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LETTER

Adaption, neutrality and life-course diversity

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Abstract

Heterogeneity among individuals in fitness components is what selection acts upon. Evolutionary theories predict that selection in constant environments acts against such heterogeneity. But observations reveal substantial non-genetic and also non-environmental variability in phenotypes. Here, we examine whether there is a relationship between selection pressure and phenotypic variability by analysing structured population models based on data from a large and diverse set of species. Our findings suggest that non-genetic, non-environmental variation is in general neither truly neutral, selected for, nor selected against. We find much variations among species and populations within species, with mean patterns suggesting nearly neutral evolution of life-course variability. Populations that show greater diversity of life courses do not show, in general, increased or decreased population growth rates. Our analysis suggests we are only at the beginning of understanding the evolution and maintenance of non-genetic non-environmental variation.

KEYWORDS

COMADRE, COMPADRE, demographic stochasticity, individual heterogeneity, life-history evolution, matrix population models, phenotypic variance, sensitivity

INTRODUCTION

Individuals in any population vary in their life courses, exemplified by differences in lifespan, reproduction and phenotypic characteristics (Endler, 1986; Hartl & Clark, 2007; Steiner & Tuljapurkar, 2012; Tuljapurkar et al., 2009). Classical evolutionary theories, founded in seminal work by Fisher (1930), Wright (1931) and Haldane (1927, 1932), explain such variation by genotypic variation, environmental variation or their interaction. According to these theories, if environments are constant over many generations, selection should erode genotypic variation by selecting for very few adaptive phenotypes and their associated genotypes; in population genetic terms, additive genetic variation should erode. However, neutral molecular variation maintains some genetic diversity without substantial phenotypic variation, if the phenotypes are selected (Crow & Kimura, 1970; Kimura, 1968). In consequence, in a constant environment, individual variation in phenotypic characteristics and life courses should decline if phenotypes are linked to fitness and trade-offs among life-history traits do not balance each other and thereby maintain phenotypic variation. These predictions are challenged by the observation that even isogenic individuals, originating from parental populations that have lived for many generations in highly controlled lab conditions, exhibit high levels of variation among individual life courses and phenotypes, even for phenotypes that directly link to fitness and that are under selection (Flatt, 2020; Jouvet et al., 2018; Steiner et al., 2019). Similarly, in less controlled genetic and environmental conditions, environmental variation, genotypic variation and their interaction only account for a small fraction of the total observed phenotypic variation in fitness components (Snyder et al., 2021; Snyder & Ellner, 2018; Steiner et al., 2021; van Daalen & Caswell, 2020). For systems where such a decomposition of genotypic, environmental and other stochastic variation is challenging because of a lack of accurate data, similar amounts of total phenotypic variation are observed as in more controlled systems (Finch & Kirkwood, 2000; Snyder & Ellner, 2016; van Daalen & Caswell, 2020). The question arises, how can such high levels of phenotypic variation be maintained, knowing that basic evolutionary theories do not readily predict the persistence of such high levels

of variability (Barton et al., 2017; Bell, 2010; Flatt, 2020; Melbourne & Hastings, 2008). From an empirical point of view, estimates of heritability of functional traits and resulting expectations of trait shifts frequently do not match observed fluctuations in phenotypic traits of natural populations (Coulson et al., 2008, 2010; Flatt, 2020). These challenges in explaining observed variability only by genotypes, environments and their interaction lead us to the view that non-genetic and non-environmental processes generate and contribute to the high levels of variation in phenotypes and life courses among individuals (Jouvet et al., 2018; Snyder et al., 2021; Snyder & Ellner, 2018; Steiner et al., 2019, 2021; van Daalen & Caswell, 2020).

The fundamental question we address here is whether such a non-genetic, non-environmental-driven variation is truly neutral, selected for or against. In the case of neutral variation, the follow-up question would be, how is such neutral variation maintained (Demetrius, 1974)? Here we do not decompose variance in genetic, environmental, phenotypic plastic (gene-by-environment) and neutral contributions to life-course variability, as previously done for datasets that have the needed depth of information or by making assumptions about partitioning (Snyder et al., 2021; Snyder & Ellner, 2018; Steiner et al., 2021). Instead, we aim at quantifying the selective forces on the processes that generate variation among life courses by relying on the analysis of structured population models (Steiner & Tuljapurkar, 2020). We describe this approach in the following section starting with structured populations and associated life courses.

In any structured population, a life course of an individual can be described by a sequence of stages that ends with death (Caswell, 2001). These stage sequences, or life-course trajectories, differ among individuals in length, i.e. age at death, and in the sequence and frequency of stages experienced. Stages can comprise many traits, e.g. size in development (say of newborns, juveniles, subadult and adults), reproductive state (say immatures, non-breeders and levels of reproductive output), other traits (such as behaviour, morphology, physiology and gene expression) and even non-biological features (say location or physical environment). Obviously, models simplify phenotypes to one or a few traits, but even so trait values will change during ontogeny within an individual and among individuals may not follow the same time sequence. Thus, stage sequences, life courses and phenotypes are linked and so is their diversity. After birth, there is a growth of diversity in stage sequences and a corresponding growth of diversity in phenotypic characteristics. In this sense, the rate at which sequences of stages diversify with increasing length, quantified by population entropy (Hernández-Pacheco & Steiner, 2017; Steiner & Tuljapurkar, 2020; Tuljapurkar et al., 2009), is also useful as a measure of phenotypic diversity.

When describing such stage sequences or life courses in population models, all individuals often start in the same newborn stage, thereby discarding differences in (often unknown) birth characteristics. But individuals can also be born into one of a few stages, as frequently modelled for plants (e.g. sexual reproductive: seed or seedling; clonal reproductive: offshoot). After being born life diversifies in stage sequences followed, and hence, phenotypic characteristics with increasing age and the rate at which these sequences of stages diversify with increasing length can be quantified by population entropy (Hernández-Pacheco & Steiner, 2017; Steiner & Tuljapurkar, 2020; Tuljapurkar et al., 2009). In age-only structured models, the length of life is the only aspect that varies among individuals, but the stage sequence is the same among individuals; if an individual survives, it simply enters the next age class as any other surviving individual does without differentiating characteristics. Demetrius' entropy (1974) quantifies the variability in reproductive output of such age-only structured populations with increasing age, and Demetrius' entropy contrasts with Keyfitz's entropy (actually, the latter is not mathematically an entropy) that also applies to age-only structured populations but quantifies changes in life expectancy caused by changes in age-specific mortality. Here, we use a measure of entropy that is a generalization of Demetrius' entropy, in that the population entropy we use emphasizes differences in reproduction/survival/growth generated by stage transition dynamics. In stage-structured population models, high population entropy leads to highly diverse life courses in short times and low entropy leads to few distinct life courses that groups of individuals follow (Hernández-Pacheco & Steiner, 2017). To be precise, entropy measures the rate of diversification in stage sequences of a cohort. As this rate relates to the diversification of life courses of such a cohort, it also relates to the diversity of life courses at different ages of a cohort. Not only the life courses, i.e. the stage sequences, but also their rate of diversification are determined by the stage transition rates (Caswell, 2001). To quantify the contributions of each stage transition to the rate of diversification of life courses (Steiner & Tuljapurkar, 2020), we can perturb each stage transition rate, i.e. elements of the population matrix model, and then compute the contributions of these perturbations to population entropy. Of course, such estimation of the sensitivity of each transition rate to the population entropy does not reveal anything about fitness—λ, the rate at which a population grows (Caswell, 2001; Steiner & Tuljapurkar, 2020).

However, the desired linkage to fitness is revealed by the sensitivities of the population growth rate, λ, to the same perturbations of the transition rates of the model. If one then examines the correlation between the sensitivities of entropy and fitness—both are estimated for each transition rate of a given model—, we can link the rate (process) of life-course diversification and selective forces (Figure 1) (Steiner & Tuljapurkar, 2020). To expand on this argument, if a perturbation of a stage transition parameter in a model leads to both an increase in entropy and population growth rate (fitness λ), selection for greater diversification

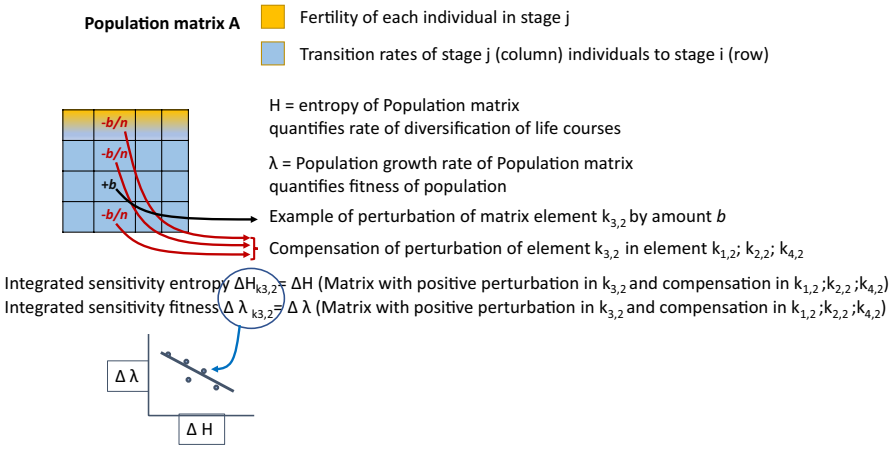

FIGURE 1 Sketch: for each population matrix, we estimated for each element (here exemplified by element $k_{3,2}$) an integrated sensitivity with respect to entropy ($\Delta H_{k3,2}$) and with respect to fitness ($\Delta\lambda_{k3,2}$) by increasing (perturbing) element $k_{3,2}$ by amount b and simultaneously reducing elements $k_{1,2}$; $k_{2,2}$; $k_{4,2}$ by b/n with n = (number of non-zero column elements) − 1. Such integrated sensitivities were then computed for each matrix element $k_{i,j}$ and for both types of sensitivities. For each population matrix we fitted a linear model through data points based on these two types of sensitivities from each of the $k_{i,j}$ elements. Each line in Figure 2 corresponds to such a correlation model.

of life courses is favoured, whereas, if a negative correlation between these sensitivities occurs, selection against diversification is suggested, and if there is no correlation between the two sensitivities, the observed variability among life courses may be neutral. We base our interpretation on the idea that selection should act more strongly on stage transitions that have higher sensitivities with respect to population growth, λ, and hence, fitness (Pfister, 1998). To illustrate the concept, imagine a mutation that changes a stage transition rate (fertility rate or other stage transition rate), if this change in transition probabilities influences fitness, λ, more than changes in other transition rates, it should be under stronger selection than those transition rates that only have little influence on fitness.

To evaluate how the diversity in life courses is selected upon—positively, negatively or neutral—, we explore the correlation of the sensitivity with respect to entropy and the sensitivity with respect to population growth for a large variety of species and taxa for which population projection models have been collected within the COMADRE and COMPADRE databases (Jones et al., 2022; Salguero-Gómez et al., 2016) (COMPADRE & COMADRE Plant Matrix Databases, 2022). Available from: https://www.compadre-db.org; accessed 7.3.2022. We estimate for each transition rate of each population projection model the sensitivity with respect to entropy and population growth, then correlate these two sensitivities for each projection model and compare these correlations across species, taxa, phyla, ontology, age (models containing at least one class that is based on age; in our case, it needs to be in addition to the stage structure), organism type and matrix dimension for plants and animals. We find that both in plants and animals, substantial variation in the correlation between the two sensitivities among species exists and we find a very weak or no overall correlation between sensitivities, suggesting close to neutral evolution of life-course variability.

We also address a different question, whether populations or species with high rates of life-course diversification exhibit high fitness compared to those that diversify at a lower rate in their life courses. Such investigation might be understood in terms of adaptive niche differentiation or specialization (Hernández-Pacheco & Steiner, 2017). Here, our findings suggest that matrices with high rates of diversification (higher entropy) do not show increased or reduced fitness. Note, only a single entropy and a single population growth rate are calculated per matrix, while for each of the many transition rates (non-zero matrix elements), sensitivities can be calculated. Overall, we find that populations that diversify at higher rates in their life courses do not show increased or decreased population growth rates and selective forces seem not to increase or decrease life-course diversification.

MATERIALS AND METHODS

Of the 3317 population matrices in the COMADRE animal database and the 8708 matrices in the COMPADRE plant database, we selected matrix models that were ergodic and irreducible (1350 and 5823, respectively). Of these, we selected only matrices that had for each stage (each matrix column, Figure 1) at least two non-zero elements (one of them could be a reproductive stage). This resulted in 37 matrices on 11 animal species, and 2144 matrices on 262 plant species. The extreme reduction in the animal matrix number reflects that many of these animal matrices are sparse matrices, for instance, age-structured-only (Leslie) population matrices. Note, most of the animal matrix models are coming from marine organisms that show slow growth, such as corals, sponges and tunicates, hence, the animal data are highly biased and not representative of all animals. This bias is not generated by theoretical limitations but rather by a lack of data on animal populations

and formulation of non-sparse stage-structured animal matrix models. Nevertheless, we end up with a biased and relatively small sample of animals.

We limited the analysis to matrices with at least two non-zero elements per stage to evaluate perturbations (sensitivities) that do not trade-off against survival, but against other stage transitions or reproductive rates (Figure 1). We call these sensitivities integrated sensitivities (Steiner & Tuljapurkar, 2020). Each integrated sensitivity evaluates by how much a perturbation of amount b, in one focal matrix element k, influences population entropy, H, and population growth, λ, when simultaneously all other non-zero elements in the given stage (column) are reduced by b/n, with n equals the number of non-zero elements in a column minus the focal element. Note, integrated sensitivities can have positive or negative signs, i.e. they can increase or decrease entropy or λ. For more details on entropy and integrated sensitivities, also see the Supplemental Information where we give an illustrative example of our estimation (SI 1).

Before we estimated the integrated sensitivities, we transformed the absorbing population projection matrices into Markov chains (Tuljapurkar, 1982) (SI 1). We then computed for each of the 41,812 non-zero matrix elements their integrated sensitivities with respect to population entropy and population growth rate λ on the plant matrices and 602 non-zero elements of the animal matrices. As the integrated sensitivities had very heavy tail distributions on both tails, we excluded extreme values that more likely arose from biologically unrealistic matrix parameter entries. Note, transition rates that were close to 1 or 0 did not result in extremely integrated sensitivities (Figure S3). We excluded extreme values of integrated sensitivities that exceeded three times the standard deviation for integrated sensitivities of entropy (13 animal matrix elements; 811 plant matrix elements) and values on integrated sensitivities of lambda that exceeded three times the standard deviation for the animal data (16), or 0.02 (a less conservative value than 3 × SD) for the plant data (559), leaving 577 integrated sensitivities and 40,640 integrated sensitivities, respectively, for the animal and plant data analysis (4 and 198 were outliers for both integrated sensitivities, of respectively, animal or plant data). Resulting distributions, after the outlier removal, remained heavy tailed.

For statistical testing, we fitted linear models (despite symmetrically long tails on both sides of the residual distribution) and used model comparison based on Akaike's information criterion (AIC). We defined a difference in AIC >2 as substantial better support (Burnham & Anderson, 2004). We evaluated the model fit and the assumptions using diagnostic plots.

For each matrix, we also computed population matrix-level entropy and population growth rate, λ; note there is one value of entropy and population growth for each matrix. We also correlated sensitivities with respect to entropy and those with respect to lambda for the 37 animal matrices and 2144 plant matrices against each other. Model comparisons were done using AIC (Burnham & Anderson, 2004).

RESULTS

We show the integrated sensitivities of entropy and those of lambda across all animal species in our data in Figure 2a. The figure shows no evident correlation between these sensitivities (supported by statistical analysis, Table 1: Model 1 [null model with only an intercept], vs. Model 2 [simple regression of the two sensitivities, slope −0.084], both models receive equal support). Hence, neither selection for nor against higher or lower rates of diversification of life courses is observed in animals. For plants, we find a weak positive correlation

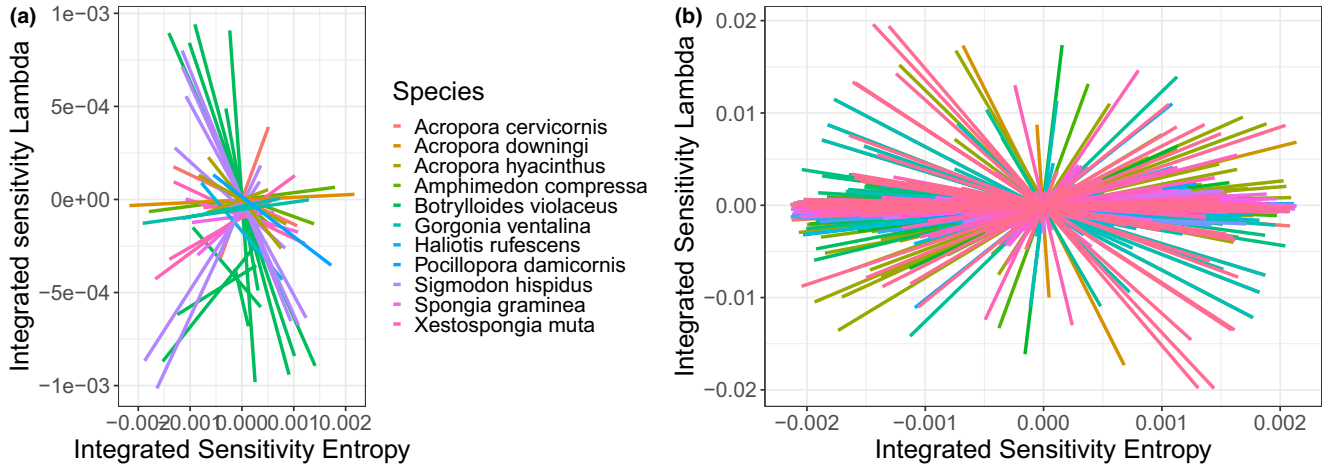

FIGURE 2 Correlating integrated sensitivities with respect to entropy and that with respect to lambda for animal populations (a) and plant populations (b). Each line fits the correlation for one population (one matrix model). Line colors reflect the different species as more than one matrix model can be fitted per species (e.g. different years, or populations). For the plant data (b) the number of species is too large to differentiate among the species. For better visibility CI (confidence intervals) are not plotted.

TABLE 1 Model selection among competing models based on animal and plant matrix population models evaluating the correlation between integrated sensitivities with respect to entropy (response variable) and integrated sensitivities with respect to population growth lambda (explanatory variable) and various covariates.

		Animals			Plants		
Model#	**Parameters**	**df**	**AIC**	**ΔAIC**	**df**	**AIC**	**ΔAIC**
1	Intercept only model	2	−7206.0	122.0	2	−514,160.2	16,740.3
2	SensEntr~SensLambda	3	−7206.8	121.2	3	−514,477.0	16,423.5
3	SensEntr~SensLambda+MatrixID	39	−7169.5	158.5	2146	−515,011.4	15,889.1
4	SensEntr~SensLambda×MatrixID	75	**−7328.0**	0.0	**4289**	**−530,900.5**	0.0
5	SensEntr~MatrixID	38	−7166.0	162.0	2145	−514,832.1	16,068.4
6	SensEntr~Species	12	−7203.3	124.7	263	−517,217.2	13,683.3
7	SensEntr~SensLambda+Species	13	−7204.5	123.5	264	−517,418.6	13,481.9
8	SensEntr~SensLambda×Species	23	**−7241.3**	86.7	**525**	**−525,791.6**	5108.9
9	SensEntr~SensLambda×Species+MatrixID	49	−7207.1	120.9	2407	−523,644.7	7255.8
10	SensEntr~SensLambda×AgeStructure	5	−7205.2	122.8	5	−514,541.0	16,359.5
11	SensEntr~SensLambda+AgeStructure	4	−7207.0	121.0	4	−514,484.8	16,415.7
12	SensEntr~SensLambda×MatrixDimension	5	−7204.4	123.6	5	514,536.1	1,045,436.6
13	SensEntr~SensLambda+MatrixDimension	4	−7206.3	121.7	4	514,475.0	1,045,375.5
14	SensEntr~SensLambda×Phylum	9	−7204.1	123.9	13	−514,793.7	16,106.8
15	SensEntr~SensLambda+Phylum	6	−7201.6	126.4	8	−514,587.3	16,313.2
16	SensEntr~SensLambda×OrganismType	11	−7202.1	125.9	21	−515,133.9	15,766.6
17	SensEntr~SensLambda+OrganismType	7	−7201.3	126.7	12	−514,491.7	16,408.8
18	SensEntr~SensLambda×Ontogeny	5	−7202.8	125.2	5	−514,555.5	16,345.0
19	SensEntr~SensLambda+Ontogeny	4	−7204.8	123.2	4	−514,476.0	16,424.5

Note: MatrixID = Data based on each Population Matrix Model evaluated with respect to the Population Matrix Model, AgeStructure = Yes/No distinction whether the Population Matrix Model included at least some age classes in addition to the stage structure, MatrixDimension = Number of stages in the matrix model. Further details on covariates can be obtained from the data source COMADRE & COMPADRE data base. Boldfaced models are best supported models, grey fond models are least supported models, non-boldfaced black models are partly equally well supported. ΔAIC compare to the best overall supported model.

(Table 1: Model 1 vs. Model2, Figure 2b), although its effect size (slope 0.056) is small (compared with effect size of non-significant animal data). Hence, for plants, selection tends to favour increased rates of life-course diversification. This said, there is substantial variation in the correlation between the integrated sensitivities among the species (Figure 2). The model that allows for one correlation per species, i.e. the model including the interaction between species and the integrated sensitivity of entropy (Table 1: Model 8) is better supported than models that are restricted to main effects or additive-only effects (Model 1, 2, 6 and 7). Similarly, there is significant variation in correlations among matrix models (i.e. slopes differ among correlations estimated for each matrix population model separately) (Figure 2) as Model 4 (Table 1) that accounts for the interaction between the matrix model ID and the correlation is better supported than models that only fit main effects or additive effects (Table 1: Model 2, 3 and 5). There is also significant variation among correlations per matrix within species, as Model 4 which fits for each matrix within a species 1 correlation is better supported than Model 8 that fits 1 correlation per species (Table 1: Model 8). These findings suggest that selection differs among species, i.e. favouring higher rates of diversification in life courses in some species while selecting against such diversity in others. In addition, variation in correlations among matrices but within species (Model 8 vs. Model 4) is significant and cannot be simply reduced to the species level. This finding shows that differences in selection are observed among populations of the same species or the same population in different years. These patterns of variance within and among species hold for both animal and plant data. These patterns are also robust when the analyses are limited to only matrix models with growth rates ≤1.5 (Figure S4), as one might be concerned that very fast-growing populations might drive our results on variance among species or populations. Figure 2 also suggests that many correlations show slopes close to 0, these correlations with shallow slopes do not show reduced residual variances compared to those with steeper slopes, i.e. slopes that are potentially under stronger selection (steeper slopes) do not have increased or reduced residual variance around their regression lines (Figure S5).

We investigated the effect on the correlation between sensitivities by using several possible grouping variables, including age (at least one age class in addition to stage structure), matrix dimension (number of stages), phylum, organism types (e.g. algae, fungi and annual for plants) or ontogeny. We found (Table 1, Figure S1) that

in animals these variables do not play an important role, while in plants they do account for a small amount of variability. Still, compared to the variance among species and within species, these grouping variables are of little importance. The number of stages per matrix (dimension) could potentially affect our findings because we found an interaction among matrices of different dimensions and integrated sensitivity with respect to lambda for plant species, but there was no general trend with increasing matrix dimension towards or against selection for variance in life courses, suggesting no systematic bias regarding matrix dimension (Figure S1).

We further asked whether high or low diversity in life courses (population entropy) is associated with high or low fitness (population growth rates). Note here, we evaluate population entropy and lambda for the total population, i.e. one value for each matrix, not as above, a measure at the matrix element level (integrated sensitivities measures). Figure 3 shows this relationship between entropy and lambda (see Table 2 for model comparison). We did not find any simple relationship between population entropy and fitness for animals as a null model with an intercept only was equally well supported than a model that fitted a correlation between lambda and entropy (Table 2, Model 1 vs. 2, Figure 3a). For plants, however, there was some tendency that matrices with higher rates of diversification had lower fitness as the model that fitted a correlation between lambda and entropy was better supported than a simple intercept-only null model (Table 2 Model 1 vs. 2, Figure 3b, slope −0.42). One necessary caution is that these results are largely driven by biologically questionable and extremely high values of population growth rates (see also Figure S2). Note, we tested that our findings on integrated sensitivities are not driven by these very fast-growing populations (Figure S4). Overall, there is significant variation in both population entropy and population growth rate but no clear correlation between the two variables. Matrix dimension explains some additional variance in the relationship between entropy and lambda, although species differences are much more important in explaining variance than matrix dimension (Table 2).

DISCUSSION

We show that across animal populations there is no clear selective force that acts towards or against increased or decreased rates of diversification in life courses, whereas for a large collection of plants, there is weak selection favouring diversification in life courses. Given that there is selection for higher rates of diversification in life courses, one might expect that populations with higher entropy would also have higher fitness, but in apparent contrast to this expectation, we find that plant populations (or species) with high rates of life-course diversification (high entropy) tend to have lower fitness than populations (or species) that show low rates of diversification. However, the actual rate of diversification (population entropy) and the selective force on that rate (integrated sensitivities of population entropy) reveal two different things. For instance, a population might have a low rate of diversification, but there might be a strong selective force of increasing that rate, or a population might have a high rate of diversification and there might be only a weak force of increasing or reducing that rate. The integrated sensitivity analyses investigate selective forces on the diversification processes within a population (Steiner & Tuljapurkar, 2020), whereas the population entropy quantifies the current rate of diversification (Tuljapurkar et al., 2009). The sensitivity analyses, therefore, focus on within-population selective processes, whereas entropy and population growth are best used for among-population comparison.

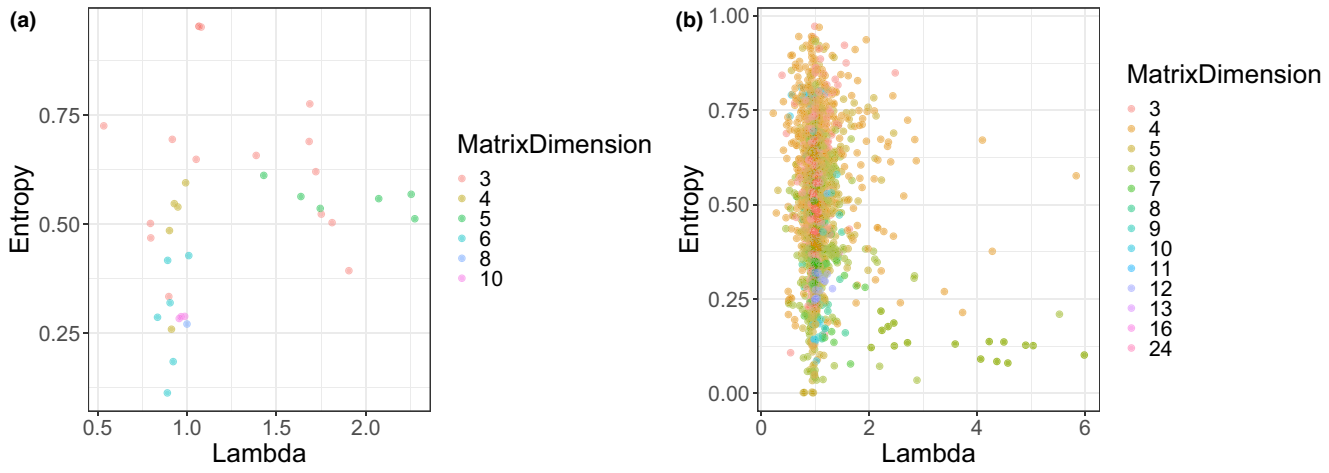

FIGURE 3 Relationship between population growth lambda (fitness) and population entropy, the rate of diversification, for animal (a) and plant (b) population models. Each data point represents one matrix model. Colors depict different dimensions of the matrix model. Populations that showed extremely low or high lambda are not plotted for better illustration. The full dataset, including the extreme values of lambda is plotted in Figure S2 and the model selection of Table 2 is also based on the full data set.

TABLE 2 Model selection among competing models based on animal and plant matrix models evaluating the correlation between population entropy (response variable) and population growth, lambda (explanatory variable), as well as matrix dimension and species comparison.

		Animals				Plants			
Model#	**Parameters**	**Slope**	**df**	**AIC**	**ΔAIC**	**Slope**	**df**	**AIC**	**ΔAIC**
1	Lambda ~ Intercept only model		2	50.19	49.72		2	2839.36	1255.66
2	Lambda ~ Entr	0.46	3	50.54	50.07	−0.42	3	2779.30	1195.6
3	Lambda ~ Entr + MatrixDim		4	52.02	51.55		4	2780.43	1196.73
4	Lambda ~ Entr × MatrixDim		5	**47.25**	46.78		**5**	**2754.89**	1171.19
5	Lambda ~ MatrixDim		3	52.16	51.69		3	2835.28	1251.58
6	Lambda ~ Species		12	19.16	18.69		263	1625.00	41.3
7	Lambda ~ Entr + Species		13	**0.47**	0		264	1624.83	41.13
8	Lambda ~ Entr × Species		20	3.52	3.05		**456**	**1583.70**	0

Note: MatrixDim = Number of stages of the matrix population model the entropy and lambda was estimated from. Boldfaced models are best supported models, grey fond models are least supported models, non-boldfaced black models are partly equally well supported. ΔAIC compare to the best overall supported model.

Our finding of substantial variation in selective forces on the rate of diversification, as well as substantial variation in the rate of diversification, might be of greater interest than the small positive selective trend favouring diversification for plant species. The interpretation of the animal models remains challenging, as the species for which non-sparse stage-structured population models are available is biased towards specific types of slow-growing marine organisms with often many offspring. These substantial levels of variability in selective forces and rates of diversification might have three different biological origins or meanings: first, they might indicate substantial (developmental) noise that leads to the observed variability in life courses and selection for or against diversification in life courses (Balázsi et al., 2011); second, it might indicate fluctuating selection or high levels of phenotypic plasticity driven by variable environmental conditions (Gillespie, 1975; Philadelphia, 1973); or third, it might indicate large numbers of distinct adaptive life courses that show similar fitness but might, for instance, fill different niches, or solve life-history trade-offs in many different ways that lead to similar fitness (Hernández-Pacheco & Steiner, 2017). In quantitative genetic terms, these options would relate to, respectively, undetermined residual variation, gene-by-environmental variation or additive variation.

If one assumes that noise explains the variability, it is suggested that selection might not act very strongly on this noise, as otherwise, the variability should be selected against and variability should collapse (Fisher, 1930; Haldane, 1927, 1932; Wright, 1931). Such neutral, or close to neutral, arguments have been used in the past to explain life-course variability but are often met with scepticism (Steiner & Tuljapurkar, 2012). Our results might indicate that selective forces on rates of diversification in life courses are not generally weak, but partly go in opposing directions, i.e. selecting for diversification in some populations or species and against in others. This interpretation is also supported by our finding that residual variance is not related to the force of selection (Figure S5). Conflicting findings as we reveal are also found commonly in other fields, for instance, in quantitative genetic studies (Charlesworth, 2015; Flatt, 2020; Johnson & Barton, 2005).

If one assumes that fluctuating environments or similar extrinsic variation causes vital rates to differ among matrices and leads to highly diverse life courses (Gillespie, 1985; Philadelphia, 1973), we might assume that a large fraction of variability would be explained by among matrix models *within* species, and less so *among* species. Model selection indicates that among-species variation is substantially greater compared to variability among matrices within species. Hence, variability among populations or time (years), or conditions (environments) within species contribute less to variability in life courses than variability among species. These arguments align with findings that phenotypic plasticity might not be in general adaptive (Acasuso-Rivero et al., 2019). The meta-analysis we did might not be ideal for such within-species evaluation, as the average number of matrices per species (3.4 for animals and 8.2 for plants) is not very large, but our analysis still provides more general insights compared to studies focusing on single-model species for which rich data exist (Flatt, 2020).

If one assumes that diversity in life courses is produced because many life courses are equally fit (Hernández-Pacheco & Steiner, 2017; Nevado et al., 2019), we would be challenged to explain the strong selective patterns against diversification that is observed for some populations and species. Under such an assumption, the optimal number of distinct life courses would need to differ substantially among species or populations. Also, from more detailed analyses of systems, certain life courses, or genotypes, that are commonly observed seem to have low fitness (Flatt, 2020; Steiner et al., 2021), suggesting that not all life-course variability might be adaptive. In addition, different solutions to life-history trade-offs that could generate life-course diversity would frequently

be comprised within a single-matrix model, potentially contributing to patterns that resemble noise and, therefore, could explain the maintenance of noisy signals.

The potential explanations that help to understand the selective forces on the rate of diversification of life courses are not mutually exclusive and we do not have means to quantify each contribution to the diversification using the models in this study. More detailed studies that focus and explore selection on diversification could help to better understand the influence of these three factors (Flatt, 2020). Studies might include how genes (or gene knockouts) influence the rate of diversification, how experimental evolution studies in stochastic environments differing in amplitude and autocorrelation (noise colour and wavelength) would lead to the evolution of different rates of diversification, how "heritability" of distinct life-course strategies potentially determine life-course diversification under different environmental conditions or how trade-offs among life-history traits maintain and generate diverse life courses. Quantitative genetics studies have identified a similar lack of understanding of the maintenance and the evolution of variability (Charlesworth, 2015; Johnson & Barton, 2005), although with a focus on genetic explanations emphasizing mutation–selection balance being driven by few strongly deleterious mutations (Charlesworth, 2015; Muller, 1950), or alternatively many polymorphic loci that maintain variability (Dobzhansky, 1955; Johnson & Barton, 2005). Such genetic variation interacts with neutral and non-genetically determined processes that influence evolutionary processes and the pace of evolution (Steiner & Tuljapurkar, 2012). For that, a purely quantitative genetic vision might be too short-sighted. Growing literature emphasizing how noisy gene regulation might scale and trigger cascading effects across levels of biological organization offers ways for more mechanistic understanding (Elowitz et al., 2002; Robert et al., 2018). Generally, we believe we are only beginning to understand selection of processes that lead to the observed variability in life courses (Flatt, 2020).

AUTHOR CONTRIBUTIONS

Ulrich Karl Steiner and Shripad Tuljapurkar developed the concept and theory. Ulrich Karl Steiner analysed the data. Ulrich Karl Steiner wrote the first and final drafts with substantial contributions from Shripad Tuljapurkar.

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DATA AVAILABILITY STATEMENT

R code and data for reproducibility are available on Figshare https://doi.org/10.6084/m9.figshare.21830235.v3. No new data were generated. This R code loads the currently available data from the COMPADRE and COMADRE Databases and is open access data under the terms of the Creative Commons CC BY-SA 4.0 licence. It can be accessed at https://www.compadre-db.org, for further details see https://doi.org/10.1111/1365-2745.12334 and https://doi.org/10.1111/1365-2656.12482.

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