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, or selected against. We find much variation 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 in understanding the evolution and maintenance of non-genetic non environmental variation.

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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, or selected against. We find much variation 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 in understanding the evolution and maintenance of non-genetic non environmental variation.

Introduction

Individuals in any population vary in their life courses, exemplified by differences in lifespan, reproduction, phenotypes, and functional traits (Endler 1986; Hartl & Clark 2007; Tuljapurkar *et al.* 2009; Steiner & Tuljapurkar 2012). 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 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 upon (Kimura 1968; Crow & Kimura 1970). In consequence, in a constant environment, among individual variation in phenotypes and life courses should decline if phenotypes are linked to fitness. 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 (Jouvet *et al.* 2018; Steiner *et al.* 2019; Flatt 2020). 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 of fitness components (Snyder & Ellner 2018; van Daalen & Caswell 2020; Snyder *et al.* 2021; Steiner *et al.*2021). For systems where such a decomposition of genotypic, environmental and other stochastic variation is challenging due to 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 such high levels of phenotypic variation can be maintained as basic evolutionary theories do not readily predict the persistence of such levels of variability (Melbourne & Hastings 2008; Bell 2010; Barton*et al.* 2017; Flatt 2020). 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 & Ellner 2018; Steiner *et al.* 2019, 2021; van Daalen & Caswell 2020; Snyder *et al.* 2021).

The fundamental question we address here is whether such 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 & Ellner 2018; Snyder *et al.* 2021; 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. In many situations every individual starts in the same newborn stage. Thereafter life diversifies with increasing age, and the rate at which these sequences diversify with increasing length can be quantified by population entropy (Tuljapurkar*et al.* 2009; Hernández-Pacheco & Steiner 2017; Steiner & Tuljapurkar 2020). High entropy leads to highly diverse life courses in short times, and low entropy leads to few distinct life courses groups of individuals follow (Hernández-Pacheco & Steiner 2017). The life courses, i.e. the stage sequences, but also their diversification are determined by the stage transition rates (Caswell 2001). To quantify the contributions of each stage transition to the process of diversification of life courses (Steiner & Tuljapurkar 2020), we can perturb each stage transition rate, i.e. elements of the population matrix, 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. If one then examines the correlation between the sensitivities of entropy and of fitness, we can link life course diversification and selective forces (Fig. 1) (Steiner & Tuljapurkar 2020). To expand on this argument, if a perturbation (of a stage transition parameter) leads to both an increase in entropy and in population growth rate (fitness λ), selection for diversification 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 vital rate (any fertility rate or stage transition rate), if this change in transition probabilities influences fitness, λ, more than changes in other vital rates, it should be under stronger selection than those vital 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 data base (Salguero-Gómez *et al.* 2016; Jones*et al.* 2022) (COMPADRE & COMADRE Plant Matrix Database (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, 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, i.e. more diverse life courses, exhibit high fitness compared to those that are less diverse 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 is calculated per matrix, while for each of the many non-zero matrix element sensitivities can be calculated. Overall, we find that populations that show greater diversity of life courses do not show increased or decreased population growth rates and selective forces seem not to increase or decrease life course variability.

MATERIAL & METHODS

Out 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, Fig. 1) at least two non-zero elements; resulting 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.

We limited the analysis to matrices with at least two non-zero elements as to evaluate perturbations (sensitivities) that do not trade-off against survival, but against other stage transition or reproductive rates (Fig. 1). We call these sensitivities, that do not trade-off against survival, 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 negative and positive effects on entropy or λ.

Before we estimated the integrated sensitivities we transformed the absorbing population projection matrices into Markov chains (Tuljapurkar 1982). We then computed for each of the 41812 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 likely arose from biologically unrealistic matrix parameter entries or transition rates that were close to 1 or 0. 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 for the plant data (559), leaving 577 integrated sensitivities and 40640 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's(Burnham & Anderson 2004).

RESULTS

We show the integrated sensitivities of entropy and those of lambda across all animal species in our data in Fig 2A. The figure shows no evident correlation between these sensitivities (supported by statistical analysis,

Table 1: Model 1 [null model], vs Model2 [simple regression, slope -0.084], both models receive equal support). Hence, neither selection for nor against the diversity of life courses is observed in animals. For plants we find a weak positive correlation (Table 1: Model 1 vs Model2, Fig. 2B), though its effect size (slope 0.056) is small (compare with effect size of non-significant animal data). Hence, for plants selection tends to favour diverse life courses. This said, there is substantial variation in the correlation between the integrated sensitivities among the species (Fig. 2; Table 1: Model 8 vs. Model 1,2,6,7), significant variation among matrices (Fig. 2, Table 1: Model 4 vs Model 2, 3, 5), and significant variation among matrices within species (Table 1: Model 4 vs. model 8). These findings suggest that selection differs among species, i.e. favouring diversity in life courses in some species while selecting against such diversity in others. On addition, comparisons among matrices for one species show differences among populations, or the same population in different years. The results reveal that variation within a single species and among populations and years is also substantial. These patterns of variance within and among species hold for both animal and plant data.

We investigated the effect on the correlation between sensitivities by using several possible grouping variables, including age, matrix dimension, phylum, organism types, or ontogeny. We found (Table 1, Fig. 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 (Fig. 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). Fig. 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 (Table 2 Model 1 vs. 2, Fig. 3A), though for plants there was some tendency that matrices with higher rates of diversification had lower fitness (Table 2 Model 1 vs. 2, Fig. 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 Fig. S2). Overall, there is significant variation in both population entropy and population growth rate but no clear correlation among the two variables. Matrix dimension explains some additional variance in the relationship between entropy and lambda, though species differences are much more important in explaining variance than matrix dimension.

DISCUSSION

We show that across a large collection of animal populations there is no clear selective force that acts towards or against increased or decreased diversification in life courses, whereas for a large collection of plants there is weak selection favouring diversification in life courses. In apparent contrast, we find that plant populations (or species) with high rates of life course diversification tend to have lower fitness than populations (or species) that show high rates of diversification. However, the two measures, the selective forces acting on the generating process of diversification in life courses, and the level of diversification, reveal two distinct aspects. The integrated sensitivity analyses investigates selection on 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.

Our finding of substantial variation in selective forces on the generating processes, 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. These substantial levels of variability 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(Philadelphia 1973; Gillespie 1975), or third, it might indicate large numbers of distinct adaptive life courses that show similar fitness but might for instance fill different niches(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(Haldane 1927, 1932; Fisher 1930; 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, such conflicting findings are not uncommon in quantitative genetic studies(Johnson & Barton 2005; Charlesworth 2015; Flatt 2020).

If one assumes that fluctuating environments, or similar extrinsic variation, causes vital rates to differ among matrices and leads to the highly diverse life courses(Philadelphia 1973; Gillespie 1985), 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) are 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.

The potential explanations that help to understand the selective forces on diversification of life courses are not mutually exclusive and we do not have means to quantify each contribution to the diversification using the data 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 color, wavelength) would lead to the evolution of different rates of diversification, or how "heritability" of distinct life course strategies potentially determine life course diversification under different environmental conditions. Quantitative genetics studies have identified a similar lack of understanding of the maintenance and the evolution of variability (Johnson & Barton 2005; Charlesworth 2015), though with a focus on genetic explanations emphasizing mutation-selection balance being driven by few strongly deleterious mutations (Muller 1950; Charlesworth 2015), 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. Generally, we believe we are only beginning to understand selection on processes that lead to the observed variability in life courses(Flatt 2020). Increased interest in stochastic gene expression and its scaling and cascading effect across biological organization illustrate efforts towards such understanding(Elowitz*et al.* 2002; Robert *et al.* 2018).

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Fig. 1: Sketch: for each population matrix, we estimated for each element (here exemplified by element $k_{3,2}$) an integrated s

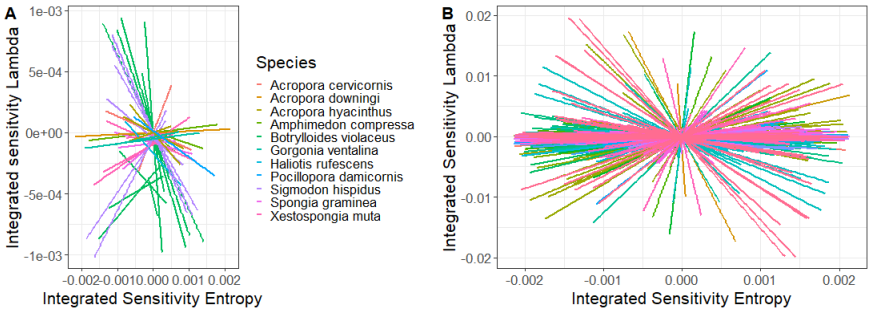

Fig. 2: Correlating integrated sensitivities with respect to entropy and that with respect to lambda for animal populations (A) and plant populations (B). Each line fits the correlation for one population (one matrix model). Line colors reflect the different species as more than one matrix model can 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.

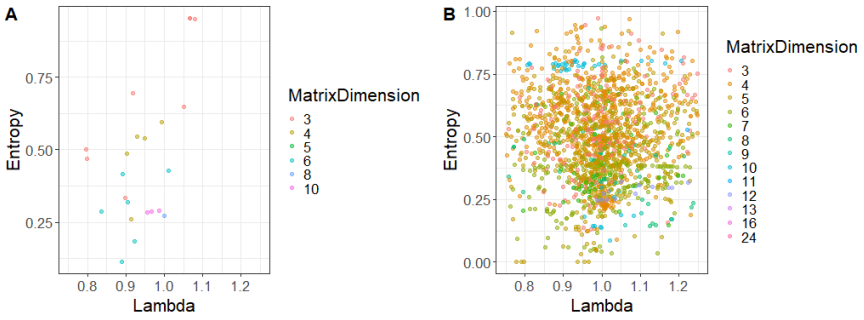

Fig. 3: Relationship between population growth lambda (fitness) and population entropy, the rate of diversification, for animal (A) and plant (B) population models. Each data point represents one matrix model. Colors depict different dimensions of the matrix model. Populations that showed extremely low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

Table 1: Model selection for animal and plant matrix data among competing 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	DF	AIC
1	Intercept only model	2	-7206.0	2	-514160.2
2	SensLambda	3	-7206.8	3	-514477.0

		Animals			Plants
3	SensLambda+Matrix	39	-7169.5	2146	-515011.4
4	SensLambda*Matrix	75	**-7328.0**	**4289**	**-530900.5**
5	Matrix	38	-7166.0	2145	-514832.1
6	Species	12	-7203.3	263	-517217.2
7	SensLambda+Species	13	-7204.5	264	-517418.6
8	SensLambda*Species	23	**-7241.3**	525	-525791.6
9	SensLambda*Species+Matrix	49	-7207.1	**2407**	**-523644.7**
10	SensLambda*Age	5	-7205.2	5	-514541.0
11	SensLambda+Age	4	-7207.0	4	-514484.8
12	SensLambda*Matrix Dimension	5	-7204.4	5	514536.1
13	SensLambda+ Matrix Dimension	4	-7206.3	4	514475.0
14	SensLambda*Phylum	9	-7204.1	13	-514793.7
15	SensLambda+Phylum	6	-7201.6	8	-514587.3
16	SensLambda*Organism Type	11	-7202.1	21	-515133.9
17	SensLambda+ Organism Type	7	-7201.3	12	-514491.7
18	SensLambda*Ontogeny	5	-7202.8	5	-514555.5
19	SensLambda+ Ontogeny	4	-7204.8	4	-514476.0

Table 2: Model selection for animal and plant matrix data among competing 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 SensLam.	DF	AIC	Slope SensLam.	DF	AIC
1	Intercept only model		2	50.19		2	2839.36
2	SensEntr	0.46	3	50.54	-0.42	3	2779.30
3	SensEntr+MatrixDim		4	52.02		4	2780.43
4	SensEntr*MatrixDim		5	**47.25**		**5**	**2754.89**
5	MatrixDim		3	52.16		3	2835.28
6	Species		12	19.16		263	1625.00
7	SensEntr+Species		13	**0.47**		264	1624.83
8	SensEntr*Species		20	3.52		**456**	**1583.70**