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SHORT COMMUNICATION

The evolution of the additive variance of a trait under stabilizing selection after autopolyploidization

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Abstract

Whole-genome duplication is a common mutation in eukaryotes with far-reaching phenotypic effects. The resulting morphological, physiological and fitness consequences and how they affect the survival probability of polyploid lineages are intensively studied, but little is known about the effect of genome doubling on the evolutionary potential of populations. Historically, it has been argued polyploids should be less able to adapt because gene duplication dilutes the effects of alleles, such that polyploids are less likely to evolve new adaptive gene complexes compared with diploids. In this paper, I investigate the short- and long-term consequences of genome doubling on the additive genetic variance of populations. To do so, I extended the classical models of quantitative traits under stabilizing selection to study the evolution of the additive variance of the trait under study after a shift from diploidy to tetraploidy. I found that, for realistic allele-dosage effects, polyploidization is associated with an initial decrease in adaptive potential. In the long term, the better masking of recessive deleterious mutations associated with polyploidy compensates for the initial decrease in additive variance. The time for the tetraploid populations to reach or exceed the additive variance of their diploid progenitors is generally lower than 200 generations. These results highlight that polyploidization per se has a negligible negative effect on the adaptive potential of populations in the short term, and a substantial positive effect in the long term.

KEYWORDS
additive variance, evolvability, polyploidy, quantitative genetics

1 | INTRODUCTION

Whole-genome duplication (WGD hereafter) is common in plants (Barker et al., 2016; Parisod et al., 2010; Ramsey & Schemske, 2002; Soltis et al., 2007) and animals (Mable, 2004), and is considered to have a broad range of effects on plant phenotypes and genome, and as an important driver of plant adaptation and speciation (Van de Peer et al., 2017). Nevertheless, young autopolyploid lineages, which are the natural outcomes of WGD, are expected to face high extinction rates (Levin, 2019). In the long term, and despite their supposed increased capacity to cope with more challenging environments (Bomblies, 2020; Rice et al., 2019; Van de Peer et al., 2021), polyploid lineages are generally as, and sometimes more, prone to extinction as their diploid progenitors (Arrigo & Barker, 2012; Mayrose et al., 2011, 2015; Soltis et al., 2014a, 2014b, 2014c). One historical argument to explain this lower adaptive potential of polyploids is that gene duplication dilutes the effects of alleles, such that polyploids are less likely to evolve new adaptive gene complexes compared with diploids (Stebbins, 1971).

How polyploidization can affect the short- and long-term response to selection compared with diploids has been an active topic of theoretical research. For an additive genetic architecture of a quantitative trait, Wright (1938) predicted that, for the same difference in phenotype between homozygous genotypes of diploids and autotetraploids and similar allelic frequencies in both cytotypes, the additive variance of the tetraploid population is half of the diploids. Whole-genome duplication is associated with immediate changes in the size of organs (Clo & Kolář, 2021; Otto, 2007; Porturas et al., 2019; Vamosi et al., 2007), and modification of homozygote genotype frequencies, and these changes are expected to modify the additive variance of a quantitative trait (Gallais, 2003), potentially modifying their capacity to respond to selection. On top of that, when considering the effect of dominance, it is theoretically expected that polyploidization can enhance the adaptive potential of populations in the long term, by increasing the frequency of recessive mutations (Haldane, 1927; Otto & Whitton, 2000; Ronfort, 1999), and due to the partial contribution of dominance variance to evolutionary potential (Walsh, 2005).

Nevertheless, the above-mentioned expectations suffer from several limitations. First, the above-mentioned models generally focused on a single locus, whereas most quantitative traits are known to be polygenic (Walsh & Lynch, 2018). The phenotypic consequences of polyploidization are unrealistically modelled, with models considering either no effect (Wright, 1938) or a twofold effect (Gallais, 2003) of genome doubling on phenotypic and genotypic values, whereas empirical data rather suggest an in-between effect (Clo & Kolář, 2021; Porturas et al., 2019). Another limitation is that the effect of selection of the evolution of gene frequencies after polyploidization is not modelled (Wright, 1938) or discussed verbally (Otto & Whitton, 2000), giving limited support and the conclusions of the models. It is known that the frequency of alleles, and notably the recessive ones, is expected to evolve after polyploidization, modifying the heritable variance of the quantitative traits under study. Nevertheless, little is known about the capacity of this higher masking capacity to compensate for the initial loss of diversity of polyploidy due to the masking effect.

Surprisingly, classical models of quantitative traits under stabilizing selection, which would allow consideration of most of the above-mentioned limitations, have not been incorporated into work on the short- and long-term consequences of autopolyploidization on the evolutionary potential of populations. In this paper, I modelled consequences of shifting from diploidy to autotetraploidy on the genetic variance of a quantitative trait under stabilizing selection previously at mutation–selection–drift equilibrium. The main conclusions of the model are that polyploidization is generally beneficial for the adaptive potential of populations. Even if the dilution effect described by Stebbins can lead to a more or less strong decrease in adaptive potential in the very first generations following genome doubling, this effect is only transient. The neotetraploid populations quickly equal and exceed the level of additive variance previously found in the ancestral diploid populations, due to more effective masking of recessive deleterious mutations, even in the most unfavourable conditions of dosage effects. These results suggest that, in this simplistic theoretical framework, the genetic consequences of polyploidization *per se* on the evolutionary potential of populations cannot explain the patterns of higher short- and long-term extinction rates of polyploid species compared with diploid ones.

2 | MATERIALS AND METHODS

2.1 | General assumptions

I considered the evolution of a quantitative trait Z in a population of size N, made of diploid individuals reproducing through obligate random mating. The phenotypic value z of an individual was determined by the additive action of L loci each with an infinite possible number of alleles and is given by

$$z = g + e, \tag{1}$$

where g is the genetic component of the individual's phenotype and is given by $g_{2x} = \sum_j^L g_j^M + g_j^P$, with g_j^M (respectively g_j^P) the additive allelic effect at locus j inherited from the maternal (respectively paternal) gamete in the diploid population. After polyploidization and with tetrasomic inheritance, the genetic component became $g_{4x} = \sum_j^L d(g_j^{M_1} + g_j^{M_2} + g_j^{P_1} + g_j^{P_2})$, with $g_j^{M_1}$ and $g_j^{M_2}$ (respectively $g_j^{P_1}$ and $g_j^{P_2}$) were the additive allelic effects at locus j inherited from the maternal (respectively paternal) gametes. The parameter d controlled for the dosage effect and determined the effect of polyploidization on the tetraploid genotypic values compared with diploid ones. The random environmental effect, e, was drawn from a Gaussian distribution of mean 0 and variance V_E, and was considered to be independent of the genetic components of fitness.

The trait underwent stabilizing selection around an arbitrary optimal phenotypic value, denoted Z_{opt} and being equal to zero in this manuscript. The fitness value W_z of an individual with phenotype z was thus described by the Gaussian function:

$$W_z = e^{-\delta^2/2\omega^2}, \tag{2}$$

where δ is the distance between the individual's phenotype z and the optimum trait value, and ω^2 is the width of the fitness function, and represents the strength of selection.

There was no dominance or epistasis at the phenotypic scale in this model, but both of which arose naturally at the fitness scale due to the non-linearity of the phenotype-fitness function. When considering stabilizing selection and a mean phenotype close to the optimum, the mean dominance of deleterious mutation is h ≈ 0.25 in a diploid population (Manna et al., 2011); this result remains true in polyploid populations (see Figure S1 for details).

2.2 | Simulation model

I considered a population of initially N diploid individuals, each represented by two linear chromosomes with L multi-allelic loci, coding

TABLE 1 Description of the model parameters, their abbreviations and tested values in simulations

Parameter	Abbreviation	Value(s)
Population size	N	250 or 1000
Number of loci	L	50
Haploid genomic mutation rate	U	0.001, 0.01 or 0.1
Variance of mutational effects	a^2	0.05
Dosage effect	d	0.5, 0.65 or 1
V_E of environmental effects	V_E	1
Strength of stabilizing selection	ω^2	From 1 to 399
Genomic map length	R	100

for a single quantitative trait under selection. At the beginning of each simulation, all individuals were genetically identical and are at the phenotypic optimum (all loci carry alleles with effect 0 and $Z_{OPT} = 0$). Each simulation consisted of two phases, the first being a burn-in time to allow the diploid population to attain mutation–selection–drift (M-S-D) equilibrium. The second phase consisted of shifting from diploidy to tetraploidy, coded by the fact that diploid individuals only produced unreduced gametes for one generation. The newly formed tetraploid population then only produced reduced gametes, allowing the population to stay at a tetraploid state. I then let the tetraploid population evolve until it reaches the new M-S-D equilibrium. In both phases, the population is considered to be at M-S-D equilibrium when the average fitness value calculated over the last thousand generations does not differ by more than one per cent from the mean fitness calculated over the previous thousand generations.

The life cycle can be summarized by the following successive events. First, there is a phenotype-dependent choice of the parents. Selection takes place as follows: if the ratio of the selected parent's fitness over the highest recorded fitness value in the current generation is higher than a number sampled in a uniform law comprised between 0 and 1, the individual is allowed to reproduce. Once the two parents are chosen, they each contribute a gamete, produced through uniformly recombining the parental chromosomes. The number of crossovers is sampled from a Poisson distribution with parameter R, the map length. From Haldane's mapping function, the recombination rate between two adjacent loci is $r = \frac{1}{2}\left[1 - \exp\left(\frac{-2R}{L-1}\right)\right]$. I choose parameters that ensure that $r = 0.5$, such that loci remain, at least physically, unlinked. This phase is then followed by the introduction of new mutations, the number of which is sampled from a Poisson distribution with parameter U (with $U = \mu L$, μ being the per locus mutation rate). The additive value of a new mutant allele is drawn from a normal distribution of mean 0 and variance a^2.

For each simulation, I computed the genic variance (V_g, the sum of genetic variance within loci), the genetic covariance among loci (COV_G), the total additive variance ($V_A = V_g + COV_G$), the frequency of the ancestral allele 0 and the mean population fitness. These parameters were computed at the diploid and tetraploid M-S-D equilibriums, five generations after the ploidization event (for letting genotypic frequencies reaching their random mating equilibriums, it will refer to neotetraploids hereafter), and every generation during the polyploid phase until reaching the tetraploid M-S-D equilibrium.

I tested whether the amount of genetic variance found in diploids at M-S-D equals the predicted amount expected under the stochastic house-of-cards approximation (equation 21.a from Bürger et al., 1989); this equation allows to predict the joint effects of stabilizing selection, mutation, recombination and random drift on the genetic variability of a polygenic character in a finite population. The observed variance never differed significantly from the expectation, confirming that the model ran correctly.

2.3 | Simulation parameter values

Simulations were run for several parameter sets. The values chosen for the mutation rate U range from 0.001 to 0.1, reflecting the *per*-trait haploid genomic mutation rate found in the literature (Halligan & Keightley, 2009). I used parameter set values similar to those explored in Bürger et al. (1989) and Ronce et al. (2009), with the number of freely recombining loci under selection $L = 50$, and $a^2 = 0.05$, $V_E = 1$. The strength of stabilizing selection varied from $\omega^2 = 1$ to 399 to fit empirical observations (Clo & Opedal, 2021; Gauzere et al., 2020). In this model, the mean deleterious effect of mutations $\bar{s}$ varied from 0.0125 to 0.0006 ($\bar{s} = a^2 / 2(\omega^2 + V_E)$, Martin & Lenormand, 2006). I also considered two population sizes $N = 250$ or 1000. The dosage parameter d can be equal to 0.5 ($g_{4x} = g_{2x}$), 0.65 ($g_{4x} = 1.3^* g_{2x}$, which is found in neo- and established polyploid populations on average; see Porturas et al., 2019 and Clo & Kolář, 2021 for meta-analyses) and 1 ($g_{4x} = 2^* g_{2x}$). In neopolyploid individuals, for which allelic frequencies are expected to be similar to those in their diploid progenitors, the diploid additive variance at M-S-D equilibrium is expected to be multiplied by $2d^2$ (Gallais, 2003; Wright, 1938). This means that neotetraploids should exhibit an initial decrease in additive variance for $d = 0.5$ or 0.65, and an initial increase when $d = 1$. The parameter sets are summarized in Table 1.

3 | RESULTS AND DISCUSSION

3.1 | The evolution of adaptive potential after polyploidization

On the short term, polyploidization was associated with an initial decrease in adaptive potential when $d = 0.5$ or 0.65 ($V_{A\text{-}Neo\text{-}4x} = 0.5*V_{A\text{-}2x}$ and $0.88*V_{A\text{-}2x}$, respectively, for $d = 0.5$ and 0.65, as predicted by Wright, 1938; Gallais, 2003), and an initial increase when $d = 1$ ($V_{A\text{-}Neo\text{-}4x} = 2*V_{A\text{-}2x}$, as predicted by Gallais, 2003) (Figure 1). In the long term, established tetraploid populations always exhibited a higher amount of additive variance at M-S-D equilibrium than their diploid progenitor (Figure 1). The population size (Figure S3), the strength of stabilizing (Figure S4) and the mutation rate of the trait (Figure S5) did not change the results qualitatively. For all the simulated scenarios, the additive genetic variance was mainly genic (Figure S6).

When observed, the initial decrease in additive variance in neotetraploid populations was due to the dilution of genetic effects described by Stebbins (1971). In the long term, however, the better masking of deleterious mutations, which were recessive at the fitness scale, allows them to reach higher frequency (Figure 2), compensating for the initial dilution effect, even for the most deleterious scenario of gene dosage effects ($d = 0.5$), as speculated by Otto and Whitton (2000). The higher the dosage effect, the smaller the masking advantage associated with tetraploidy, because a higher dosage effect increases the deviation from the phenotypic optimum associated with a deleterious mutation in polyploids compared with diploids.

Empirical investigations of the short- and long-term consequences of genome doubling are lacking in the literature. Martin and Husband (2012), using an artificial selection experiment, found that the realized heritability for flowering time is 0.52 and 0.31, respectively, in neo- and established autotetraploids of *Chamerion angustifolium*, whereas diploid populations of the same species exhibited a significantly higher realized heritability of 0.40 in the same experiment. If the initial increase in adaptive potential can be explained theoretically, the long-term negative effect is more surprising. It can come from other side effects associated with polyploidization, such as a shift in the mating system (Barringer, 2007; Husband et al., 2008) or a better capacity to colonize harsher environments (Rice et al., 2019), which are known to modify the evolutionary potential of populations in diploids (Clo et al., 2019; Martínez-Padilla et al., 2017; Pennington et al., 2021). No other direct comparisons among tetraploids and diploids of the same species in controlled environments are available in the literature, but the few estimates of heritability found in natural autotetraploid populations are in the range of what is found in diploid populations (Burgess et al., 2007; Clo et al., 2019; O'Neil, 1997).

More estimates are necessary to have a precise idea of how polyploidization affects evolvability in the short and long term. Artificial selection experiments seem the most straightforward way to accurately study this question, due to the contribution of dominance variance to adaptive potential. Those experiments can determine the realized heritability or evolvability of a population (containing both additive and dominance contributions). Decomposing the genetic variance of a population from sibling or pedigree analyses, and then inferring the evolvability could lead to biased estimates, notably due to the limited statistical power to infer correctly the dominance variance in such experiments (Walsh & Lynch, 2018; Wolak & Keller, 2014). Another consideration is that polyploidy is not only associated with a change in genetic variance but also frequently associated with a change in the size of morphological traits (Clo & Kolář, 2021; Porturas et al., 2019; Vamosi et al., 2007). In such a case, to accurately compare the evolutionary potential among cytotypes, one needs to standardize the heritable variance (Falconer & Mackay, 1996).

3.2 | The transient negative effect of polyploidization

As shown previously for realistic dosage effects, polyploidization came with an initial decrease in additive variance (Figure 1). Nevertheless, this initial decrease was only temporary, and the evolutionary potential of polyploid populations quickly exceeded the one of their diploid progenitors (Figure 3). The strength of stabilizing

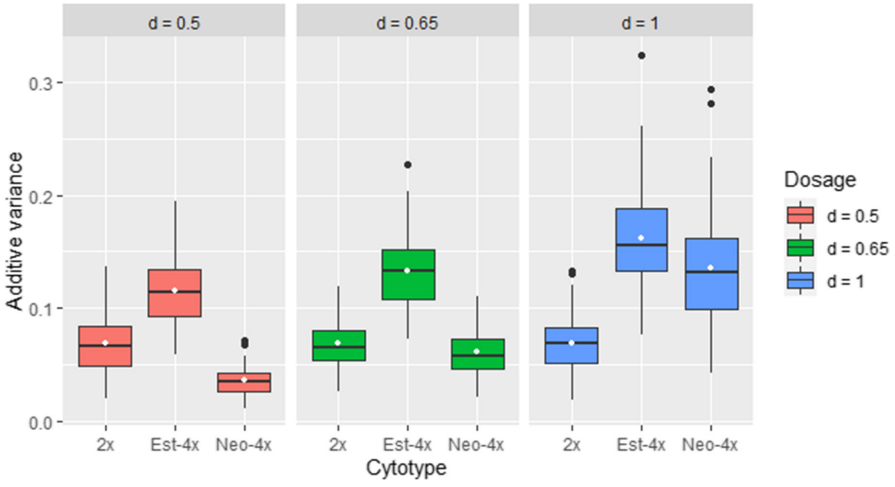

FIGURE 1 Boxplot of the evolution of the additive variance of the quantitative trait under study in neotetraploids (Neo-4x) and established tetraploids (Est-4x) compared with their diploid progenitors (2x), for different genes' dosage effects. The white points are the average values computed on $n = 100$ simulations. Other parameters are $N = 250$, $U = 0.01$ and $\omega^2 = 1$

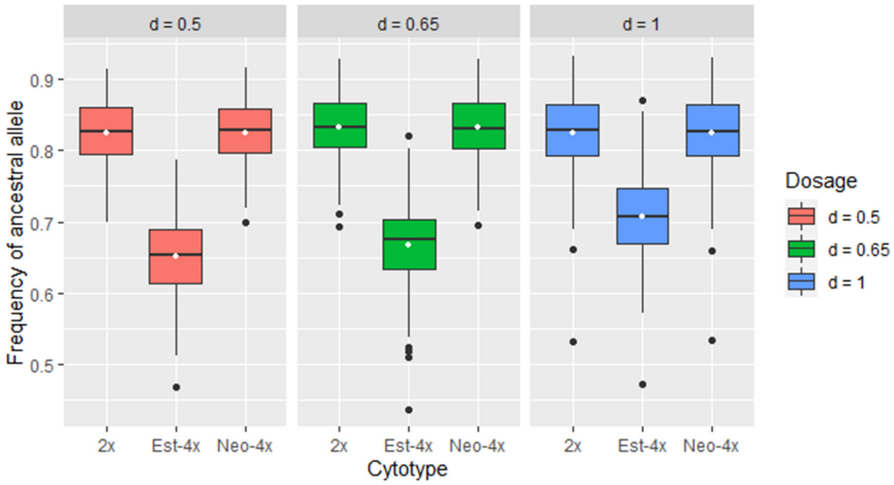

FIGURE 2 Boxplot of the evolution of the frequency of the ancestral adapted allele in neotetraploids (Neo-4x) and established tetraploids (Est-4x) compared with their diploid progenitors (2x), for different genes' dosage effects. The white points are the average values computed on $n = 100$ simulations. Other parameters are $N = 250$, $U = 0.01$ and $\omega^2 = 1$

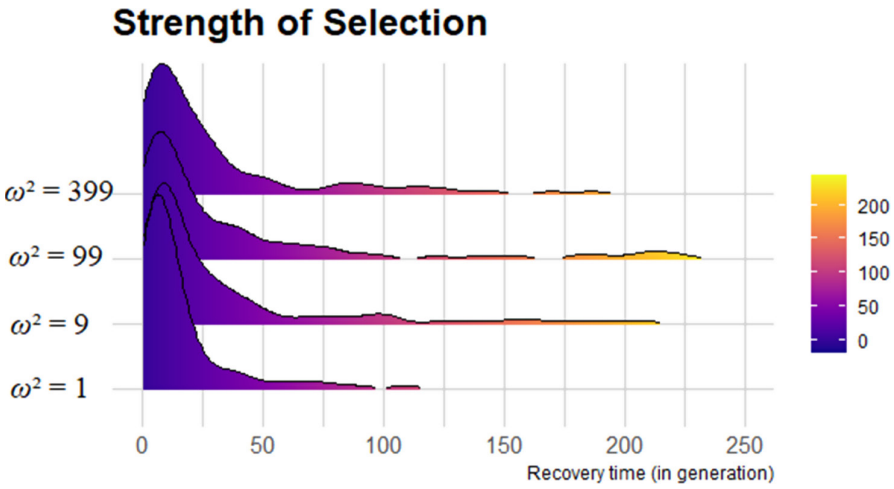

FIGURE 3 Recovery time for tetraploid populations to reach or exceed the genetic variance of the ancestral diploid population, as a function of the strength of stabilizing selection. The distribution has been computed based on $n = 100$ simulations. Other parameters are $N = 250$, $U = 0.01$ and $d = 0.65$

selection was the main factor affecting the recovery time within a dosage effect (Figure 3); the weaker the stabilizing selection, the longer the time for polyploids to surpass their diploid progenitors. This is due to the fact that with weaker stabilizing selection, deleterious mutations are in higher frequency in diploid populations, and the masking effect associated with polyploidization is then smaller. Even in the worst case, only a few hundred generations were necessary for tetraploids to surpass their diploid progenitors (Figure 3). The initial decrease in adaptive potential can nevertheless be compensated by the immediate shift in phenotypic values associated with polyploidy. In a similar theoretical framework, Oswald and Nuismer (2011) showed that neotetraploid populations can adapt more efficiently than their diploid progenitors if the populations undergo an environmental change and that this change is aligned with the phenotypic modifications found in tetraploid individuals.

This result suggests that the effect of polyploidization *per se* on the adaptive potential of populations is unlikely to explain the short- and long-term high extinction rates sometimes found in polyploid species (Arrigo & Barker, 2012; Levin, 2019; Mayrose et al., 2011, 2015), even if the current model remains simplistic, and did not consider other effects such as the modification of the reproductive system or the effect of the minority cytotype exclusion. At worst, the extinction rate of polyploid species should be negatively correlated with the age of species, as it has been found for self-fertilizing species for example (Alexander et al., 2016), but such an analysis does not exist for polyploid taxa. In such a case, how can we explain the higher extinction risks associated with polyploidy? Polyploidization arises with several costs in neopolyploid lineages, such as genomic instability, mitotic and meiotic abnormalities, reduction in fitness and minority cytotype exclusion (Clo & Kolář, 2021; Comai, 2005; Doyle & Coate, 2019; Levin, 1975; Otto, 2007; Porturas et al., 2019). In the long term, polyploidy can be associated with higher genetic load (due to recessive deleterious mutations or transposable elements), slower selection on recessive beneficial mutations and slower population growth rate (Baduel et al., 2018; Monnahan & Brandvain, 2020; Otto, 2007; Otto & Whitton, 2000), which can explain the higher extinction rate found in some phylogenetic analyses.

4 | CONCLUSIONS

In this paper, I theoretically investigated how genome doubling affects the evolvability of newly formed autotetraploid populations, and how it evolves through time. I found that, in general, the evolvability of neotetraploids decreases in the short term compared with diploid populations. This reduction is, however, only transient, and

the evolutionary potential of established tetraploid populations always exceeds the one of their diploid progenitors, due to the better masking of recessive deleterious mutations associated with polyploidization. These results suggest that the effect of polyploidization *per se* on the adaptive potential of populations is unlikely to be the major driver of the high extinction rates found in polyploid populations compared with diploid ones, in both the short and the long term.

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CONFLICT OF INTEREST

I have no conflict of interest to declare.

AUTHOR CONTRIBUTIONS

J.C. initiated the project, wrote the simulation model, wrote the first draft of the manuscript and edited it.

DATA AVAILABILITY STATEMENT

The simulation model has been deposited on GitHub (https://github.com/JosselinCLO/Stabilizing_selection_polyploidy).

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