure 2E), and the fitness values were high and similar for both ploidies, 0.96 and 0.97, respectively (Figure 2F).

Simulations with small population size

None of the 20 replicates of the small tetraploid populations in the partial recessiveness scenario reached 30,000 cycles due to earlier population extinction. This early extinction coupled with the strong influence of genetic drift, prevented genetic load from reaching equilibrium, a process that actually occurs in drastically reduced populations (LaBar & Adami, 2017; Willi et al., 2013). Nevertheless, all replicates reached at least cycle 7,000, and Figure 3 shows the results up to this cycle.

Under partial recessiveness, tetraploids showed a masked load between two and three times larger than diploids (Figure 3A and Supplementary Table S1). The realized load increased in both diploids and tetraploids, but the absolute values and the increased speed were larger in the latter (Figure 3B). The higher realized load in tetraploids resulted in a more drastic decline in fitness. After 7,000 cycles, the mean fitness was 0.30 for tetraploids and 0.67 for diploids (Figure 3C).

Under complete recessiveness, the genetic load and fitness patterns were similar to those observed under partial recessiveness. Similar to what is found in large populations, and consistently with the expectation that fitness is reduced only when deleterious variants are in homozygosity, the masked load was much greater in tetraploids than in diploids (Figure 3D). For the same reason, the increase in the realized load was lower for both ploidies (Figure 3E), compared to the scenario under partial recessiveness. At the end of the simulation, the mean fitness of small tetraploid populations was 0.63, while for small diploid populations was 0.87 (Figure 3F).

Simulations of bottlenecked populations

Genetic load and fitness levels before the bottleneck had the same values observed in the large population scenarios (see Figures 2

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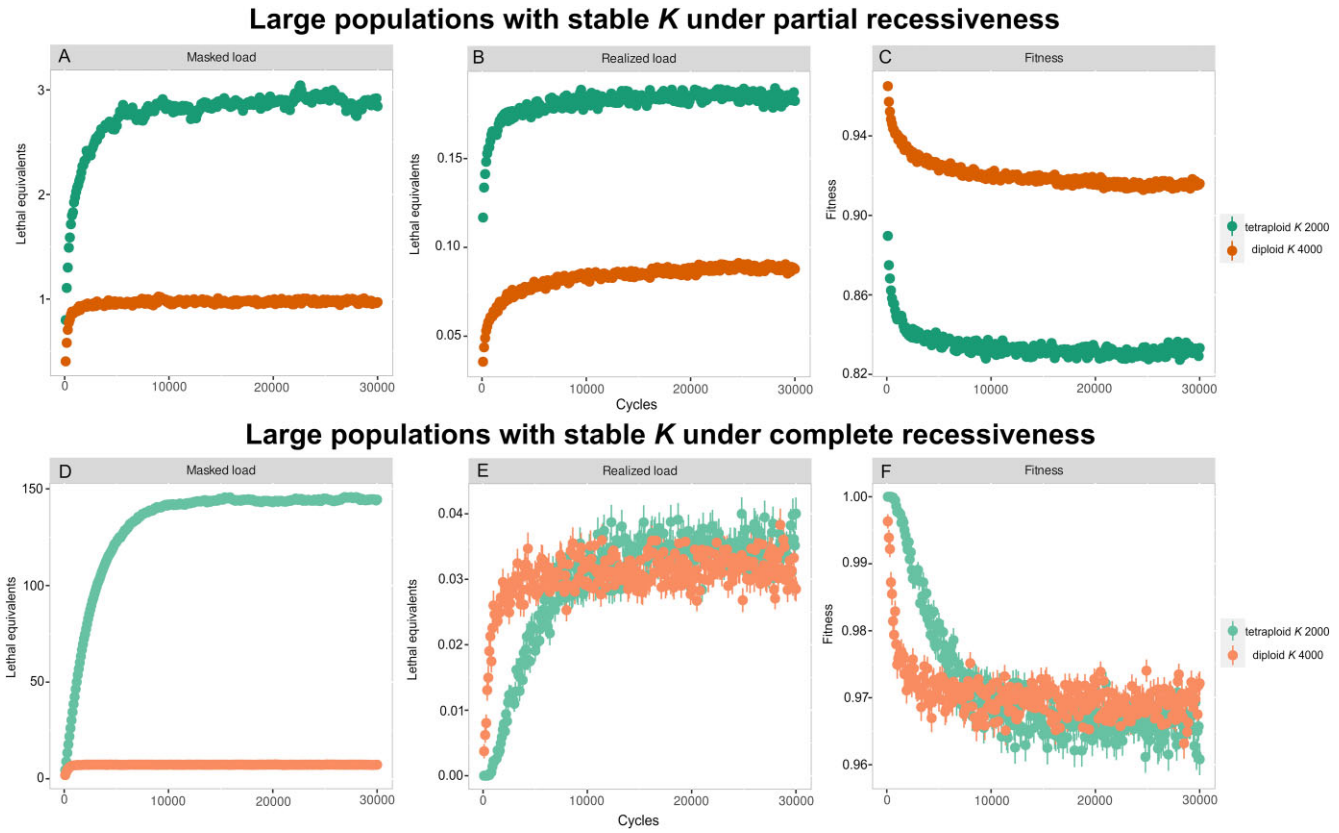


Figure 2. Simulations of large populations under partial and complete recessiveness comparing diploid and tetraploid individuals. Each point represents the mean value across 20 replicates computed for a sample of 100 individuals every 100 cycles. The whiskers of the points are the standard error. Note that the scales are different for each graphic.

and 4). After the bottleneck, a fraction of the masked load was converted into realized load producing a fitness decline, but the impact of the bottleneck was different in the scenarios with partial or complete recessiveness.

Under partial recessiveness, both masked and realized loads are always greater in tetraploids than in diploids (Figure 4A and B), with a fitness loss 5,000 cycles after the bottleneck of approximately 60% in tetraploids and 35% in diploids (Figure 4C). On the other hand, under complete recessiveness, the realized load had a rapid increase after the bottleneck, followed by a decrease likely due to purging and a subsequent increase more intense in diploids where masking was less efficient. This is the only scenario where diploids had a higher (almost double) realized load than tetraploids (Figure 4E). At the end of the simulations, the percentage of fitness loss 5,000 cycles after the bottleneck was approximately 7% and 14% in tetraploids and diploids, respectively (Figure 4F).

The total load (the sum of masked and realized loads) is much larger in tetraploids than in diploids, due to the larger masking effects when recessiveness is complete and four copies of the deleterious variants are required to reduce the individual fitness. However, the direct negative effect on the fitness of the bottleneck appears larger in diploids when recessiveness is complete since the collapsed diploid populations carry more homozygous mutations than tetraploid populations (Supplementary Table S2).

Additional simulations

To assess the robustness of our analysis, we performed additional simulations using identical carrying capacities and distribution of fitness effects for both diploids and tetraploids. We also used a gamma distribution for s (Kardos et al., 2021) and dominance coefficients from an additive model (Booker & Schrider, 2025). The patterns observed in genetic load and fitness differences between tetraploids and diploids were consistent across all these tests (Supplementary Tables S3 and S4 and Figures S1–S4).

Discussion

We used individual-based forward genetic simulations under different demographic scenarios and dominance models to compare the genetic load dynamics between tetraploid and diploid populations. We analyzed the realized and masked components of the genetic load, which are indices of the present and possible future negative effects on fitness. We also tracked temporal fitness trends assuming a multiplicative model of multilocus effects.

While some studies proposed that tetraploidy may reduce genetic load through the masking of recessive deleterious mutations (Comai, 2005; Hedrick, 1987; Husband, 2016), our results align more with evidence from empirical and theoretical studies indicating that this effect is transient and context dependent. For instance, Ronfort (1999) demonstrated that tetrasomic inheritance limits the efficacy of purifying selection, leading to greater load accumulation over time. Otto & Whitton (2000) similarly argued that polyploid genomes may buffer mutations initially but are prone to relaxed selection. Empirical data from yeast (Selmecki et al., 2015) confirm that polyploids often accumulate mutations post-duplication. Baduel et al. (2018) further emphasized that polyploidy follows a high-risk, high-gain path, which often leads to

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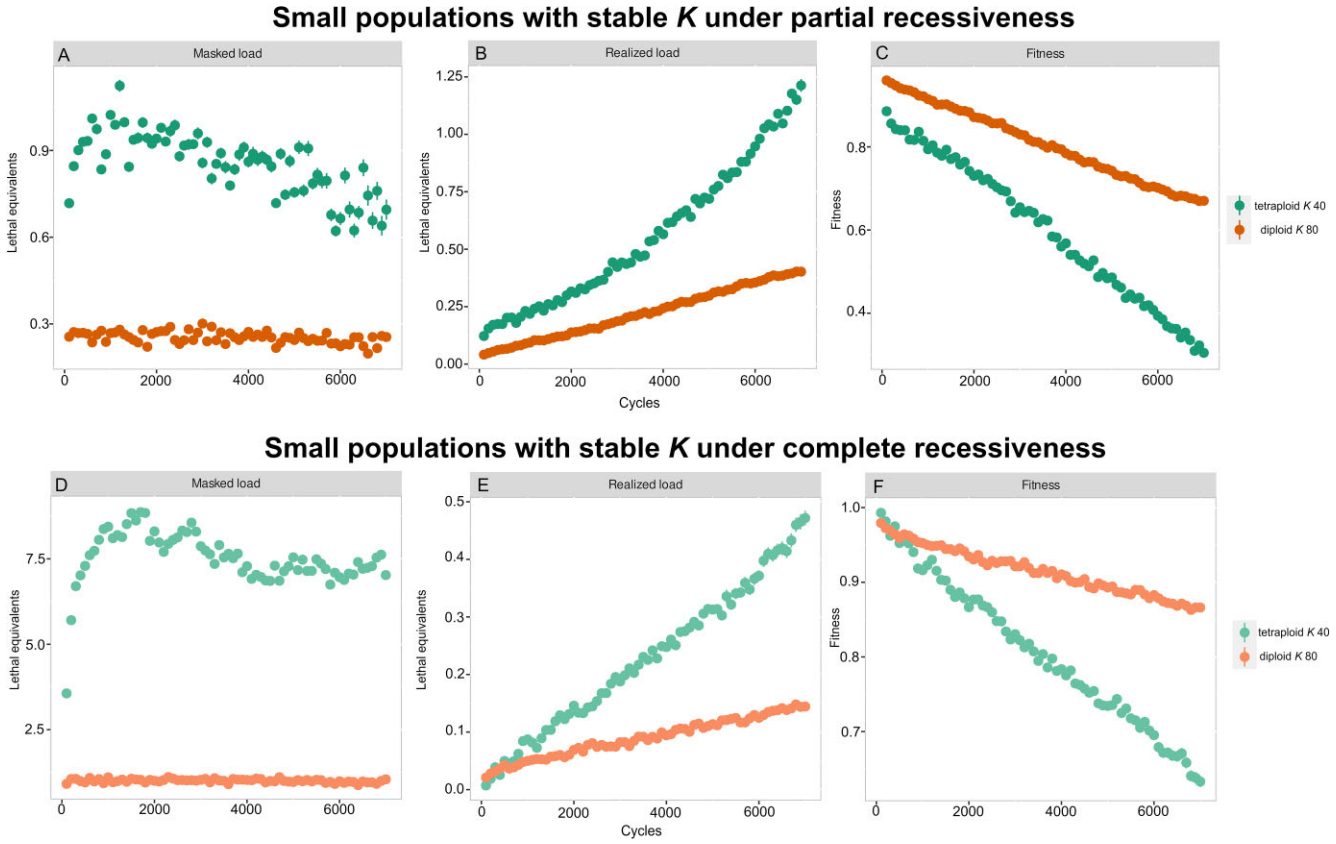


Figure 3. Simulations of small populations under partial and complete recessiveness comparing diploid and tetraploid individuals. Each point represents the mean value across 20 replicates computed for all individuals every 100 cycles. The whiskers of the points are the standard error. Note that the scales are different for each graphic.

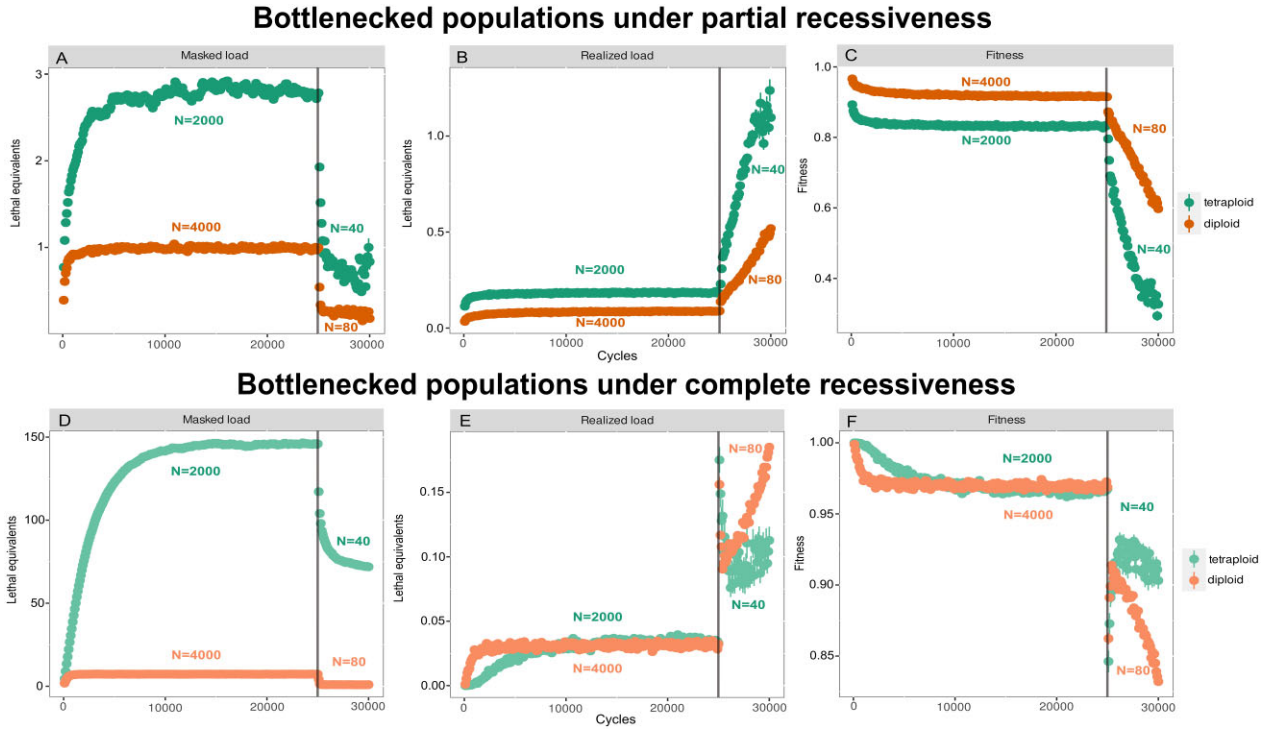


Figure 4. Simulations of bottlenecked populations under partial and complete recessiveness. Each point represents the mean value across 20 replicates computed for a sample of 100 individuals in the large populations and for all individuals in the post-bottleneck populations every 100 cycles. Bottleneck starts in the cycle 25,000 (black line). The post-bottleneck population ran for another 5,000 cycles. Note that the scales are different for each graphic.

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diploidization, with long-term disadvantages outweighing early benefits. Vlček et al. (2025) concluded that WGDs may shift adaptive potential but also increase realized load in *Arabidopsis arenosa*.

Theoretical predictions suggest that assuming a large population size, a low deleterious mutation rate, a partial recessiveness, and tetrasomic inheritance for tetraploids, the predicted genetic load is proportional to 4μ for tetraploids and to 2μ for diploids, where μ represents the deleterious mutation rate (Otto & Whitton, 2000). This simple twofold ratio between the genetic load in tetraploids and diploids is rarely observed in our simulations. We should, however, consider that the simulations allow the study of different non-equilibrium demographic scenarios, incorporate probability distributions of selection and dominance coefficients, and partition the load into components that differently affect fitness in present and future generations. In addition, while previous studies examined genetic load as the number of deleterious mutations, we measured it using a metric, LE, designed to more realistically weigh mutations differently based on their effects in various genotypes.

In both large and small populations, whether under partial or complete recessiveness, tetraploids exhibited almost always a higher realized and masked load than diploids.

In large populations under partial recessiveness, the masked and realized loads in tetraploids were three and two times higher than in diploids, respectively. This is because deleterious alleles at heterozygous sites are only partially masked and tend to be more harmful in tetraploids. In small populations, both under partial and complete recessiveness, the realized load is constantly increasing until population extinction in both diploids and tetraploids, but again masked and realized loads are always higher in tetraploids. In large populations under complete recessiveness, the situation is slightly different. Tetraploids had a 20-fold greater masked load than diploids, but the realized load was similar between the two ploidies. The dynamics differed before reaching equilibrium, with diploids initially showing a slightly higher realized load than tetraploids. This could be attributed to the longer time it takes to accumulate homozygous alleles in tetraploid populations. These dynamics are particularly interesting in scenarios where populations are out of equilibrium, such as small edge populations during geographic expansions (Griswold, 2021) or populations recovering from a demographic crash (Layman & Busch, 2018). In such cases, the delayed accumulation of homozygous alleles in tetraploids may temporarily mitigate the negative effects of mutations, potentially giving tetraploids an advantage in maintaining higher fitness during bottlenecks. Therefore, depending on the fraction of completely/partially recessive deleterious mutations, there might be situations where tetraploids might have an advantage over diploids by effectively masking most deleterious mutations (Madlung, 2013).

In bottlenecked populations under partial recessiveness, the pattern of tetraploids having greater masked and realized loads than diploids is confirmed. Much of the masked load is lost after the bottleneck, but some becomes expressed due to genetic drift. The realized load continues to grow in small post-bottleneck populations (as in the scenario with small populations). Two thousand cycles after the demographic drop, tetraploid individuals have an average of 44.4 homozygous mutations, compared to 90.9 in diploids (Supplementary Table S1). What is particularly noteworthy is that, although tetraploids have fewer homozygous mutations, the mutations tend to be more harmful (average s-value of –0.01 for tetraploids and –0.002 for diploids). This difference is likely due to the higher masking efficiency in tetraploids that allowed some highly deleterious mutations to escape purifying selection. These mutations, when expressed in post-bottleneck populations, resulted in lower fitness for tetraploids compared to diploids.

The bottleneck scenario under complete recessiveness reveals distinct dynamics between diploid and tetraploid populations, providing important insights into the interplay between dominance coefficients and demographic factors. After the bottleneck and an initial unstable phase, the realized load increased faster and fitness declined more quickly in diploids than in tetraploids. This difference is likely due to the higher number of homozygous mutations per individual in diploids compared to tetraploids (26.2 vs. 8.2 after 30,000 cycles, respectively, Supplementary Table S2). In this simulation setting, tetraploids have an advantage due to two key factors. First, the lower probability of reaching homozygous mutations in tetraploid genotypes provides a genetic buffer against recessive deleterious alleles. Second, under complete recessiveness, purifying selection is more effective in tetraploids at removing highly deleterious mutations in homozygosity, as these mutations are fully exposed to selection. Since mutations in tetraploids tend to be more deleterious in our simulations, they are more efficiently eliminated compared to diploids. This results in the persistence of mutations that are only slightly deleterious in both ploidy levels.

Model limitations

As with all simulation-based approaches, several details can be incorporated to enhance the realism of the model. However, certain assumptions are necessary to ensure interpretability and computational feasibility. In particular, positive selection (i.e., the occurrence of beneficial mutations) and epistasis (i.e., the interaction between genes) are not considered in our study. Empirical studies (e.g., Hijmans et al., 2007; Karunarathne et al., 2018) and simple theoretical models based on one or two biallelic loci (e.g., Griswold, 2021; Griswold & Williamson, 2017; Mostafaee & Griswold, 2019) suggest that the establishment, dispersal, and successful growth of polyploid populations outcompeting diploid populations, especially during the colonization of new habitats, can be mediated by new mutations that increase the fitness and/or by strong interaction effects. Our study focuses on the negative consequences of independent mutations. However, it directly quantifies genetic load in individual genomes, something not possible when modeling only one or a few loci, and it explicitly distinguishes between the immediate and future effects of deleterious mutations.

Furthermore, it is important to note that our simulations assume pure tetrasomic inheritance. While this assumption is appropriate for the scope of our study, it might not fully apply, for example, to allopolyploids, in which chromosome pairing often occurs in pairs rather than tetrads (Spoelhof et al., 2017; Svačina et al., 2020) or to autotetraploids experiencing secondary diploidization, with intermediate disomic-tetrasomic inheritance (Booker & Schrider, 2025; Stift et al., 2008).

Conclusions

We demonstrated that genetic load dynamics in tetraploid or diploid genomes depend on population size and its temporal changes, as well as on the distribution of dominance coefficients of deleterious mutations. Ploidy also influences the distribution of selection coefficients, further complicating its relationship with genetic load.

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As a general conclusion, our results suggest that if deleterious mutations are partially recessive (i.e., they have a negative fitness effect in both homozygotes and heterozygotes), tetraploids consistently experience greater genetic load accumulation and expression compared to diploids. A similar pattern is observed in small populations under complete recessiveness (i.e., deleterious mutations negatively impact fitness only in homozygous genotypes), but in large populations the realized load remains consistently small and comparable between ploidy levels. The only scenario in which tetraploids appear to have a smaller realized load than diploids, and therefore a fitness advantage, is during a demographic decline under complete recessiveness. However, given the reasonable assumption that many deleterious mutations exhibit some degree of dominance, particularly in tetraploid genotypes with two or three copies of the variant, we support the conclusion that tetraploid populations or species are generally more prone to genetic load issues than diploid groups.

This raises an important question: if tetraploids tend to accumulate greater genetic load, how do some polyploid lineages achieve long-term success? Several factors not modeled here can play a critical role in the establishment of polyploid populations, counterbalancing the negative load effects we highlighted. These include higher rates of beneficial mutations, altered gene interactions, and processes such as subfunctionalization or neofunctionalization (Baduel et al., 2018; Nieto-Feliner et al., 2020). However, our simulations suggest that, all else being equal, fitness reduction due to genetic load is higher in tetraploids than in diploids under both demographic stability and bottlenecks. Future models incorporating additional genomic and ecological factors may clarify when and how polyploidy can be advantageous. From a practical conservation perspective, our results highlight that managing genetic diversity in polyploid endangered populations or stocks intended for release into the wild is as crucial as in diploid populations. For example, in *ex-situ* conservation programs of critically endangered species where available stocks are often highly inbred (Boscari et al., 2014), incorporating multiple genetically diverse lines can help increase population heterozygosity and mitigate the effects of genetic load and inbreeding depression in polyploids as well as in diploids. However, balancing genetic variability is critical, and appropriately selecting source populations in genetic management programs is key. Individuals from historically large populations besides having high genetic variability may also carry a high number of deleterious mutations (Kyriazis et al., 2021). Ideally, breeders or individuals chosen for reintroduction programs should be selected by striking a balance between increasing genetic variability and minimizing genetic load (Kyriazis et al., 2021; Maroso et al., 2023; Ralls et al., 2020). Such an approach would optimize the fitness and resilience of restored populations, aligning with best practices in conservation genetics.

Supplementary material

Supplementary material is available online at *Evolution Letters* .

Data and code availability

All simulation and plotting scripts are available at: https://github.com/VictorHerp/simulations_tetraploidVSdiploid

Author contributions

Conceptualization, G.B. and V.H.M.M.; methodology, V.H.M.M., H.E.M., and G.B.; writing original draft manuscript, V.H.M.M.; writing review and editing, V.H.M.M., G.B., H.E.M., F.G., and L.C.; funding acquisition, G.B. and L.C.

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Conflict of interest

The authors declare that they have no conflict of interest.

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