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Genome size and phenotypic change: insights on contemporary evolution across biological groups

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Abstract

Environmental changes threaten the persistence of wild populations and can drive adaptive phenotypic changes. However, predicting the phenotypic outcome of an environmental change based on features of the species remains challenging. Genome size has been linked to variables that could influence the amount and rate of phenotypic change on contemporary time-scales. Two contrasting hypotheses have been proposed. The first suggests that larger genome sizes are associated with greater amounts and rates of phenotypic change, due to increased genetic variance and phenotypic plasticity. The second suggests the opposite based on the negative effect of genome size on both generation time and cell duplication time. We tested these two hypotheses using the Phenotypic Rates of Change Evolutionary and Ecological Database. We analysed the relationships among genome size, phenotypic trait variability, mean trait change, and generation time. We found no support for the either of the tested hypotheses. Overall, the genome size explained less than 2% of the total variance in the amount of phenotypic change. In plants only, we found a consistent and negative effect of genome size on phenotypic change. However, we found no relationship between genome size and generation time, and we found no relationship between genome size and phenotypic variance. However, the consistent and relevant effect of phenotypic variance on the mean-scaled phenotypic change is worth highlighting from a conservation perspective because assessing the phenotypic variability of traits related to the niche breadth in different dimensions could inform us about the vulnerability of natural populations facing unexpected environmental changes.

Keywords phenotypic change, contemporary evolution, genome size, C-value, darwins, haldanes

Introduction

Phenotypes interface with the environment and therefore are direct determinants of fitness (Endler, 1986; Hendry et al., 2017; Schluter, 2000). Because selective pressures experienced by a population vary in strength and direction along time (Grant & Grant, 2002; Siepielski et al., 2009, 2013), the ability of populations to respond to those changes becomes key for their persistence and success. We know that traits in natural populations frequently change on contemporary time scales (e.g., a few years to a few centuries) in response to environmental changes (Hendry & Kinnison, 1999; Kinnison & Hendry, 2001; Sanderson et al., 2021). The phenotypic microevolutionary mechanisms of quantitative traits and phenotypic plasticity are well known (Falconer, 1989; Stamp & Hadfield, 2020). However, we know little about the species features underpinning the phenotypic change. Here, we aim to assess the relationship between the genome size of species and their rates and amount of phenotypic change on contemporary time scales of populations facing an environmental change.

The genome size (i.e., the total amount of DNA in the unreplicated haploid nucleus) is a biological property of organisms that can affect a broad range of traits and biological processes, some of which could affect phenotypic change. Even when the variability in genome size is largely explained by noncoding DNA (C-value paradox; Gregory, 2001), noncoding DNA has genetic and epigenetic functions (Shanmugam et al., 2017; Zuckerkandl, 2002), as well as nongenetic effects that can affect or set conditions for the further evolution of organisms in different directions (Gregory, 2005). Some evidence shows that genome duplication (even with the same genetic information, neoautotetraploid vs. diploid) increases the early response to selection (Martin & Husband, 2012). Conversely, some evidence exists that plants and some vertebrate species with large genomes are more prone to extinction (Knight et al., 2005; Vinogradov, 2003, 2004) which might suggest that species with larger genome sizes are less likely to adapt.

In the context of rapid environmental changes, phenotypic plasticity and evolutionary responses based on existing genetic variance in a population can play a decisive, adaptive role (Barrett

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& Schluter, 2008; Lacy, 1997; Lande & Shannon, 1996; Stamp & Hadfield, 2020). Genome size and mutation rate per locus are positively correlated (Abrahamson et al., 1973; Sparrow et al., 1961), suggesting that higher genetic variance in larger genomes might be expected. Also, because of the regulatory functions of the noncoding DNA (Shanmugam et al., 2017; Zuckerkandl, 2002), we expect more phenotypic plasticity in large genome size species. Therefore, we hypothesize that because of genome size effects on genetic variance and phenotypic plasticity, genome size will be positively correlated with phenotypic rates (and amount) of trait change and the level of phenotypic variance (hypothesis 1, Box 1). We predict that: (1.i) the phenotypic variance (pooled standard deviation—hereafter denoted as *pSD*) will be positively correlated with amounts and rates of mean-scaled phenotypic change (darwins' numerators and darwins); (1.ii) the genome size (C-value) will be positively correlated with the phenotypic variance of the traits; (1.iii) the genome size (C-value) will be positively correlated with the amount and rate of phenotypic change (darwins' numerators and darwins); and (1.iv) because haldanes rate of change is *SD* scaled, the first prediction is not appropriate when amount and rates of change are assessed in haldanes' numerator and haldanes (variance-scaled phenotypic change), respectively; and the third predictions will not be true (Box 1).

However, there is evidence of regulatory functions for only a small fraction of the noncoding DNA (Palazzo & Gregory, 2014). If most of the noncoding DNA is indeed "junk" and not regulating gene expression at all, then hypothesis 1 would not be supported. However, this is not the only mechanism through which genome size could affect phenotypic rates of change. Genome size can affect the cell nucleus size, which affects cell size, and therefore cell division and development time/rates in plants and animals such as invertebrates, amphibians, and fishes, but not amniotes (Bennett, 1987; Gregory, 2005). Therefore, patterns of phenotypic change could be linked to generation time depending on the type of phenotypic change. If the change is gradual and continuous through time, the longer the generation time, the slower the rate of change (in darwins) and the less accumulated change in both darwins' and haldanes' numerator. If the change is abrupt and bounded, as previously found (Gorné & Díaz, 2019; Kinnison & Hendry, 2001), the effect of generation time will decrease with more time elapsed but with the same trend as in the previous case. Therefore, we will expect a negative effect of the genome size on phenotypic rates of change in some groups but not in others due to its effect on generation time (Hypothesis 2, Box 1). We predict that: (2.i) the generation time will be positively correlated with genome size (C-value) in plants, insects, and fishes, but not in birds and mammals; (2.ii) the genome size (C-value) will be negatively correlated with rates of mean-scaled phenotypic change (darwins) and amount of phenotypic change (darwins' and haldanes' numerator) in plants, insects, and fishes, but not in birds and mammals; and (2.iii) because haldanes rate of change is per generation, the prediction 2.ii is not appropriate when rates of change are assessed in haldanes (hypothesis 2, Box 1).

M&M

The database

We performed our analyses using the Phenotypic Rates of Change Evolutionary and Ecological Database (PROCEED; Gorné et al., 2025). Briefly, PROCEED includes studies that measure intraspecific change in quantitative (continuous or count) traits and report the time elapsed from the onset of environmental novelty or refer to a historical or biological event reported in other sources (e.g., a mine opening; a well-documented biological invasion). The elapsed times included in this database span from a single generation or year to a few hundred generations (<300 generations) or years (<300 years). The included studies followed a single population through time (allochronic study) or compared two or more populations diverged from an original source population due to an environmental change of known age (synchronic study). We took advantage of a comprehensive database to assess whether any, general patterns between genome size and phenotypic changes at the intraspecific level (microevolution and phenotypic plasticity) can be detected. The database includes systems (i.e., the populations, with their traits, of a given species experiencing a specific driver of change) in natural or experimental conditions, but in which reproduction was not directly manipulated. The experimental systems in the database manipulated the environment of the populations being studied, but did not deliberately select for specific traits (e.g., an herbivory exclusion experiment, where the type and load of herbivory were manipulated and the plant populations evolved under this new condition). PROCEED does not include systems, where the phenotypic change is due to interspecific hybridization, polyploidy, or other chromosomal mutations.

We used an updated version (v.6.2) of PROCEED (Gorné et al., 2025). The previous version (v.5) was published by Sanderson et al. (2021, 2023). This newer version has been updated by adding the rates of change in plant populations surveyed in the review by Gorné & Díaz (2019) that were not previously in PROCEED. We include 1,838 new rates belonging to 138 new systems of 92 new species from 110 new articles. This new, curated version totals 9,222 rates belonging to 1,798 systems of 425 species from 322 studies. PROCEED is available on its website (https://proceeddatabase.weebly.com/) as well as in the Borealis stable repository (https://doi.org/10.5683/SP3/NXSL3Q).

We also added information about genome size, the C-values for each species calculated as the amount of DNA, in picograms, in a haplontic nucleus. The main sources for C-values are the Plant DNA C-values Database (Leitch et al., 2019; https://cvalues.science.kew.org/; Pellicer & Leitch, 2020) and the Animal Genome Size Database (Gregory, 2021; http://www.genomesize.com). When C-values were not available in these databases, we searched Google Scholar for this information. For plant species in the Plant DNA C-values Database, when more than one value was available for a specific species, we chose the "prime genome size estimate" (i.e., the most consistent value obtained using best-practice methods, as defined by Bennett & Smith, 1976). However, if the "prime estimate" was not related to ploidy and the chromosome number, we used the estimates with these data. For animal species in the Animal Genome Size Database, when more than one value was available for a specific species, if there was no difference in ploidy level or chromosome number among records or measuring method, the C-values were averaged. However, records using Feulgen densitometry, flow cytometry, 4′,6-diamidino-2-phenylindole (DAPI) citometry, Fulgen microdensitometry, Gallocyanin-chrome alum densitometry, or Fulgen image analysis densitometry methods were preferred over other methods. When available, records using these methods were averaged. Because birds have small variability in genome size (Andrews, 2009), if the genome size was not available for a given species but there was information avail-

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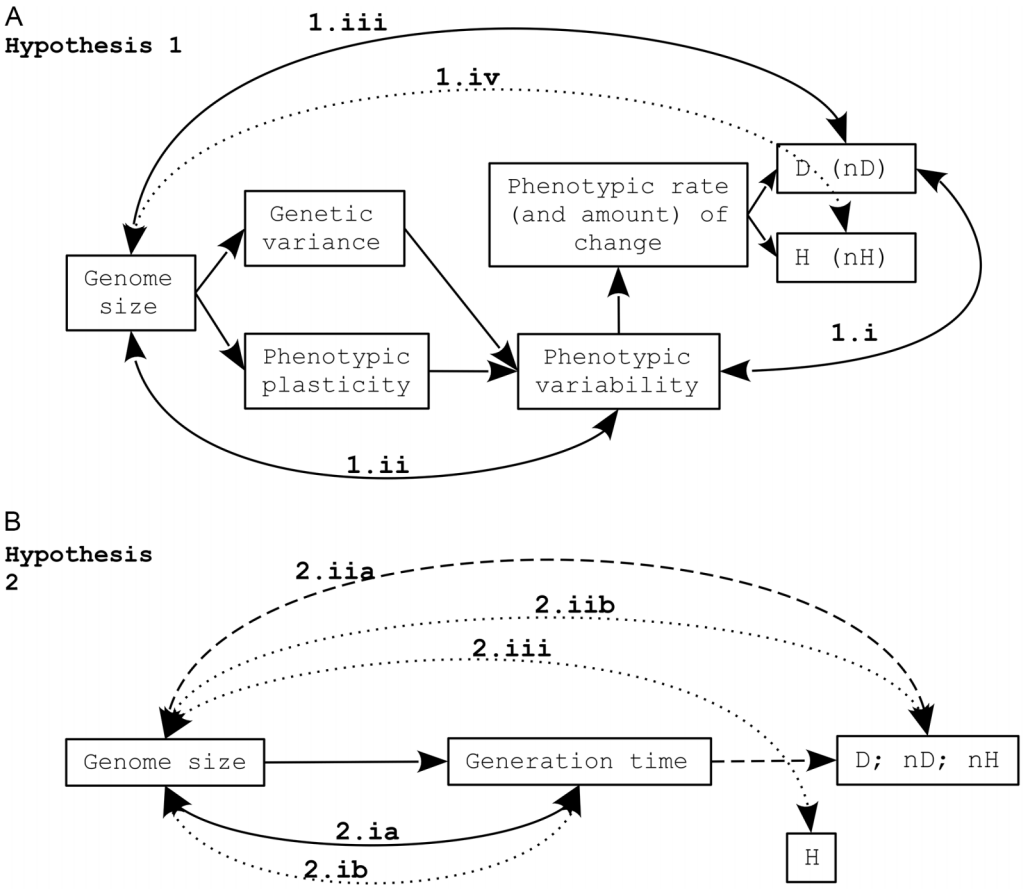

C **Summary of results**

Prediction	Plant	Arthropod	Fish	Bird	Mammal
1.i	yes	yes	yes	yes	yes
1.ii	no	no	no	no	no
1.iii	opposite	no	no	no	no
1.iv	no (<0)	yes	yes (H); no (nH<0)	yes	yes
2.ia	no	no	no	—	—
2.ib	—	—	—	yes	yes
2.iia	yes	no	yes (nH); no (nD, D)	—	—
2.iib	—	—	—	yes	yes
2.iii	no (<0)	yes	yes	yes	yes

Box 1. Hypotheses, predictions, and summary of the main results. (A) Hypothesis 1 and its predictions. (B) Hypothesis 2 and its predictions. (C) Summary of the results. In the hypotheses and predictions, the straight arrows represent the causal structure of the hypotheses; the curved double-headed arrows represent the predictions we tested; continuous lines represent positive relations between linked variables; dashed lines represent negative relations between the variables; and dotted lines represent no relation between the linked variables. The predictions are numbered (i–iv). (1.i) phenotypic variance (*pSD*) will be positively correlated with amounts and rates of phenotypic change (*nD* and *D*); (1.ii) the genome size (C-value) will be positively correlated to the phenotypic variance of the traits (*pSD*); (1.iii) the genome size (C-value) will be positively correlated to the amount and rate of phenotypic change (*nD* and *D*); and (1.iv) the third prediction will not be true when measuring the phenotypic change in haldanes (*nH* and *H*). (2.i) the generation time will be positively correlated with the genome size (C-value) in plants, insects, and fishes, but not in birds and mammals; (2.ii) the genome size (C-value) will be negatively correlated with the rate (*D*) and amount of phenotypic change (*nD* and *nH*) in plants, insects, and fishes, but not in birds and mammals; and (2.iii) no correlation is expected between genome size (C-value) and *H*.

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able for congeneric species, we used the C-value of the closest phylogenetically related species as a proxy for that species. For both plants and animals, when the genome size was estimated from several subspecies, the value for each subspecies was averaged first and then averaged among subspecies. In general, but especially in plants, when a species has more than one genome size due to differences in ploidy level or chromosome number, the system was revisited looking for information about particular ploidy levels of the involved populations for each case. For two plant species (*Hypericum perforatum* and *Senecio inaequidens*), that information was unavailable, and the available C-values were averaged. The estimated C-value is more variable depending on the methodology than between populations of the same species (Doležel & Greilhuber, 2010). This is the main reason for having preferred methods and averaging among them. Also, this justifies the use of a single C-value for each species, even when the analyses of phenotypic change included multiple observations per species.

Data filtering

We used a subset of PROCEED that had all the necessary information to perform the analyses. All included data had the C-value (genome size), the SD of the traits and the generation time, as well as all the variables to be tested and included as potentially relevant predictors in the model (Table 1). Computing haldanes related effect size (ES) requires a pooled SD different from zero and therefore observations with pooled SD equal to zero were removed. Computing darwins related ES requires means not equal to zero and therefore observations with a data point mean equal to zero were also removed. Additionally, darwins must be computed only for data with a ratio scale (i.e., a constant interval scale with a true zero) and if the mean of the raw scale is known or can be computed (e.g., no transformed scales like sin^{-1} or other transformations). Lastly, we restricted our time frame of change to 300 years or less. As we only had 6, 22, and 27 data points, belonging to one or two species, respectively, for the "Amphibian," "Mollusc," and "Reptile" groups, we excluded them from the analyses. A SD equal to zero for quantitative traits is unexpected and likely a sign of insufficient sampling size. Therefore, we also eliminated cases where one of the sampling SDs was equal to zero. A general positive correlation is expected between the mean of sample 1 and sample 2 because both samples will have approximately the same order of magnitude. An observation in which the difference between sample 1 mean and sample 2 mean spanned more than 2 orders of magnitude was removed from the dataset. The median values of the coefficient of variation (CV) for both sample 1 and sample 2 are close to 10% (11.22% and 10.76%, respectively). Observations with CVs smaller than 0.1% or larger than 1,000% were also removed ($N = 4$). The resulting dataset used in our analyses has 6,064 rates from 1,392 systems belonging to 140 species. The maximum elapsed time in this dataset is 250 years, but 75% of the observations have elapsed times shorter than 72 years. The filtered dataset used in this article can be found in the supplementary material (filteredPROCEEDv6.2_gorne.csv).

Response variables

The response variables analysed here were the standard deviation (*pSD*) of the traits and two microevolutionary rates with their numerators, haldanes (H) and darwins (D). To account for the different sample sizes among studies (spanning from 5 to 14,296) and weight them accordingly, we applied meta-analytic tools as proposed by Gorné & Díaz (2017, 2019). First, the *pSD*-related ES ($\ln\hat{\sigma}$) and its sampling variance were calculated according to Nakagawa et al. (2015) as follows:

$$ln\hat{\sigma} = \left(\frac{(n_1-1)\cdot SD_1{}^2 + (n_2-1)\cdot SD_2{}^2}{n_1+n_2-2}\right)^{\frac{1}{2}} + \frac{1}{2\cdot(n_1+n_2-1)}, \tag{1a}$$

$$V_{lnS} = \frac{1}{2\cdot(n_1+n_2-1)}, \tag{1b}$$

where n_1 and n_2 are the sample sizes of samples 1 and 2, respectively, SD_1 and SD_2 are the standard deviations of samples 1 and 2, respectively.

Haldane's numerator-related ES was the standardized mean difference Hedges *g* (Gorné & Díaz, 2017, 2019). This ES and its sampling variance were computed according to standard meta-analytic procedures (Cooper et al., 2009; DeCoster, 2004; Hedges, 1982) as follows:

$$g = \frac{\overline{x_2}-\overline{x_1}}{pSD}\cdot\left(1-\frac{3}{4\cdot(n_1+n_2-2)-1}\right), \tag{2a}$$

$$S_g^2 = \frac{n_1+n_2}{n_1\cdot n_2} + \frac{\left(\frac{\overline{x_2}-\overline{x_1}}{pSD}\right)^2}{2\cdot(n_1+n_2-2)}, \tag{2b}$$

where $\overline{x_1}$ and $\overline{x_2}$ are the means of samples 1 and 2, respectively, and *pSD* is the pooled standard deviation. Additionally, because we are not interested in the direction of the change but only in the amount of change, we used the absolute values of *g*. This produces a folded distribution, highly asymmetric to the right. Therefore, we worked with the $\log_{10}$-transformed variable and its sampling variance according to the propagation errors theory (Bevington & Robinson, 2003; Gorné & Díaz, 2017):

$$LG = log_{10}(|g|), \tag{2c}$$

$$S_{LG}^2 = \frac{S_g^2}{g^2\cdot ln^2(10)}. \tag{2d}$$

Similarly, we computed the ES related to haldanes rate of change:

$$h = \frac{|g|}{t_g}, \tag{3a}$$

$$S_h^2 = \frac{S_g^2}{t_g^2}, \tag{3b}$$

where t_g is the elapsed time in generations.

$$LR = log_{10}(h), \tag{3c}$$

$$S_{LR}^2 = \frac{S_h^2}{h^2\cdot ln^2(10)}. \tag{3d}$$

Darwins numerator-related ES is the Response Ratio. This ES and its sampling variance were calculated according to Hedges et al. (1999) as follows:

$$RR = ln\left(\frac{\overline{x_2}}{\overline{x_1}}\right), \tag{4a}$$

where *ln* is the natural logarithm.

$$S_{RR}^2 = \frac{SD_2^2}{n_2\cdot\overline{x_2}^2} + \frac{SD_1^2}{n_1\cdot\overline{x_1}^2}. \tag{4b}$$

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Table 1. Variables and interactions included in the analyses. Some of the variables are related to specific predictions (in bold), others are being included to control for variability because they can have effects on the rates and amounts of phenotypic change, but they are not related to our hypotheses and predictions. See Box 1 for predictions.

Variables	Short definition	Potential predictor of	Rationale
C-value	Total amount (pg) of DNA in the unreplicated haploid nucleus.	*pSD*, *D*, *nD*, *H*, *nH*	Predictions 1.i, 1.iii, 1.iv, 2.ii, and 2.iii
pSD	Pooled standard deviation. Equation 1.	*D*, *nD*	Prediction 1.ii
Years	Elapsed time between sample 1 and sample 2.	*D*, *nD*, *H*, *nH*	Depending on the dominant processes of phenotypic evolution underlying the observed amounts and rates of change, the elapsed time will have different effects (Hunt, 2012).
Design	Studies following a population through time (allochronic) or studies looking at two contemporary populations with a common origin and known time of divergence (synchronic).	*D*, *nD*, *H*, *nH*	Synchronic studies measure divergence between populations (and both populations can change in opposite directions), so rates are expected to be larger for these kinds of studies than for allochronic studies (Hendry & Kinnison, 1999).
Genphen	This term distinguishes studies measuring individuals that developed in nature (phenotypic) from those based on measurements of individuals bred in common-garden conditions or applying other quantitative-genetic methods (genetic).	*pSD*, *D*, *nD*, *H*, *nH*	Phenotypic studies might display more variance because of environmental heterogeneity. Phenotypic studies might display more change because phenotypic plasticity can be involved (Hendry & Kinnison, 1999).
Trait type	Types of traits based on Kingsolver & Diamond (2011), with some additional categories (Gorné et al., 2025).	*pSD*, *D*, *nD*, *H*, *nH*	It is known that different trait types display different amounts of phenotypic and genetic variances (Houle, 1992) and therefore, differences in evolvability.
Disturbance	Classification of the environmental change (or driver/disturbance) driving the phenotypic change, based on the categories used by Hendry et al. (2008) and Palkovacs et al. (2012).	*D*, *nD*, *H*, *nH*	Some disturbances lead to larger amounts of change (e.g., Sanderson et al., 2021) because different human influences will have different selection differentials.
Experimental	States if the environmental change is imposed by experimental conditions.	*D*, *nD*, *H*, *nH*	Environmental changes due to experimental settings could be more contrasting (e.g., involving stronger selection pressures) than in nonexperimental systems.
Out-in	This term states if the system involves population(s) occurring outside or inside its native range.	*D*, *nD*, *H*, *nH*	Selection pressures in nonnative distribution areas could be different than the selection pressures in native areas.
Environmental change	This term distinguishes between systems where the environmental change has been occurring through time and the phenotypic change was measured in a temporal window within this process (but not from the starting point) from systems where the environmental change has a defined starting point and phenotypic change was measured from this point.	*D*, *nD*, *H*, *nH*	Environmental change is expected to affect the amount and rate of phenotypic change because the response to an environmental change could be abrupt, with most of the change occurring in a short initial period (Kinnison & Hendry, 2001; Gorné & Díaz, 2019).

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Table 1. Continued

Variables	Short definition	Potential predictor of	Rationale
Data type	Classification factor according to the dimensionality of the measured trait.	*pSD*, *D*, *nD*, *H*, *nH*	The dimensionality of the trait is expected to affects the variance (e.g., for the same sample $\sigma_{\text{(body length)}} < \sigma_{\text{(body mass)}}$). In the same way, it is expected to affect darwins ($nD_{\text{(body mass)}} \sim 3 \cdot nD_{\text{(body length)}}$). Dimensionality is not expected to affect haldanes, but this assumption was tested.
Transformed data	Classification factor accounting for if the trait measurement is expressed in the raw scale or has been transformed somehow before computing means and standard deviations.	*pSD*, *D*, *nD*, *H*, *nH*	It is expected that a transformation of the scale affects the variance (e.g., $\sigma_{\ln(x)} = \sigma_x/x$). In the same way, it is expected to affect darwins. It is not expected to affect haldanes, but this assumption was tested.
Taxa	A coarse classification of species according to the colloquial use of the terms, not necessarily a clade (monophyletic group).	*pSD*, *D*, *nD*, *H*, *nH*	Different biological groups may experience different developmental and evolutionary constraints leading to differences in variability of traits, as well as rates and amounts of change.
Taxa*C-value	Interaction	*pSD*, *D*, *nD*, *H*, *nH*	Hypothesis 2 predicts different effects of genome size on the phenotypic change among taxa. Furthermore, it is important to test this interaction, for both hypotheses, to avoid general conclusions biased by a specific group, better represented, or with a stronger correlation between phenotypic change and genome size.
Trait type*C-value	Interaction	*pSD*, *D*, *nD*, *H*, *nH*	There are no specific hypotheses and predictions depending on the trait type, however, it is important to avoid general conclusions biased by a specific trait type, better represented trait types, or with stronger correlation.
Years*C-value	Interaction	*D*, *nD*, *H*, *nH*	If a larger genome size provides more genetic variance in the population (hypothesis 1), this could affect the slope of the amount or rate of change over time (always depending on the predominant evolutionary process). This would not be expected for haldanes but it was tested.
Years*Disturbance		*D*, *nD*, *H*, *nH*	Suppose different disturbances produce different patterns of phenotypic change (e.g., abrupt change vs. gradual change). In that case, the effect of time will be different among disturbances and so, the effect of the genome size on the amount and rate of change mediated by the generation time will differ as well (prediction 2.ii).
Years*Env. change	Interaction	*D*, *nD*, *H*, *nH*	Depending on the pattern of phenotypic change (e.g., abrupt change vs. gradual change), including or not including the initial response to the environmental change might affect the relation between time and phenotypic change.

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Table 1. Continued

Variables	Short definition	Potential predictor of	Rationale
Years*Genphen	Interaction	*D*, *nD*, *H*, *nH*	Phenotypic plastic responses to environmental changes are expected to be faster than genetic changes in the population and so, the effect of time on the amount and rates of change is expected to be different between these two groups.
Years*Design	Interaction	*D*, *nD*, *H*, *nH*	Because a synchronic design is measuring divergence, it is expected that larger amounts of change accumulate over time than allochronic designs (always depending on the predominant evolutionary process).

Similarly to haldanes, we are only interested in the amount of the change and not in the direction. So, we worked with the $\log_{10}$-transformed absolute value of *RR*:

$$LnD = log_{10}\left(|RR|\right). \tag{4c}$$

$$S^2_{LnD} = \frac{S^2_{RR}}{RR^2 \cdot ln^2\left(10\right)}. \tag{4d}$$

For those traits expressed in natural logarithm scale, we followed the approximation proposed by Lynch (1990), i.e.:

$$ln\left(\overline{x_i}\right) \approx \overline{ln\left(x_i\right)} + 0.5 \cdot \sigma^2{}_{ln(x_i)}, \tag{5}$$

where $\sigma^2{}_{ln(x_i)}$ is the variance of the natural logarithm of the trait.

Finally, the darwins-related ES and its sampling variance were computed as follows:

$$dw = \frac{|RR|}{t_y \cdot 10^{-6}}, \tag{6a}$$

where t_y is the elapsed time in years.

$$S^2_{dw} = \frac{S^2_{RR}}{\left(t_y \cdot 10^{-6}\right)^2}, \tag{6b}$$

$$Ldw = log_{10}\left(dw\right), \tag{6c}$$

$$S^2_{Ldw} = \frac{S^2_{dw}}{dw^2 \cdot ln^2\left(10\right)}. \tag{6d}$$

In summary, to analyse trait variance we used the $\ln\hat{\sigma}$ effect size (Equation 1a and 1b); to analyse amount of change as haldanes numerator we used the LG effect size (Equation 2c and 2d); to analyse the rate of change as haldanes we used the LR effect size (Equation 3c and 3d); to analyse amount of change as darwins numerator we used the LnD effect size (Equation 4c and 4d); and to analyse the rate of change as darwins we used the Ldw effect size (Equation 6c and 6d).

The predictors C-value, *pSD*, and elapsed time (both in years and in generations) showed a highly asymmetric distribution to the right. Therefore, these variables were log_{10} transformed to meet assumptions of normality.

Analyses

The relevant statistical hypotheses in the present work are related to the relationship (direction and magnitude of the slope) between the C-value (or *pSD*) and the response variables. Other factors expected to affect the response variables, but are not involved in our predictions, were included as "control variables" (Table 1). The effects of these control variables have been previously discussed, and they were revisited by Sanderson et al. (2021). To test our predictions, we analysed the relationship (direction and magnitude of the slope) between the C-value and the following response variables: (i) the *pSD*—$ln\hat{\sigma}$—, (ii) the rate of change darwins—*Ldw*—, (iii) the amount of change in darwins' numerator—*LnD*—, (iv) the rate of change haldanes—*LR*—, and (v) the amount of change in haldanes' numerator—*LG*—. We also analysed the relationship (direction and magnitude of the slope) between *pSD* and the darwins-related variables *Ldw*, *LnD*.

To estimate the slopes, we performed meta-analyses via multilevel linear (mixed) models using the "*metafor*" R-package (Viechtbauer, 2010). Cinar et al. (2022) have shown that models that decompose the random variance of the sampling design into nonphylogenetic and phylogenetic components provide less biased estimates. We applied this approach as follows: the species identity was included as a random factor nested within genus, the genus nested within families, and so on. We used the standard taxonomic levels: phylum, class, order, family, genus, and species. Additionally, we built a number of factors allowing us to create a dichotomous tree-like classification from the root up to the order level. Then, we used a selection-model procedure to retain the taxonomic levels of these nested factors that best fit the data. In this way, the lack of independence of the samples due to their relatedness is accounted for. Finally, the study system was added as an independent random factor that controls for the lack of independence of several measurements of a given sample, accounting for the nonphylogenetic component. After selecting the random structure of the models, in the analyses of each response variable, we selected a "base model" for the fixed-effect factors that includes only control variables (Table 1) by performing a "backward" selection procedure using the Akaike Information Criterion. Once the "base model" was selected, we added the C-value (or *pSD*) and their interactions (Table 1) to test our predictions (Box 1). Based on these final models, we computed the contrasts related to the predictions.

To assess the interaction between C-value and trait type (or *pSD* and trait type), we analysed only three trait types (otherLH—life-

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history traits different from phenology or growth, othermorphology, and size) because only these three categories are well represented among the different "*taxa*" groups (e.g., PROCEED only has behavioural traits for fish and mammals, and phenological traits are only available for bird and plant groups).

To calculate the deviance explained by the different factors of interest (genome size and phenotypic variance), we computed the change in a pseudo R^2 (McFadden, 1974) between the base model and the model including the hypothesis factors. Similarly, we computed the pseudo R^2 of the base model by comparing it with a model that holds only the random factors and an intercept as the fixed part of the model.

To look at the relationship between C-value and generation time (Box 1, prediction 2.i), we used a linear mixed model, at the species level, using the "*nlme*" R-package (Pinheiro & Bates, 2000; Pinheiro et al., 2022). Generation time was the response variable. C-value, "*taxa*," and their interaction were the fixed-effect factors. The nested taxonomic structure was the random-effect part of the model. When multiple systems were studied for the same species and different generation times were computed for those populations, we averaged the generation time. For each species, only one pair of C-value and generation time was used. All analyses were performed in the R environment version 4.2.2 (R core team, 2022).

Results

Darwins and phenotypic variance (prediction 1.i)

The inclusion of *pSD* improved the base model for both darwins-numerator and darwins (Table S1). The interactions between *pSD* with "*taxa*," the trait type, and the elapsed time also improved the model (Table S1).

The mean-scaled amount and the rate of change were positively correlated to the phenotypic variance (*pSD*) of the traits, for all "*taxa*" and trait types. Also, there was an interaction between the elapsed time and phenotypic variance: the longer the elapsed time was, the larger (more positive slope) the effect of phenotypic variance on the mean-scaled amount and rate of change was. In the comparison among "*taxa*," we can see two groups: (i) plants and arthropods with a shallower slope, and (ii) fishes, birds, and mammals with a steeper one. There were also differences in the amplitude of the confidence interval among "*taxa*." They were narrow for plants and fishes, wide for birds, and intermediate for arthropods and mammals. Among trait types, life-history traits showed the steepest slope in the relationship between mean-scaled phenotypic change and phenotypic variance (Figure 1; Table S2, Figure S1).

The addition of the phenotypic variability, and its interaction with the elapsed time and taxa, contributed an additional 3.58% to the explained deviance by the base model (53.46% for *LnD* and 68.93% for *Ldw*; Table S3). The interaction between phenotypic variability and trait type added 11.2% to the explained deviance of the models.

Phenotypic variance and genome size (prediction 1.ii)

The addition of C-value and its interactions with "*taxa*" did not improve the base model for Ln$\hat{\sigma}$ (Table S1). The slope of the relationship between the phenotypic variance and C-value was consistently neutral for all "*taxa*" (Figure 2). In accordance with this result, the contribution of genome size to the explained deviance of the phenotypic variability was almost zero (<0.001%) over a base model accounting for 36.37% of the deviance (Table S3). Conversely, there was an effect of genome size on the phenotypic variance depending on the trait type with a negative effect for morphological traits (other than size) and a positive one for life-history traits (Figure 2; Table S2). This interaction explained 6.55% of the deviance.

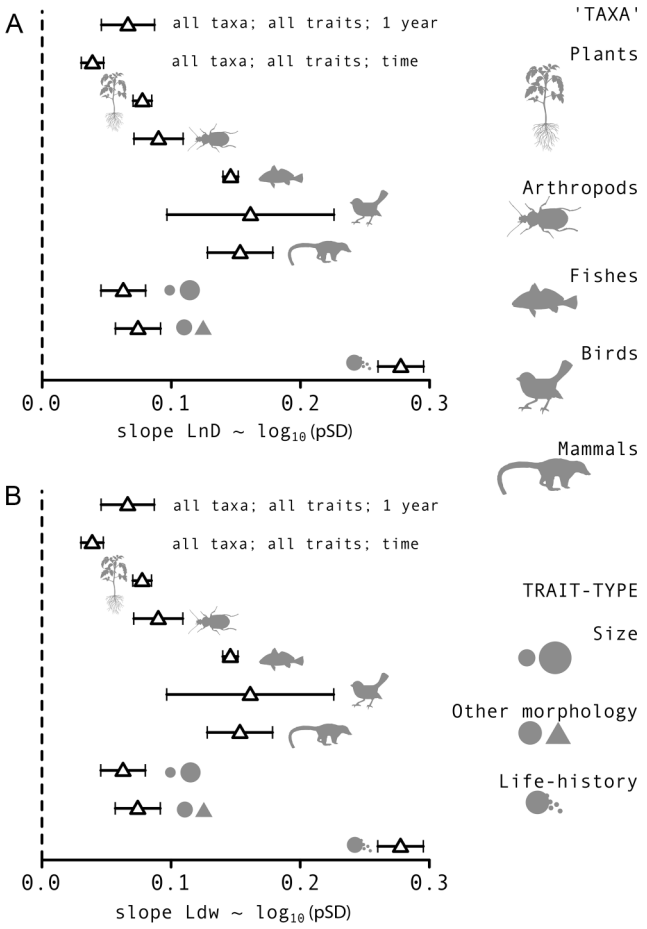

Figure 1. Slopes (and 95% confidence intervals) associated with prediction 1.i (see Box 1). We computed slopes separately by "*taxa*" (top section in each panel) and then by trait types (bottom section in each panel). (a) Slopes of the amount of change (darwins-numerator effect size, *LnD*) as function of the phenotypic variance. (b) Slopes of the rate of change (darwins effect size, *Ldw*) as function of the phenotypic variance. Trait type: "size" is the total body mass, or an overall aspect/measurement of body size as a whole; "othermorphology" are all remaining morphological traits; "otherLH" life-history traits different form phenology or growth.

Darwins, genome size, and generation time (predictions 1.iii, 2.i, and 2.ii)

The addition of C-value and its interactions with the elapsed time and the trait type improved the base model for the mean-scaled rates and amount of phenotypic change (Table S1). However, the addition of the interaction between C-value and "*taxa*" did not

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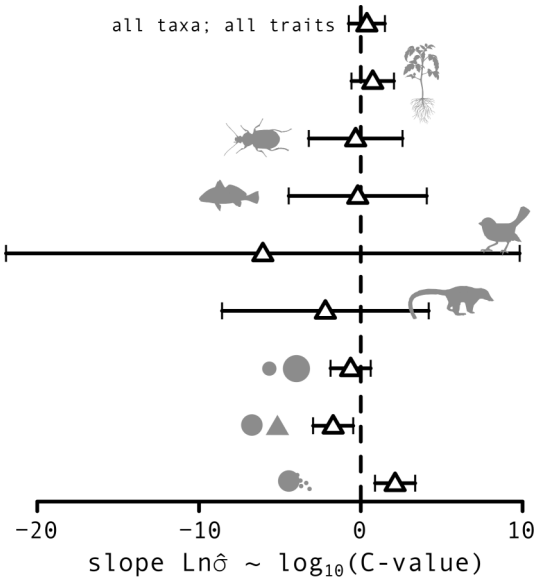

Figure 2. Slopes (and 95% confidence intervals) associated with prediction 1.ii (see Box 1). We computed slopes separately by "*taxa*" (top section) and then by trait types (bottom section). Relationship between phenotypic variance (ln$\hat{\sigma}$) and genome size (C-value). Iconography is as in Figure 1.

Table 2. Relationship (slope), for each "*taxa*" group, between the log$_{10}$(C-value) and log$_{10}$(generation time).

"*taxa*"	*n*	Slope (Standard error)	*p*-values
Plant	55	0.1988 (0.1550)	.200
Arthropod	8	0.1486 (0.3603)	.680
Fish	23	0.8075 (0.6506)	.215
Bird	13	0.3937 (1.3278)	.767
Mammal	41	−0.5021 (0.5221)	.336

contribute to the production of a better model (Table S1). The slope of the mean-scaled amount (LnD) and rate (Ldw) of phenotypic change along the genome size (C-value) was overall negative at an early stage (1 year), but with a positive interaction with the elapsed time (Figure 3; Table S2). No significant differences were found among "*taxa*," or among trait types. However, in plants and for life-history traits, negative slopes different from zero were observed (Figure 3; Table S2).

The contribution of the genome size in explaining the amount and rate of mean-scaled phenotypic change was small. The addition of genome size and the interaction with the elapsed time contributed 0.025% to the explained deviance (Table S3). The interaction between genome size and trait type added 1.75%. Finally, we found no relationship between genome size and generation time in any "*taxa*" (Table 2).

Haldanes and genome size (predictions 1.iv, 2.ii, and 2.iii)

The base models fitted to the variance-scaled amount (LG) and rate (LR) of phenotypic change were improved by adding the

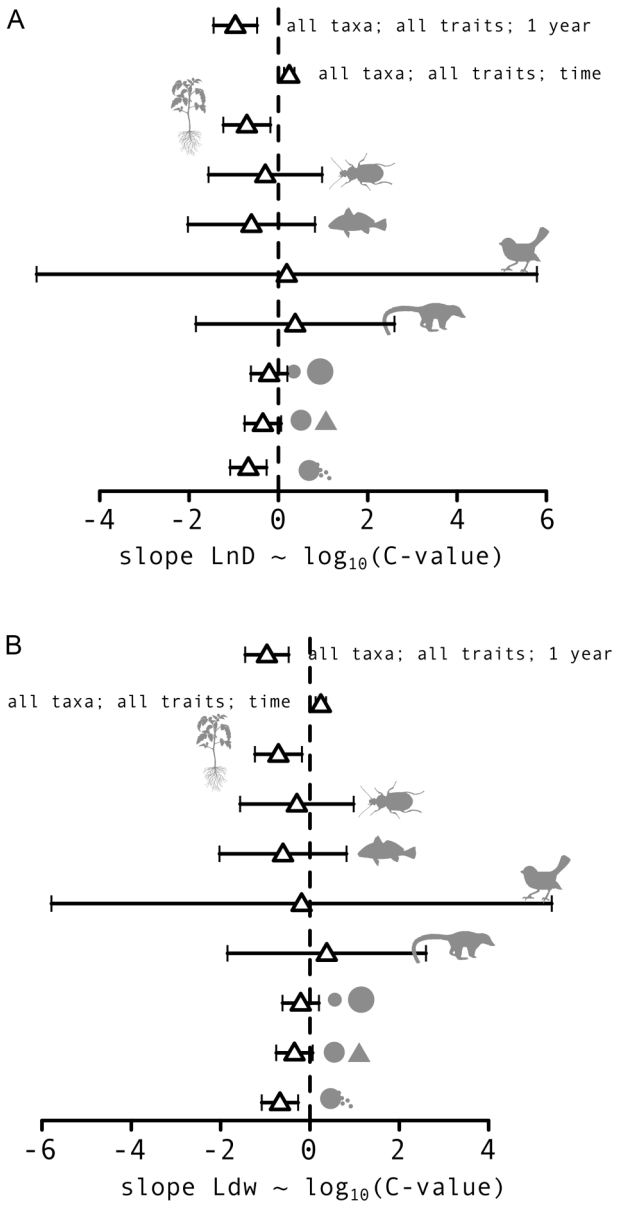

Figure 3. Slopes (and 95% confidence intervals) associated with predictions 1.iii and 2.ii (see Box 1). We computed slopes separately by "*taxa*" (top section in each panel) and then by trait types (bottom section in each panel). (a) Slopes of the amount of change (darwins-numerator effect size, *LnD*) as a function of the genome size (C-value). (b) Slopes of the rate of change (darwins effect size, *Ldw*) as a function of the genome size (C-value). Iconography is as in Figure 1.

C-value and the interactions between C-value and trait type (Table S1). However, including the genome size and its interactions (with taxa, elapsed time, and trait type) contributed <1% to the explained deviance of the variance-scaled phenotypic change (Base models explained 10.61% and 29.5% of LG and LR deviances, respectively; Table S3). Also, there were no significant differences among trait types, as well as among "*taxa*" (Figure 4). Overall, the larger the genome size, the lower the amount of phenotypic change was observed (Figure 4; Table S2), but this overall pattern was driven by plants and fishes. Other "*taxa*" showed no relationship between variance-scaled phenotypic change and genome size (Figure 4; Table S2). A similar situation was observed

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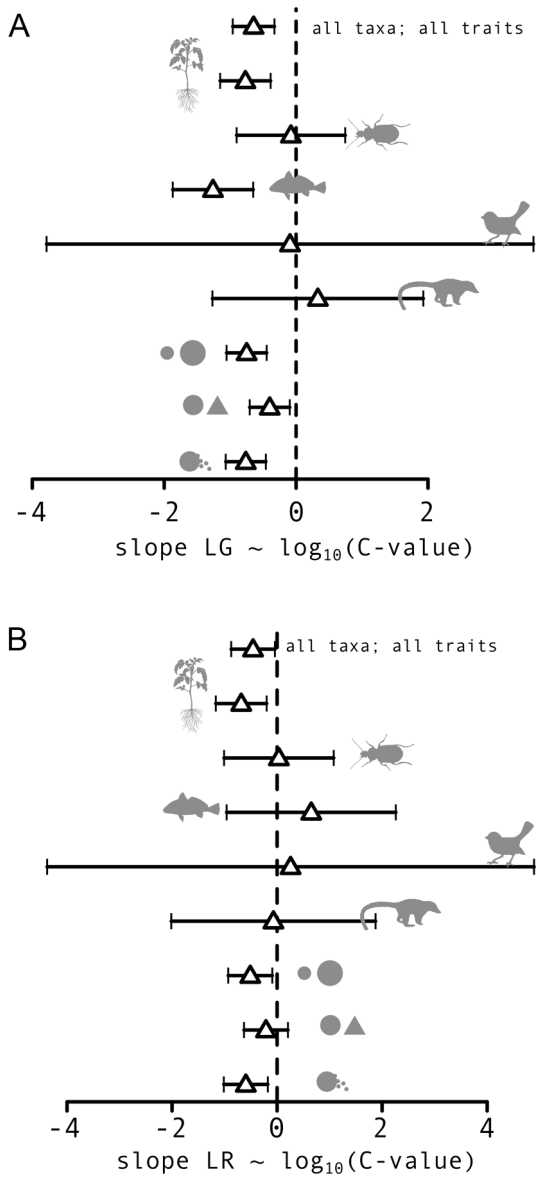

Figure 4. Slopes (and 95% confidence intervals) associated with predictions 1.iv, 2.ii, and 2, iii (see Box 1). We computed slopes separately by "*taxa*" (top section in each panel) and then, by trait types (bottom section in each panel). (a) Slopes of the amount of change (haldanes-numerator effect size, *LG*) as a function of the genome size (C-value). (b) Slopes of the rate of change (haldanes effect size, *LR*) as a function of the genome size (C-value). Iconography is as in Figure 1.

in the relationship between the variance-scaled rate of change and genome size, the overall slope was negative but this pattern was driven by plants, which is the only "*taxa*" with a slope significantly different from zero (Figure 4; Table S2).

Discussion

Our hypotheses

Our goal was to test the hypotheses that (1) a larger genome size correlates with greater and faster phenotypic change because larger genome size allows an increase in genetic variance and phenotypic plasticity; and (2) a larger genome size correlates with slower phenotypic rates of change for plants, arthropods and fishes, but not for birds and mammals, because of the effect of genome size on the generation time of the species. We found evidence of a relationship between genome size and the amount and rate of phenotypic change only for fishes and plants and not for other "*taxa*." However, none of the proposed hypotheses were supported.

In the first hypothesis, only the first prediction was fulfilled. That is, (1.i) the phenotypic variance (*pSD*) was positively correlated with the phenotypic change measured as darwins. According to the theoretical models, that is the expected situation for most of the environmental patterns of change if the phenotypic change is adaptive (Lande & Shannon, 1996). These results confirm that PROCEED records, in general, phenotypic changes of traits linked to fitness in situations where the environment is changing or has changed in a nonrandom (uncorrelated) way. This also highlights the relevance of the standing diversity within populations that face a changing environment, especially the challenges posed by humans to natural populations.

A salient result we found while assessing the effect of the phenotypic variance on the mean-scaled phenotypic change (prediction 1.i) was the interaction between phenotypic variance and elapsed time (Figure 1). The first one has, at least, two relevant implications that are perspectives on the same process. The longer the elapsed time, the more positive the slope of phenotypic variance on the mean-scaled phenotypic change was, indicating that the greater the phenotypic variance in the trait, the more the change accumulates over time. The second perspective is that the larger the phenotypic variance of the trait, the longer the time a population can sustain a directional change. According to previous studies, phenotypic change on contemporary time scales fit a pattern of bounded evolution (Kinnison & Hendry, 2001; Gorné & Díaz, 2019), characterized by an abrupt initial change followed by a period of stasis or some degree of random changes (Uyeda et al., 2011). The positive interaction between phenotypic variance and the elapsed time (on the phenotypic change) implies that the "abruptness" of the pattern of change could depend on the degree of phenotypic variance available for a given trait, such that the more the phenotypic variance, the less abrupt (and more sustained) the directional change across time will be.

A second salient result we found while assessing prediction 1.i was the difference in the slope among trait types. Life-history traits showed the steepest slope in the relationship between phenotypic change and phenotypic variance. This pattern is consistent with expectations for traits closely related to fitness to then experience stronger selection pressures (but see Hereford et al., 2004).

Unlike prediction 1.i, prediction 1.ii was not supported consistently across all "*taxa*" (Figure 2). This is key because, from that, any relationship between genome size and phenotypic change cannot be explained by our hypothesis 1. In line with this, the genome size contributed only marginally in explaining the mean-scaled phenotypic change ($<$1.8%), and the slope of phenotypic change as a function of the genome size was neutral for all taxa but plants. However, this slope was negative rather than positive. In this case, the negative slope of phenotypic change as a function of genome size cannot be explained by our hypothesis 2 either (see below).

We unexpectedly found a positive interaction between genome size and time on the mean-scaled phenotypic change (Figure

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3). As previously discussed for the interaction between phenotypic variance and time, this suggests that the "abruptness" of the pattern of change could depend on the genome size, such that the larger the genome size, the less abrupt (and more sustained) the directional change across time will be. However, the phenotypic variance would not mediate such an effect of genome size.

In the second hypothesis, the key prediction 2.ia was not supported (Table 2), but some results seemed to adjust to the expectations, especially in plants. For the three "*taxa*" we predicted a negative relationship between genome size and phenotypic change (prediction 2.ii), yet our results are different for each biological group. In arthropods, we found no relationship between genome size and generation time (prediction 2.i) or phenotypic change (prediction 2.ii), but the number of species with different genome sizes in PROCEED is too small to draw a robust conclusion about this group (Table 2). In plants, we found no relationship between genome size and generation time (prediction 2.i). However, we found a negative relationship between both mean-scaled and variance-scales phenotypic change and genome size (prediction 2.ii). Previous studies have reported a positive correlation between genome size and minimum generation time in plants (hereafter, MGT: the duration of the minimum period from germination until production of the first mature seed) (Bennett, 1987). The MGT and generation time (i.e., the time, in years, in which a population replaces itself) might be weakly correlated in plants, especially in long-lived perennial species. Many long-lived perennial species start reproducing early in their life cycle and continue reproducing for a long period. Consequently, the population and its trait values are replaced gradually by a new generation. This could explain the observed patterns of phenotypic change as a function of the genome size, as well as the unexpected negative relationship between genome size and the haldanes rate of change. For a given generation time, larger genomes would correspond to fewer MGT intervals within that period, resulting in less phenotypic change per generation—that is, lower haldane values. Finally, in fishes we found no correlation between genome size and generation time (prediction 2.i). We also found no relationship between the genome size and the mean-scaled phenotypic change (prediction 2.ii, partially) or the variance-scaled rate of change (prediction 2.iii). However, we found a negative effect of genome size on the variance-scaled amount of change (prediction 2.ii, partially).

As expected for amniotes (here represented by birds and mammals), no relationship was found between genome size and generation time or the phenotypic change.

An intriguing result is the particularly large confidence interval observed for the slope of phenotypic change in birds (Figures 1, 3, and 4). This large uncertainty is due to a combination of low variability in bird genome size and phenotypic variance, the predictor variables we used, and large variability (not smaller than in other groups) in the amount and rate of change (Figure S2). The genome size in birds is constrained due to selective pressures that favour small cells, high metabolic rates, and brain complexity (Andrews, 2009).

Alternative hypotheses

The Large Genome Constraint Hypothesis (Knight et al., 2005) proposes that large genome sizes constrain the ability of species to deal with stressful environments and reduce phenotypic plasticity (Meyerson et al., 2020). A reduction in the phenotypic plasticity would contribute to a reduction in the amount of phenotypic change if this were not compensated for by an increase in the change due to a genetic change. However, the evidence found here does not support such a hypothesis. The LGCH is a general proposition, and only the plants displayed a negative effect of the genome size on the amount and rate of change.

The effective population size (i.e., a measurement of the rate of evolutionary change caused by genetic drift in a finite population—Falconer, 1989; Charlesworth, 2009) could also play a role in the observed patterns. The smaller the population, the larger the effect of genetic drift (Charlesworth, 2009). Because the genetic drift operates as noise (a random error), the smaller the population, the lower the effectiveness of selection in driving a change in the adaptive direction because of larger random variation (Lopes de Souza et al., 2000; Charlesworth, 2009). In a broad analysis that included all "*taxa*," the effective population size was negatively correlated with genome size (Lynch & Conery, 2003). This general pattern was also observed in fishes (Yi & Streelman, 2005), regardless of the causal relationship between genome size and effective population size (Gregory & Witt, 2008), but not for mammals (Roddy et al., 2021), plants (Whitney et al., 2010), or insects (Alfsnes et al., 2017, most of the arthropod species in this database are insects). The relationship between genome size and effective population size has not been documented for birds. However, if the genome size was a good proxy for fish effective population size in the present work, we would expect a consistent negative relationship between genome size and all measures of phenotypic change (mean-scaled, variance-scaled, amount, and rate), and not only for one of them.

Conclusion

The genome size can affect the amount and rate of phenotypic change at contemporary time scales (microevolutionary process), but only in plants, and not in mammals, birds, arthropods, or fishes. The observed effect may be mediated by the relationship between genome size and the minimum period from germination until production of the first mature seed. Certainly, the contribution of genome size to phenotypic change variability is small (<2% in our analyses), and it is unlikely to be mediated by an effect on phenotypic variance or on generation time. Our results also provide evidence against the Large Genome Constraint Hypothesis and against an effect of genome size mediated by a relation with effective population size. Even when largely confirmatory from an evolutionary point of view, the consistent and relevant effect of phenotypic variance on the mean-scaled phenotypic change is worth highlighting from a conservation perspective. Assessing the phenotypic variability of traits related to the resource use strategies or the niche breadth in different dimensions may provide relevant information about the vulnerability of natural populations facing unexpected environmental changes. Finally, in a search for species properties that may be predictive of the microevolutionary outcomes, we think it is necessary to make a direct assessment of the effect of the generation time, the MGT, and the population size (or some proxy of them) on the phenotypic change in contemporary time scales.

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Supplementary material

Supplementary material is available at *Journal of Evolutionary Biology* online.

Data availability

PROCEED v6.2 is available at its webpage (https://proceeddatabase.weebly.com/) as well as in the stable repository Borealis (https://doi.org/10.5683/SP3/NXSL3Q).

Author contributions

Lucas D. Gorné (Conceptualization [lead], Data curation [lead], Formal Analysis [lead], Visualization [lead], Writing – original draft [lead], Writing – review & editing [equal]), Andrew P. Hendry (Conceptualization [supporting], Funding acquisition [equal], Writing – review & editing [supporting]), Fanie Pelletier (Conceptualization [supporting], Funding acquisition [equal], Writing – review & editing [supporting]), and Kiyoko M. Gotanda (Conceptualization [supporting], Data curation [equal], Funding acquisition [equal], Project administration [lead], Supervision [lead], Writing – review & editing [equal])

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Conflict of interest

We have no conflict of interest to declare.

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