Plant populations track rather than buffer climate fluctuations

**Abstract**

Climate change not only affects mean temperature and precipitation but also exacerbates temporal fluctuations in these conditions. However, we know relatively little about how species respond to such climate fluctuations, with respect to variation in vital rates (i.e. survival, growth and reproduction of individuals) and population fluctuations. We examine whether populations display evidence of buffering against environmental variation in one of two ways: (1) through negative covariances among vital rates, or (2) reduction of variation in those vital rates to which population growth is most sensitive. We analyse time series of demographic data for 40 plant species and show that there is no evidence for either of these mechanisms. In species in which there is evidence for vital rate covariation, positive covariances between reproduction and survival rates predominate, and tend to magnify the effect of variability. Increasing climate variability is therefore expected to increase population fluctuations and extinction risks.

**Keywords**

Buffering, climate change, life history, life span, population dynamics, trade-offs, variance decomposition.

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**INTRODUCTION**

The response of species to climate change is variable (Root et al. 2003; Rosenzweig et al. 2008): some studies show that species can track interannual shifts in the onset of seasons through phenotypic plasticity (Charmantier et al. 2008) and/or micro-evolution (Nussey et al. 2005), while others emphasize the limits of species responses (Keith et al. 2008). However, not only are mean climate conditions changing, but also temporal fluctuations in climate, e.g. in temperature and precipitation (Salinger 2005; IPCC 2007). Previous work shows that increasing interannual variation in population growth rate, due to temporally fluctuating climate conditions, will generally reduce the long-term stochastic growth rate of a population and increase its risk of extinction (Boyce et al. 2006). Unfortunately, relatively little is known about how specific vital rates (i.e. survival, growth and reproduction of individuals) respond to fluctuations in particular climatic variables, which makes it difficult to predict population dynamics (Drake 2005; Melbourne & Hastings 2008). However, time series of stage-structured population monitoring provide data on the variability of vital rates and make possible analysis of how such variability affects population growth. An important question we can ask using such data is, does the pattern of past variability in vital rates suggest that populations have evolved to buffer their population size fluctuations against the impact of climate variability (Pfister 1998; Morris & Doak 2004)?

One possible mechanism that deflates variation in population growth rates is the existence of significant negative correlations between vital rates (Pfister 1998; Morris & Doak 2004). A different possible mechanism for buffering is that those vital rates to which average population growth rate is most sensitive are also the least variable (Pfister 1998). Previous
studies have shown that such a relationship does hold for some vital rates, but this relationship can be due to a spurious correlation between vital rate means and variances (Morris & Doak 2004). Also the argument for this mechanism ignores the possibility that vital rates may covary (Pfister 1998; Morris & Doak 2004; Haridas & Tuljapurkar 2005).

Here, we conduct a new analysis that tests whether these two buffering mechanisms (negative vital rate correlations and reduced variability in important vital rates) are significant in a large collection of plant species. A crucial element of our analysis is that we use a novel randomization method to ask whether correlations between rates are indeed significant, whether they be positive or negative. Thus, our analysis does not assume that we are accurately estimating vital rate covariance. This is especially important because most currently available datasets include far fewer years than the number of vital rates to be estimated, and thus cannot reliably estimate covariances (Al-Khafaji et al. 2007).

To study potential buffering mechanisms, we analyse matrix models estimated from data for 40 plant species. These datasets were selected to only contain temporal variation with no major changes in site management, and climate fluctuations are therefore likely the major source of annual variation in plant performance. We follow previous workers (Pfister 1998; Morris & Doak 2004; Morris et al. 2008) and quantify buffering in terms of the contribution of temporal variability in vital rates to stochastic population growth rate. We use Tuljapurkar’s small-noise approximation (Tuljapurkar 1982; Haridas & Tuljapurkar 2005), which states that the long-run stochastic population growth rate is equal to the deterministic population growth rate, but diminished by contributions of vital rate variation. The latter is the sum of the variance contributions of all vital rates and all contributions of the covariances between all vital rate pairs. Morris & Doak (2004) used this decomposition to point out how important it is to have an accurate estimate of the correlations between rates. Morris et al. (2008) quantified buffering using the magnitude of stochastic elasticities compared to the variances of the vital rates, but their results depend on the assumption that the available data provide an accurate estimate of the correlations between vital rates. Here, we compare the actual variance, covariances and stochastic $\lambda$ of populations with expectations of a null model with random (but realistic) vital rate variances, mimicking the absence of any systematic negative or positive covariances between vital rates. By analysing and comparing the observed and simulated vital rate variability patterns, we were able to test for evidence supporting either or both buffering mechanisms. Specifically, we expected that the observed contributions of vital rate variation would be significantly lower than in the simulations due to: (1) more negative correlations among vital rates or due to, (2) more negative correlations between elasticity values and coefficients of variation.

**MATERIALS AND METHODS**

We analysed demographic data for single populations of 40 different perennial plant species with a wide range of life spans. We only included studies for which demographic data were available for at least three annual transitions (see Appendix S1 for a complete species list). We analysed the variance and covariance contributions of the 958 vital rates with temporal variation. These patterns were compared to the same set of vital rates in 10 000 random datasets of the same size (i.e. the same number of years per species as in the original dataset). The vital rates in the random datasets were independently drawn from statistical distributions (beta for survival and growth rates; gamma for low reproduction rates, and negative binomial for high reproduction rates) that were shaped by the mean of the vital rates and by the general mean-variance relationships of vital rates for all species in the original datasets (see Appendix S1 for further details). We deemed the use of these general mean-variance relationships a more reliable method for shaping the statistical distributions than the use of the observed individual vital rate variances since these individual estimates were limited by the often relatively short study duration. Alternative simulations, in which observed variances were used instead, show that the simulation results and our conclusions are very robust to the choice of variance estimation method (see Appendix S2). We used discrete, negative binomial distributions (Ver Hoef & Boveng 2007) for high reproduction rates because these rates are often defined as the discrete number of seeds produced per flowering plant and show large variances compared to their means. We also performed additional simulations with the continuous log-normal distribution for either high reproduction rates or all reproduction rates and again found that our analyses are not sensitive to these methodological choices (Appendix S2). The vital rates that did not vary in the real dataset were kept constant in the new random-variation datasets. These simulations randomly redistribute the total amount of vital rate variance in the original database while retaining the general life cycle patterns of the 40 species.

For our analyses, we use Tuljapurkar’s small-noise approximation, $\log \lambda_s = \log \lambda_0 - V/2$, in which the long-run stochastic population growth rate ($\lambda_s$) is equal to the deterministic population growth rate ($\lambda_0$), but diminished by contributions ($V$) of vital rate variation. This small-noise approximation fitted the stochastic population growth rates well in all 40 species in both the data and in the simulations (Appendix S3). We focused on the contributions of vital rate
variation $V = \sum_{k,l} e_{kl} \cdot c_{kl} \cdot \text{Corr}(k,l)$, which consists of the correlation matrix of all pairs of vital rates $k$ and $l$, weighted by the elasticity values ($e$, proportional sensitivity of the projected population growth rate) and coefficients of variation ($c$) of the involved vital rates (Tuljapurkar 1982; Haridas & Tuljapurkar 2005). $V$ can be decomposed: $V = \sum_k c_{kk}^2 + H$, where $\sum_k c_{kk}^2$ is the sum of the variance contributions of all $k$ vital rates and $H$ the sum of the contributions of the covariances between all vital rates $k$ and $l$: $H = \sum_{k \neq l} e_{kl} \cdot c_{kl} \cdot \text{Corr}(k,l)$. A negative $H$ value means that the contributions of negative vital rate correlations outweigh those of the positive correlations, while positive $H$ values indicate the opposite pattern. Values of $H$ close to zero show that the contributions of negative and positive vital rate correlations are balanced. Negative $H$ values are expected if negative covariances among vital rates act as a mechanism significantly buffering temporal variation in stochastic population growth rates.

A negative relationship between the coefficient of variation ($c_k$) and elasticity values ($e_k$) (Morris & Doak 2004) is expected for survival and growth rates, because these rates range from 0 to 1 and have their highest variance around 0.5. However, as the random draws of vital rate values in our simulations were mean-dependent, spurious correlations are accounted for in the simulations: differences between the correlation patterns and the $c_k$–$e_k$ relationship in the data vs. those in the simulations must therefore be due to correlations in the data that are absent in the simulations. Based on this comparison, we identified a subset of 28 species in which the correlation of contributions to population growth (i.e. $H$) was not significantly different from zero. For these 28 species, we searched for evidence of the second buffering mechanism: that values of vital rates with relatively high deterministic elasticities are relatively less variable. Finally, we checked that the investigated parameters and resulting patterns were not related to the duration (i.e. the number of annual transition) over which the 40 species were studied (Appendix S4).

**RESULTS**

The first hypothesis we tested states that environmental variation is buffered by negative correlations between, e.g. reproduction and survival or growth rates. This would lead to negative $H$ values. We tested this hypothesis by comparing data on populations of 40 species with simulated datasets that mimicked the original dataset but in which the vital rates were varied independently around the observed means. We found that only 1 species (i.e. the relatively short-lived perennial monkeyflower *Mimulus cardinalis*, Angert 2006) of the 40 species had a significant negative covariance contribution to stochastic population growth (Fig. 1). In contrast, 11 species had significantly positive $H$ values. The latter was due to both positive correlations between different vital rate groups (reproduction, survival, growth) and positive correlations within each of the vital rate groups (Fig. 2b). Most other species did not have large negative within-group correlation contributions either (Fig. 2a).

The second hypothesis we tested states that natural selection has reduced the variability of those vital rates that are most important for population growth; the resulting negative relationship between $e_k$ and $c_k$ would reduce $V$ and increase growth rates (Pfister 1998). Plotting log-transformed coefficients of variation against log-transformed elasticity values indeed showed negative slopes (Fig. 3a–c): important vital rates had lower coefficients of variation than low-elasticity vital rates. In the simulated data, we expected such a pattern of negative slopes simply because of the mean-variance constraints (what Morris & Doak (2004) call spurious correlations). If the second hypothesis is correct, we expect the real data to show a significantly more negative correlation, but we found that the log $c_k$–log $e_k$ slopes did not differ between the original and the simulated data. The
Figure 2  Large correlation contributions are explained by positive correlations between vital rate groups (panels a and b). For each of the 40 species, $H$ was split into 2 values: on the $x$-axes the sum of the contributions of the correlations WITHIN vital rate groups (i.e. R–R, S–S and G–G), and on the $y$-axes the sum of the contributions of the correlations BETWEEN vital rate groups (i.e. R–S, R–G and S–G). R = reproduction, S = survival, G = growth. Note that large negative within-group correlation contributions ($x$-axis) do not occur at all, and that species with significantly positive $H$ values (panel b) also have mostly positive between-group correlation contributions ($y$-axis). Because the outlier in panel b is a relatively short-lived species, we corrected for life span in panel c by multiplying all values with the square of life span (see Supporting Information). Symbols and colouring as in Fig. 1: from short-lived (red) to long-lived species (blue).

Figure 3  $c_k$–$\ell_k$ and $V_k$–$\ell_k$ relationships are not significantly different from null model predictions. Relationships between coefficients of variation ($\log c_k$) and elasticity values ($\log \ell_k$) (panels a, b and c) and between variance contributions ($\log V_k$) and elasticity values (panels d, e and f). Solid lines are linear regressions, dashed lines represent regressions from the simulated datasets (using median regression coefficients from the 10 000 simulations). Correlation coefficients ($r$) and 95% confidence intervals (between brackets) of the slope are given in each panel. Only those 28 species for which $H$ was not significantly different from 0 were included in these analyses. Symbols and colouring as in Fig. 1: from short-lived (red) to long-lived species (blue).
lack of evidence for the second buffering hypothesis is also shown by the positive relationship between log $V_k$ and log $e_k$: no significant differences were found between the slopes in the original and simulated datasets for any of the three vital rate groups (Fig. 3d–f). Therefore, we conclude that there is no evidence in this large dataset that selection has lowered the variation in high-elasticity vital rates.

One pattern that is evident in our dataset as well as in other studies (Garcia et al. 2008; Morris et al. 2008) is that longer-lived species have more stable population sizes. The variability contributions (both $V$ and $H$) decrease in magnitude with life span (Fig. 3d–f, Supporting Information). However, that stability is not due to the operation of the two buffering mechanisms hypothesized here: life span did not explain how significantly the $H$ values (i.e. the contributions of covariances to the stochastic population growth rate) deviated from 0 (Fig. 1), nor did life span corrections qualitatively change the overall picture of the between- and within-vital-rate group contributions (compare Fig. 2b and c). Our results of increasing population stability with life span strengthen the conclusions of the study by Morris et al. (2008) on stochastic population growth rates of 15 plant and 21 animal taxa, because that study relied on estimated correlations while ours does not.

**DISCUSSION**

The demographic variation in the analysed datasets was likely caused by interannual climate fluctuations, both directly and indirectly through other ecosystem components such as other species and other abiotic factors. For example, winter harshness (as quantified by the number of days on which the temperature does not exceed 0 °C) significantly reduced survival and increased shrinking of *Asplenium scolopendrium* ferns, thereby having a large impact on population growth (Bremer & Jongejans 2010). In the cactus *Opuntia raietra*, annual rainfall was positively correlated with sexual reproduction and growth, but negatively with clonal propagation (Mandujano et al. 2001). Most of the studies, however, were too short to allow direct analysis of the link with climate and other environmental parameters.

Our analysis shows that, at the population level, many species appear to display no significant correlations between vital rates. In species where rates do appear to be correlated, positive rather than the hypothesized negative correlations predominate, by which not only growth but also survival and reproduction are favoured in good years and reduced in bad years, likely mediated through increases in plant size or plant quality (Reznick et al. 2000). It has often been noted that the negative trade-offs (e.g. increased reproduction at the cost of growth or survival) that may govern evolutionary change (Stearns 1989) are masked at the level of populations by variation in resource acquisition due to environmental fluctuations, which affects multiple vital rates jointly (van Noordwijk & de Jong 1986; Roff & Fairbairn 2007). Positive or negative correlations between vital rates may also simply reflect the independent responses of vital rates to the same climate variables, as shown by Knops et al. (2007) for growth and reproduction in oaks. We expect that vital rate correlations may play a more significant role in the dynamics of populations in extreme habitats where individuals are more stressed, or in ecosystems with infrequent but large disturbances like fires or hurricanes. In more common, stable habitats, as represented in our dataset, trade-offs do not seem to be important.

We also did not find evidence for the second hypothesis: coefficients in vital rates with high elasticity were not lower than what was expected from our random null model. This is consistent with findings of an analysis of coefficients of variation scaled to their maximally possible values (sensu Morris & Doak 2004) applied within vital rates of a wide range of species (Burns et al. 2010), although covariances are ignored in analyses of scaled coefficients of variation. Lack of evidence for reduced variation in important vital rates might theoretically be expected in particular cases in which plant traits do not linearly change with an environmental driver. If there is a convex relationship, increased environmental fluctuations might not only increase vital rate variability but also the mean of a vital rate (Koons et al. 2009). If this positive effect on the mean is large enough to overcome the detrimental effect of increased vital rate variation on the stochastic population growth rate, decreased vital rate variation might not be expected to be selected against after all. However, it is still unclear whether this phenomenon is common or not, and it would probably only play a potential role in short-lived species. For very long-lived species, we might not see reduced variation of important vital rates because the costs of vital rate variation for stochastic population growth are generally low due to their long life span. However, our results suggest that selection for reduced variability specifically in high-elasticity vital rates is undetectable over the entire range of life spans of the studied species.

The environmental (abiotic and biotic) fluctuations experienced by the 40 plant species in our study resulted in a predominance of positive vital rate covariances. It is likely that such species in relatively stable habitats will increasingly experience such effects as climate fluctuations are predicted to continue to increase. Furthermore, mult trophic species interactions are disrupted due to asynchronous climate tracking of different trophic levels (Tylianakis et al. 2008), indirectly leading to larger population fluctuations and higher extinction risks (Schmitz et al. 2003; Ims & Fuglei 2005). Clearly, more research is needed to elucidate the mechanisms through which species respond to climate fluctuations in their ecosystem setting (Keith et al. 2008).
For instance, a possible buffering mechanism not tested in this study is lagged (between-year) negative vital rate correlations. To determine, whether lagged correlations matter to buffering, we need to answer several questions. First, given an observed time series of vital rates, how do we evaluate the pattern and magnitude of serial correlation? Second, how can we disentangle the relative importance of environmental drivers and (lagged) life history correlations? Third, how do we use theory on temporal autocorrelation (Tuljapurkar & Haridas 2006; Tuljapurkar et al. 2009) to construct an extension of the kind of test that we have developed and applied in this paper to include lagged correlations? These are substantial questions. We realize that our current analyses and conclusions are conditional on the absence of significant effects of lagged correlation (although 1-year-lagged correlations between vital rates are equally often positive as negative in the studied datasets; E. Jongejans, H. de Kroon, S. Tuljapurkar and K. Shea, unpublished data). Nonetheless our analyses are a useful advance that allows us to examine the magnitude and effects of within-year correlations. In future work, we plan to assemble and analyse the kinds of long-term data that will make it possible to study the effects of lagged correlations as well. Combinations of such modelling studies with analyses of long-term demographic datasets promise to generate new insights into the population-level role of individual-level trade-offs and multi-species interactions, for instance by quantifying novel interaction terms in the stochastic population growth ($log\ \lambda$) equation that formulate how environmental drivers influence individual trait and vital rate relationships.

The importance of positive covariance contributions and the lack of buffering of important vital rates imply that plant populations track rather than buffer climate fluctuations. This rather passive tracking of year-to-year climate and other environmental fluctuations by populations is different from the more active tracking by individuals of seasonal shifts within years caused by global warming. Such climate-induced seasonal shifts have been shown for, e.g. the onset of flowering in many plant species (Fitter & Fitter 2002; Walther et al. 2002) and the egg laying date of birds (Nussey et al. 2005; Charman-tier et al. 2008). While active tracking of seasons seems to be a strategy for individuals to make the most of a particular year, it is much more difficult to respond to increases in unpredictable fluctuations in, e.g. rainfall and temperature than to gradual and consistent seasonal shifts. There is thus a need for new theory and data that link individual fitness decisions (e.g. related to either season-tracking or delaying reproduction in unfavourable years) to extinction risks associated with fluctuating population sizes. Our analyses suggest that processes like season-tracking may not effectively ameliorate the impact of an unpredictably varying climate on population size fluctuations across years.

Climate tracking, as opposed to climate buffering, is another factor negatively affecting the survival of species under global climate change. Unfortunately, population viability analyses (PVA) do not yet properly include the impacts of increased climate fluctuations on extinction risks. Including ecological drivers and model uncertainties in PVAs is complex, and few sophisticated attempts (Smith et al. 2005; Bakker et al. 2009) exist. However, these few examples show the great potential for future studies to incorporate the processes of climate tracking mechanistically, and hence to unravel how much increased climate variability adds to extinction risks. Fluctuating climate drivers will likely affect individual traits and vital rates non-linearly both directly, and indirectly through other traits (Drake 2005; Koons et al. 2009). These effects of climate variability also will need to be investigated in the context of the problems that many species already face: invasive species, habitat losses, habitat fragmentation and range shifts due to changes in mean climate parameters (Walther et al. 2002; Thomas et al. 2004; Thuiller 2007; Thuiller et al. 2008). Our results suggest an apparent inability of plant populations to buffer climate fluctuations, and we therefore predict that increased climate fluctuations will interact importantly with other global change threats to plant species.

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REFERENCES


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**SUPPORTING INFORMATION**

Additional Supporting Information may be found in the online version of this article:

- **Appendix S1** Demography dataset and simulation methods.
- **Appendix S2** Testing the robustness of the results to different randomization methods.
- **Appendix S3** Small-noise approximation.
- **Appendix S4** Robustness of the results to study duration.

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