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REVIEWS

Review

The Demographic Buffering Hypothesis: Evidence and Challenges

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In (st)age-structured populations, the long-run population growth rate is negatively affected by temporal variation in vital rates. In most cases, natural selection should minimize temporal variation in the vital rates to which the long-run population growth is most sensitive, resulting in demographic buffering. By reviewing empirical studies on demographic buffering in wild populations, we found overall support for this hypothesis. However, we also identified issues when testing for demographic buffering. In particular, solving scaling problems for decomposing, measuring, and comparing stochastic variation in vital rates and accounting for density dependence are required in future tests of demographic buffering. In the current context of climate change, demographic buffering may mitigate the negative impact of environmental variation and help populations to persist in an increasingly variable environment.

Demographic Buffering of Vital Rates

Climate change is expected to severely impact most ecosystems. Alongside the changes in mean values, temporal variation in temperature, precipitation, and wind speed also increases, leading to more frequent occurrences of extreme weather events [1,2]. How species will respond to these changes depends on both the mean and the variance of these new environmental conditions [3,4] and their effects on the population growth rates [5]. The population growth rate is a direct function of the **vital rates** (see Glossary) that are influenced by individual attributes, such as sex or size, as well as by **environmental fluctuations** [6,7]. A key result from stochastic modeling of population dynamics is that environmental fluctuations that increase the variance of vital rates usually decrease the **stochastic long-run growth rate** of populations [8,9], increasing their extinction risk [10,11]. This was first demonstrated 50 years ago by Lewontin and Cohen [12], who showed that, for unstructured populations, the logarithm of the **geometric mean** of annual population growth rates provides a more accurate estimate of the long-run growth rate than the logarithm of the arithmetic mean, because population growth is a multiplicative process. Using the logarithm of the arithmetic mean of annual population growth rates ($\ln \lambda_1$) would overestimate the long-run growth rate increasingly with increased variance in annual population growth rates (Λ). Thus, assuming that the annual population growth rates are **identically and independently distributed**, decreasing $\ln \lambda_1$ or increasing variance in the annual population growth rate (σ_Λ^2) both decrease the long-run growth rate $E(\ln \Lambda)$ (Equation 1):

$$E(\ln \Lambda) \cong \ln \lambda_1 - \frac{\sigma_\Lambda^2}{2\lambda_1^2}. \qquad [1]$$

Tuljapurkar [13] showed that, for large populations neither subjected to **density dependence** nor influenced by **demographic stochasticity** and with small temporal variation in vital rates [i.e., coefficient of variation (CV) <<1] the stochastic long-run population growth rate, a, is negatively affected by the temporal variance of the vital rates as well as by the covariance among

Highlights

Climate change is expected to increase the environmental variation of ecosystems on Earth, highlighting the need to understand how populations will respond to these new environmental conditions.

The demographic buffering hypothesis is derived from classical models of population dynamics. It predicts selection for a reduction in variance of the vital rates with the strongest influence on population growth and individual fitness.

We review current knowledge about demographic buffering and critically assess the various methods and results published so far.

A pattern of reduced variation in the most influential vital rates emerges from the review.

Differences in methodology highlight the need for further studies with standardized methods to reveal whether the observed pattern is a direct result of selection for lower variation of the most influential vital rates.

Modern statistical methods that allow decomposing variation in population growth rate into environmental variation, density dependence, and demographic stochasticity can stimulate the search for improved models to predict the effect of increasing environmental variation on population dynamics and life-history evolution.

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them (positively for negative covariance, Box 1). Ignoring for now covariances among vital rates and **environmental autocorrelations** that also affect the stochastic growth rate [14], the negative effect of temporal variation in vital rates depends on their **sensitivity** (Box 1, Equations I and II). Thus, the vital rates with the highest sensitivities are expected to be under strong selection for a reduction in temporal variation [15], while those with lower sensitivities can vary more freely or even be selected to vary (**demographic lability**) in response to changes in environmental conditions. Therefore, we expect a negative correlation between the sensitivity of vital rates and their temporal variation, a pattern termed '**demographic buffering**' [16].

In the context of climate change, demographic buffering provides a clear framework based on life-history theory to link temporal variation in vital rates and Malthusian fitness (i.e., the long-run population growth rate [17,18]). If present, demographic buffering may allow populations to persist in a changing environment by reducing the effects of variation in environmental conditions on the long-run growth rate (Box 1). Several studies have investigated the demographic buffering hypothesis, but evidence supporting its existence in the wild remains ambiguous. The inconsistent results across studies could originate from the complex biological mechanisms involved in demographic buffering or from the use of different methods and metrics to assess it.

Here, we provide a comprehensive review of the published studies that have examined the demographic buffering hypothesis. We present the different methods used for testing this hypothesis and identify scaling problems associated with these tests. In some cases, demographic buffering is not expected to occur and we discuss to what extent the species-specific life history, population size, and the functional relationship between vital rates and their environmental drivers enable the prediction of whether demographic buffering should occur. Testing for demographic buffering

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Box 1. Population Growth Rate and Demographic Buffering

Assuming small variation and no temporal autocorrelation in vital rates, Tuljapurkar [13] showed that the long-term stochastic growth rate a of a large population not subjected to density dependence could be approximated using Equation I as:

$$a \approx \ln\lambda_1 - \frac{1}{2\lambda_1^2}\left[\sum_{i,j}\mathrm{Var}(A_{i,j})s_{i,j}^2 - \sum_{i,j\neq k,l}s_{i,j}s_{k,l}\,\mathrm{cov}(A_{i,j},A_{k,l})\right], \qquad [\mathrm{I}]$$

where λ_1 is the dominant eigenvalue of the average vital rate matrix A, $\mathrm{Var}(A_{i,j})$ is the variance of the different **projection matrix** elements (vital rates) in row i and column j, $s_{i,j}$ is the sensitivity $\left(\frac{\delta\lambda_1}{\delta A_{i,j}}\right)$ of λ_1 to a given change in the mean vital rate $A_{i,j}$ and $\mathrm{cov}(A_{i,j},A_{k,l})$ is the covariance between the different matrix elements. Given that the difference between the terms between brackets in Equation I corresponds to the variance in annual population growth rates, the variance component is larger than that of the covariance ([86] p. 93–94, [75] p. 397). Because sensitivities correspond to the absolute change in λ_1 for an absolute change in a vital rate [87], it is difficult to interpret the relative contribution of the variation in each vital rate to changes in λ_1 when the vital rates are on different scales. Elasticities $\left(\frac{\delta\log\lambda_1}{\delta\log A_{i,j}}\right)$ [88]) enable the comparison of the impact of variation in vital rates on population growth rate on a common (proportional) scale (but see Box 2 in the main text). Using elasticities, a becomes a function of the CV of the matrix elements and their corresponding elasticities [13] (Equation II),

$$a \approx \log\lambda_1 - \frac{1}{2}\left[\sum_{i,j}e_{i,j}^2 CV_{i,j}^2 + \sum_{i,j\neq k,l}e_{i,j}e_{k,l}\frac{\mathrm{cov}(A_{i,j},A_{k,l})}{A_{i,j}A_{k,l}}\right], \qquad [\mathrm{II}]$$

where CV^2 is calculated as the variance divided by the mean square of the vital rate. Maximization of the long-term growth rate, a, can be achieved by increasing the mean growth rate λ_1 or by reducing the variance in the growth rate. The demographic buffering hypothesis addresses the second option, that is, reducing $\sum_{i,j}e_{i,j}^2 CV_{i,j}^2$ or $\sum_{i,j}\mathrm{var}(A_{i,j})s_{i,j}^2$. This represents an explicit prediction of the demographic buffering hypothesis that can be quantitatively tested by assessing whether the relationship between the squared sensitivity and the variance across vital rates (or the relationship between the squared elasticities and CV^2) has a negative slope [20]. Serial correlations of vital rates between years could have either a positive or negative effect on a [14,67].

requires reliable estimates of how temporal variation in vital rates is affected by fluctuations in environmental variables. Thus, we also identify and discuss the methodological difficulties that can influence the estimation of the temporal variation in vital rates, and thereby the outcome of any test of demographic buffering. Finally, we suggest approaches to solve these difficulties and provide recommendations and guidelines on how to improve the test of the demographic buffering hypothesis in future studies.

Review of Empirical Evidence

The demographic buffering hypothesis has been tested empirically in mammals [19–22], birds [23,24], reptiles [25], and plants [16,26–28] (see Table 1 for explicit tests directly performed on temporal variation in vital rates and the supplemental information online for studies that support the buffering hypothesis without explicitly testing it), but the empirical evidence accumulated so far is equivocal. Even though there is general support of the demographic buffering hypothesis, several studies did not detect the expected negative relationship (e.g., [27,29]), or they found results ranging from negative to positive relationships (demographic lability) [28], between the temporal variation in vital rates and their sensitivity or **elasticity**. Although these results may reflect a true absence of demographic buffering in some species, they may also stem from various difficulties associated with the test of this hypothesis.

The results reported in Table 1 show that the presence of demographic buffering is generally examined by testing for a negative correlation between year-to-year variance of vital rates and their squared sensitivity, or between the squared CV (CV^2, i.e., the variance divided by the squared mean) and squared elasticity (Box 1 and Figure 1). This negative relationship is sometimes tested using rank-based tests in which only the sign of the relationship is assessed (e.g., Spearman correlations [15,26]). Although rank-based statistics make the tests less sensitive to the scaling of the variables (e.g., square transformation of the sensitivity), they only represent qualitative assessment of the hypothesis because they enable the identification of the presence of demographic buffering without providing any information about its strength. Quantitative assessments involve estimating the slope (i.e., the strength of demographic buffering) of the relationship between the variance and the squared sensitivity (or squared elasticity and CV^2) of the vital rates (e.g., [20,27]) (Table 1).

The information reported in Table 1 also reveals that, besides these statistical considerations, studies on demographic buffering also vary in several aspects, from the type of temporal variance estimated (e.g., type of vital rates included and whether they account for demographic stochasticity and sampling variance), the type of correction and/or transformation applied to the variance estimates, to the type of tests performed (e.g., comparing different vital rates measured in different species versus comparing a single vital rate across species or across age classes or developmental stages within a single species). Some of these differences stem from the type of data available to test the hypothesis, but others reflect specific decisions taken by the authors to overcome the difficulties associated with the test of the demographic buffering hypothesis. Here, we present these difficulties and their consequences.

When to Expect Demographic Buffering

In fluctuating environments, natural selection is expected to maximize the long-run growth rate of a population [18,30]. The long-run growth rate can be maximized through three main pathways: (i) increasing the mean value of vital rates with a positive effect on λ_1, or decreasing the mean if the effect is negative; (ii) reducing the temporal variances and/or covariances when positive among vital rates; or (iii) exploiting variation in vital rates [7,31] (see [32] for a theoretical study showing how, at high levels of environmental variation, a mutant with negative correlations among fertilities

Glossary

Allee effect: decrease in the per capita rate of population growth at low population densities or small population sizes (also called positive density dependence).

Demographic buffering: selection against temporal variation (caused by variation in environmental conditions) in the vital rates with the strongest demographic impact (measured by sensitivity or elasticity) on the population growth rate. Demographic buffering does not make any assumption about the shape of the relationship linking the focal vital rate and environmental variation; neither does it make any assumption about the shape of the relationship linking the focal vital rate and environmental variation.

Demographic lability: selection for fluctuating vital rates in response to variation in environmental conditions. Demographic lability only occur when the relationship with environmental variation is convex (positive second derivative) and generally targets the less influential vital rates for the population growth rate, making lability and buffering likely to act synergistically on different vital rates.

Demographic stochasticity: random within-year variation in reproductive success or survival among individuals. Demographic stochasticity is more influential at low population size.

Density dependence: negative association between the population growth rate and the population size in the current or a previous year (also called negative density dependence or population regulation). Density dependence is mostly observed at high population densities.

Elasticity (of a vital rate): proportional incremental change in the population growth rate in response to a proportional incremental change in a vital rate.

Environmental autocorrelation: influence of environmental conditions on the vital rates affecting subsequent time step(s) and/or correlated environmental conditions among time steps.

Environmental fluctuations: variation in environmental conditions over time that may cause temporal variation in vital rates.

Environmental stochasticity: temporal variation in vital rates due to stochasticity in environmental conditions (e.g., temperature or precipitation) that affects all the

can invade a population comprising individuals with identical average fertilities that are independent or positively correlated). The demographic buffering hypothesis specifically targets the second pathway and predicts that, in the absence of density dependence, selection should reduce the susceptibility of vital rates to environmental variation, leading to less temporal variation in the vital rates for which a given change most strongly affects the population growth rate [15] (Box 1). However, the functional relationship linking environmental drivers and vital rates, as well as species-specific life histories and population-specific environmental conditions, are all likely to affect the amount of temporal variation in vital rates and, therefore, whether demographic buffering should be expected to occur.

Functional Relationship between Environmental Drivers and Vital Rates

To understand the consequences of variation in environmental drivers on the temporal variation in vital rates and, in turn, on the stochastic population growth rate, it is necessary to identify the shape of the relationship between environmental drivers and vital rates [4,33]. In particular, when the effect of an environmental driver on the vital rate is convex, increasing temporal variation in this vital rate at low values can increase its mean and thereby increase the stochastic growth rate [4], leading to selection for higher variance of this vital rate (demographic lability) [34]. However, this positive effect of temporal variation in a vital rate on the long-run population growth rate is more likely to occur when variation in another vital rate is constrained (i.e., buffered) [35]. For example, in long-lived iteroparous species, a high and constant adult survival rate can increase environmentally induced temporal variation in reproduction or juvenile survival through the occurrence of reproductive pauses or decreased offspring survival during poor years [36]. When reproductive costs are mostly paid in terms of future reproduction, as is the case in long-lived mammals [37] and birds [38], temporal variation in reproductive traits should increase. Thus, selection is more likely to favor buffering on one rate and lability on another [35]. Even though demographic lability can be important, as Barraquand and Yoccoz pointed out [35], it is not an alternative to demographic buffering for coping with environmental variation. Rather, demographic lability of a vital rate is likely to be observed when there is buffering of another vital rate. Few studies to date have investigated demographic lability, but a recent paper by McDonald *et al.* [28] found widespread evidence for either demographic buffering or lability in a range of plant species. However, this work did not distinguish between buffered and labile vital rates within the same species. This distinction is necessary and may be a promising avenue for further research [35].

Life-History Strategies and Demographic Buffering

Traditionally, life-history strategies have been ranked along an axis of variation from slow to fast life histories, where slow species are characterized by long lifespan, late age at first reproduction, and low fecundity, while fast species are characterized by the opposite strategy. This slow–fast continuum explains the largest amount of life-history variation observed in birds and mammals [23,39–41]. However, a substantial amount of life-history variation still exists for a given position on the slow–fast continuum. A second axis of variation often involves variation in reproductive tactics, such as ranking along the altriciality–precociality or semelparity–iteroparity continuums, as reported in mammals [39,42], birds [40], and plants [43]. Although the presence of demographic buffering does not depend on the species position along these axes of life-history variation, the pace of life of a given species (i.e., its position on the slow–fast continuum) enables the identification of which vital rates should be buffered. In slow-living species, such as large mammals and seabirds, population growth rates are mostly sensitive to changes in adult survival, whereas, in fast-living species, population growth rates are more sensitive to changes in fecundity or juvenile survival [23,44]. Consequently, adult survival should be buffered in slow-living species, while fecundity or early survival should more often be buffered in fast-living species (generation time <2 years for species with annual reproduction [44]). This prediction was tested by Gaillard

individuals or groups of individuals in a population.

Geometric mean: mean of a set of products. Defined as the Nth root of the product of n values (instead of as the sum of the n values divided by n for the arithmetic mean). Both means are equal when the values are constant over time. The geometric mean is increasingly smaller than the arithmetic mean with increasing variance in values.

Identically and independently distributed (IID): a collection of random variables (e.g., vital rates in different years) that have the same probability distribution and are independent from each other. For vital rates, it implies that they do not display any temporal trend or autocorrelation.

Projection matrix: Leslie (for age-structured populations) or Lefkovitch (for stage-structured populations) matrix that contains estimates of (st)age-specific vital rates and describes the transition of the population from time t to time t+1. The matrix can be built from a prebreeding or postbreeding census.

Sensitivity (of a vital rate): absolute incremental change of population growth rate λ in response to an absolute incremental change in a vital rate.

Stochastic long-run population growth rate: mean population growth rate over time on a logarithmic scale in a stochastic environment in the absence of demographic stochasticity and density dependence. It can be calculated for an age-structured population using Tuljapurkar's approximation based on the mean values, variances, and covariances among vital rates (see Box 1 in the main text).

Vital rate: element or a component of an element of the Leslie/Lefkovitch matrix (i.e., age-specific survival and fecundity or stage-specific transition rates).

Table 1. Review of Studies Published on Demographic Buffering[a]

Author (year)	Taxonomic group(s)	Species	N_{years} (min–max)	Vital rates	Scale correction (Y/N)[b]	Variance measure and statistical test	Test statistic	Senescence correction[c]	Corrected for σ_d or σ_s[d]	Results
Pfister (1998) [15]	Plants and animals	20 species, 30 populations	2–23	Survival, fecundity, growth, shrinkage	N	S versus Var ($\chi^2_{60} = 145.73$, $P <0.001$) (negative correlation); E versus CV ($\chi^2_{60} = 194.75$, $P <0.001$) (negative correlation)	Spearman correlation and partial correlation coefficients	N	No information	'…stage of the life cycle with the highest variance generally has the lowest effect on population growth rate λ'
Sæther and Bakke (2000) [23]	Birds	15 species	≥10	Survival, fecundity	N	S versus SD ($r = -0.72$, $n = 18$, $P <0.001$)	Linear regression	N	No	'there was an inverse relationship between the.. (its elasticity) and its relative variability'
Gaillard and Yoccoz (2003) [20]	Mammals	14 ungulate species, 22 populations	NA	Survival	Y^1	$log\left(\frac{S_{ad}}{S_{juv}}\right)$ vs. $log\left(\frac{Var_{ad}}{Var_{juv}}\right)$ ($r = -0.474$, slope $= -1.001 \pm 0.372$, $P = 0.013$)	Linear regression	Y^1	σ_s for some species	'…a negative covariation occurs among mammals between the demographic sensitivity of fitness components and their sensitivity to environmental variation…'
Pico et al. (2003) [55]	Plants	Hypericum cumulicola 13 populations	6	Seed survival, recruitment, and production; seedling production; plant survival	N	E versus CV: statistically significant negative relationship in 8 of 13 populations	Spearman correlation	N	NA	'The relationship between elasticity and temporal variability in vital rates was significant in 8 of 13 H. cumulicola populations'
Morris and Doak (2004) [16]	Plants	Silene acaulis; 5 populations	5	Reproduction, stasis, growth, reversion	Y^1	S versus Var, negative slopes; E versus C, negative slopes	Spearman correlation and quantile regression	N	σ_s	'…all 5 of the rank correlations between the relativized variances and the sensitivities of the 0-to-1 vital rates were significantly negative'
Doherty et al. (2004) [90]	Birds	Phaeton rubricauda (red-tailed tropic bird)	16	Survival, reproduction, P_{breed}	Y^2	Sin^{-1}-scaled Var versus sin^{-1} sensitivity ($r = -0.31$, $P = 0.49$); Log(s) versus var ($r = 0.03$, $P = 0.95$)	Pearson correlation	N	σ_s	'Results did not support our predictions that the variables to which λ was least sensitive…would be the variables most affected by El Niño events'
Ezard et al. (2006) [109]	Birds	Sterna hirundo (common tern)		Survival	N	E versus SD: females: $F_{1,6} = 18.75$, $P <0.01$, $R^2 = 0.72$; males: $F_{1,6} = 15.95$, $P <0.01$, $R^2 = 0.68$	ANOVA	Y	N	'There was a significant negative relationship between a demographic rate elasticity and the rate's variance'
Delean (2007) J.S.C. Delean. PhD thesis. James Cook University, 2007	Mammals	Petrogale assimilis (rock wallaby)	11	Survival, fertility, transition rates, proportion births	Y^1	S versus Var ($\rho = -0.65$, $P = 0.05$); E versus CV ($\rho = -0.34$, $P = 0.007$)	Spearman correlation	Y	σ_s	'there was a significant negative rank-order correlation between the sensitivities and variances of the vital rates…'
Forcada et al. (2008) [19]	Mammals and birds	2 seal and 3 bird species	16–39	Survival, breeding propensity	Y^1	E versus CV: slopes $= (-0.4, -0.1, -0.5, -0.5, -0.3, -0.5)$; P-values ($<0.01$, 0.4, <0.02, <0.05, <0.1, <0.1)	Quantile regression	N	No information	'…Antarctic fur seals have lost life history buffering to increasing environmental variability'; '…support of life history buffering in the other species…'

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Table 1. (continued)

Author (year)	Taxonomic group(s)	Species	N_{years} (min–max)	Vital rates	Scale correction (Y/N)[b]	Variance measure and statistical test	Test statistic	Senescence correction[c]	Corrected for σ_d or σ_s[d]	Results
Schmutz (2008) [24]	Birds	59 species, 62 populations	5–42	Survival	Y[1,2]	E versus CV: arcsine: slope = –0.905 ± 0.164, r^2 = 0.29; relative CV: slope = –1.439 ± 0.272, r^2 = 0.318	Linear regressions	N	σ_s	'…in an analysis of 62 populations, I confirm her (Pfister's) prediction by showing a negative relationship between the proportional sensitivity (elasticity) of adult survival and the proportional variance (CV) of adult survival'
Burns et al. (2010) [77]	Plants	185 iteroparous and 19 semelparous species	NA	Survival, fecundity	Y[1]	No significant correlation between log(CV) and log(S) or log(E)	Generalized least squares	N	CV with bias correction for small N	'…the direction of the trends for the correlation between the CV and the sensitivity for fecundity and survival were negative in some cases…these relationships were not statistically significant…'
Jongejans et al. (2010) [27]	Plants	40 species	3–21	Survival, fecundity, growth	N[1]	No difference in slope of log (CV) versus log(E) between observed and simulated data	Linear regression	N	No information	'…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'
Miller et al. (2011) [25]	Reptiles	*Thamnophis elegans* (garter snakes); 2 populations	7-13	Survival, litter size, proportion gravid	Y[1]	E versus CV (r = –0452, P = 0.01)	Pearson correlation	N	σ_s	'Our results…support for the buffering hypothesis'; '…we show that life-history differences among populations of a single species are consistent with buffering'
Morris et al. (2011) [66]	Mammals	6 primate species	22–45	Survival	Y[1]	No statistical test. Var_{ad}/Var_{juv} >1 in 4 of 6 species	No test	N	σ_s	'We did observe this result in 4 of 6 primate species…'; '…the other 2 did not show the predicted higher variability in newborn survival'
Reed & Slade (2012) [21]	Mammals	Four rodent species	8–25	Survival, fecundity	Y[1]	E versus CV (r = –0.17, P = 0.05); (r = –0.19, P = 0.04); (r = –0.34, P <0.01); (r = –0.15, P = 0.2)	Pearson correlation	N	σ_s	'We found significant negative relationships between vital rate elasticity and the CV of the vital rate in 3 populations…'
Rotella et al. (2012) [22]	Mammals	*Leptonychotes weddellii* (Weddel seal)	28	Survival, fecundity	Y[1]	S versus Var (ρ = –0.89, P <0.001); VSS versus Var (ρ = –0.78, P <0.001)	Spearman correlation	N	σ_s	'In support of the buffering hypothesis… greater temporal variation in breeding probability than in survival…whereas λ_1 was more sensitive to changes in survival…'; 'Results of correlation analyses were also in keeping with the prediction…'
Jäkäläniemi et al. (2013) [29]	Plants	*Silene tatarica*, 24 populations and *Erigerion acrisr*, 17 populations	3–8	Survival, fecundity, growth, retrogression	Y[1]	S versus log(CV) (r = –0.217, P = 0.034); (r = –0.479, P <0.0001; E versus CV (r = –0.166, P = 0.1); (r = 0.117, P = 0.342)	Spearman correlation	N	σ_s	'…observed an overall inverse relationship between the importance and temporal variation of average main vital rates…'; '…more likely to indicate a methodological artefact rather than demographic buffering'
Koons et al. (2014) [110]	Birds	13 species of waterfowl (data from [24]	NA	Survival	Y[1]	E versus CV: slope = NA, P >0.10	NA	N	σ_s	'Among waterfowl, currently available data for temporal variation in adult survival do not support the DB hypothesis'

Reference	Taxon	Study system	No.	Vital rates[a]	Scale correction[b]	Test detail	Statistical test	Senescence correction[c]	Corrected variation[d]	Conclusion
Li and Ramula (2015) [26]	Plants	23 populations of 4 woody and 4 populations of perennial herbs	3–5	Survival, fecundity	Y^1	S versus Var and E versus CV; no correlation coefficients reported	Spearman correlation	N	N	'…populations of woody invaders exhibited buffering regardless of the method…'; '…for the populations of herbaceous species, deterministic calculations suggested buffering and stochastic…suggested lability'
Elderd and Miller (2015) [111]	Plants	Tree cholla cactus (*Opuntia imbricate*)	10	Survival, growth, probability of flowering, fertility	NA	S versus Var: no information on test	NA	N	σ_s	'Vital rate sensitivities were generally negatively correlated with their spatial and temporal variances such that higher-sensitivity vital rates exhibited lower variability'
Bjørkvoll *et al.* (2016) [62]	Mammals	*Rangifer tarandus platyrhynchus* (Svalbard reindeer)	18	Survival, fecundity	$Y^{1,3}$	Logit: no buffering; Log(E) versus CV: full age structure (r = −0.57, −0.61, −0.66, all $P<0.003$); aggregated age-classes: (r = −0.22, P = 0.25)	Linear regression (logit)/Spearman (relative CV)	Y	σ_d, σ_s	'…demographic buffering was not required to explain the observed negative relationships between CV and ln(E)'; '…using relativized CV as a measure of variation gave some support for the demographic buffering hypothesis'
Chantepie *et al.* (2016) [112]	Birds	*Gyps fulvus* (Griffon vulture)	32	Survival	Y^1	High survival and low variance in mid-age vultures compared with juveniles and senescents	No test	Y	σ_s	'In agreement with…the buffering hypothesis…found high survival probabilities and low temporal variance in the survival of mid-age…survival rates in juvenile and old…were lower and more variable…'
McDonald *et al.* (2017) [28]	Plants	COMPADRE data set, 73 species, 141 populations	NA	Unspecified demographic rates	Y^3 and Y^4	67.4% of species with negative correlations	Spearman correlation	N	No information	'We have…revealed a continuum of demographic strategies…from demographic buffering to demographic lability'

[a]Vital rates: growth, probability of transition to next age/stage class; shrinkage/reversion/retrogression, probability to revert to previous stage class; stasis, survival without growth; breeding propensity, probability of breeding after having bred the previous year; P_{breed} = probability of breeding.

[b]Scale correction: Y^1, 'amount of possible variance/relative variance', Var/Var_{max} or CV/CV_{max}. Y^2, Arcsine scaling, $q(\mu) = \sin^{-1}(\sqrt{\mu})$ [91]. Y^3, Logit scaling, $q(\mu) = logit(\mu) = \ln(\mu/1-\mu)$. Y^4, Log scaling. Variance measure: VSS, Variance stabilized sensitivity.

[c]Senescence correction: Y^1, only prime-aged individuals considered.

[d]Corrected variation: σ_d, demographic stochasticity, σ_s, sampling variance.

Collect
data

Vital rates

Create
matrix

Estimate variance and
sensitivity or elasticity

Test for buffering

(A)

Individual-level
data

Population-
level data

(B)

Survival

Fecundity

Projection
matrix

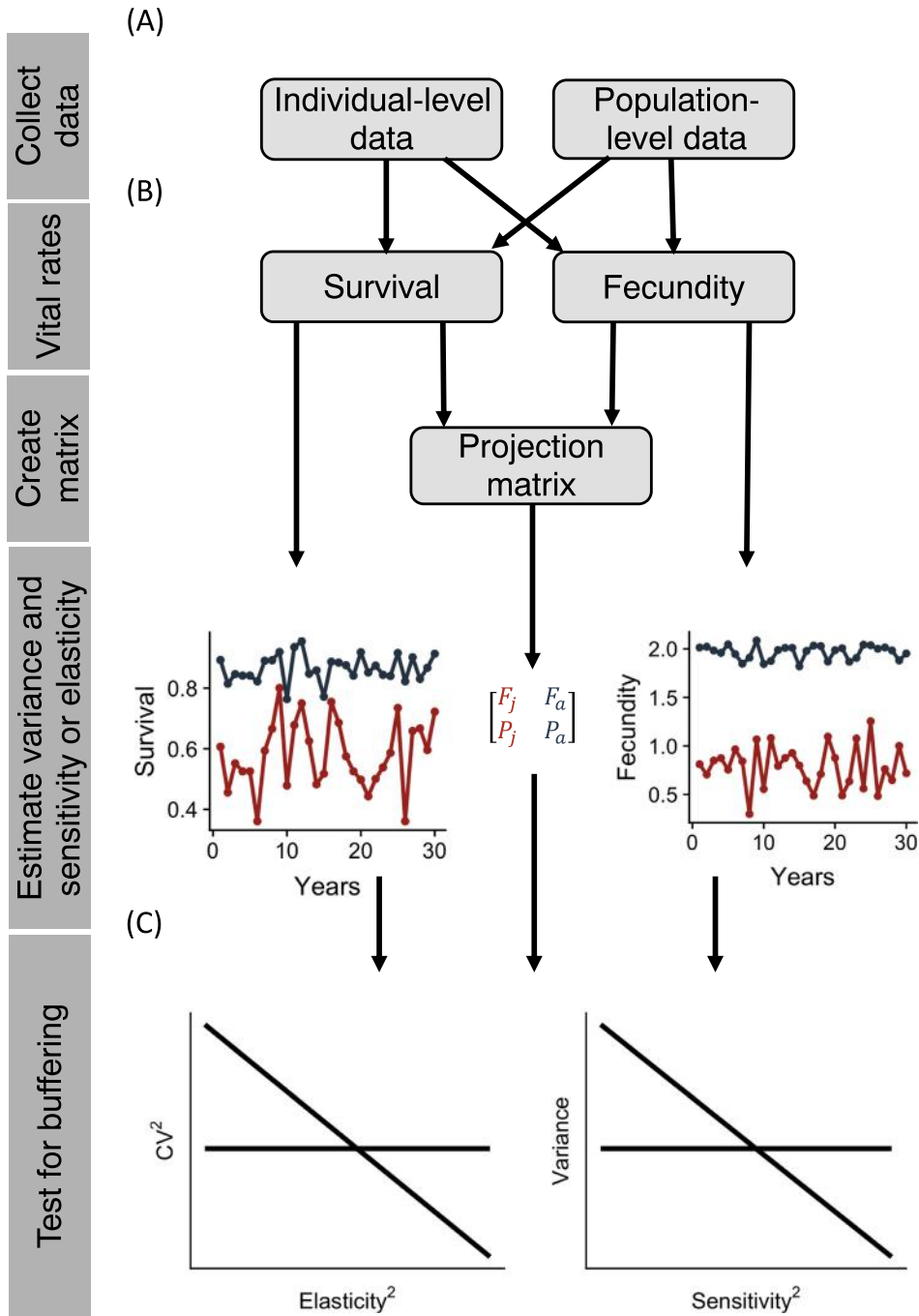$$\begin{bmatrix} F_j & F_a \\ P_j & P_a \end{bmatrix}$$

(C)

Figure 1. Visual Framework for Demographic Buffering Analyses. (A) Population-level data (e.g., counts) and/or in-dividual level data [e.g., capture–recapture (CR) data and population productivity data] are used to calculate the vital rates: (st)age-specific survival and fecundity. (B) Annual estimates of the vital rates (F = realized fecundity (defined as the number of female offspring surviving to 1 year of age), P = probability of survival) can be used to calculate temporal variance and/or CV (taking demographic stochasticity into account) for each age or stage class (e.g., juveniles j in red and adults a in blue) and to construct the projection matrix. The projection matrix is used to calculate the sensitivities or elasticities of the popula-tion growth rate to changes in the different age- or stage-classified vital rates. (C) The estimated sensitivities^2 or elasticities^2 are regressed against the estimated temporal variances or CV^2 to test for demographic buffering.

and Yoccoz [20], who found lower temporal variance in juvenile survival relative to variance in adult survival in fast-living mammals compared with slow-living ones across 27 mammalian populations. By contrast, Reed and Slade [21] found that the populations of four species of rodents with fast life histories were mostly sensitive to monthly changes in adult survival [21]. While the first study only considered univoltine species, the latter considered multivoltine species with several generations per year and used month as timescale unit. This change of timescale for multivoltine species makes the species demography similar to the annual demography of slow-living species, where survival should be more buffered than fecundity [45]. Given that natural selection acts on the timescale of biological events, comparing fast- and slow-living species in terms of demographic buffering should be performed at a standardized timescale (i.e., month or year for both types of life history).

Interestingly, evidence is accumulating in both animals and plants that short-term demographic responses to environmental disturbances, also called transient dynamics, are shaped by the position of the species along the slow–fast continuum. Several metrics have been proposed to measure transient dynamics, such as the damping ratio, to assess the time taken to return to the stable population structure after a disturbance, or some amplification/attenuation index to evaluate the largest/smallest possible density reached after a disturbance on the short term [6,46]. Analyses of transient dynamics have revealed that species with a slow pace of life often exhibit short-term variation in population size of low magnitude compared with fast species, indicating that population size is buffered against environmental variation in slow species [47–49].

Population Density and the Strength of Demographic Buffering

Population density, and particularly how close the population size is to the carrying capacity (K), also influences the demography of a population. Because elasticities of the population growth rate to changes in vital rates depend on how close a population is to K [50,51], the relative population size (measured as N/K) might give information on the strength of selection against variation in a focal vital rate in a given population. Accordingly, Sæther *et al.* [51] showed that the elasticity of fecundity was negatively correlated with population size, while the elasticity of survival increased with population size across 13 bird species.

Furthermore, when populations are close to K, the life cycle generally slows down and the elasticity of survival relative to reproduction increases. Thus, we expect strong selection against temporal variation in survival under such conditions. By contrast, at low population size, density dependence is virtually absent, and individuals can maximize both survival and reproduction. In this case, vital rates are expected to be less variable over time [52,53]. Therefore, conditions for demographic buffering to occur are most likely to be met when the population size fluctuates near K. It is important to account for density dependence when testing the demographic buffering hypothesis because the structure of the population, and thereby the relative demographic impact of vital rates, changes with population size in complex ways [54]. For example, in the study by Reed and Slade, demographic buffering was detected in three out of four rodent species [21]. The lack of buffering in the remaining species could be due to its decreasing population size during the study period, which increased the elasticity for reproduction.

Comparing Temporal Variance of Vital Rates on Different Scales

In the first study assessing the demographic buffering hypothesis across a diversified set of species (i.e., 30 populations belonging to 20 different plant and animal species), Pfister [15] found a negative correlation between sensitivity and temporal variance of vital rates, such as survival, fecundity, or growth. However, comparing variance and sensitivity among variables with different scales such

as survival and fecundity may provide spurious results generated by inherent differences in the mean–variance relationship of such variables [16] (Box 2). Several methods have been used to account for the specific mean–variance relationship that characterizes vital rates expressed on a constrained scale (Box 2 and Table 1). However, none offers a solution for comparing temporal variation in vital rates based on different underlying statistical distributions. Some studies that have pooled variances and sensitivities of vital rates on different scales have failed to detect the expected negative relationship [26,29], while others did report such a relationship (e.g., [15,55]) (Table 1).

Identifying which statistical distribution fits the observed mean–variance relationship of the vital rates is important in order to scale and estimate the temporal variance of vital rates consistently [56]. We further advise students of demographic buffering to derive testable predictions that restrict the comparison of temporal variation in vital rates to rates with similar mean–variance relationships. For example, variation in survival can be compared among age classes, but not with fecundity, unless the species gives birth to a single offspring (i.e., monotocous species).

Sources of Variation in Vital Rates and Targets of Natural Selection

Stochastic variation in vital rates results from **environmental stochasticity**, demographic stochasticity, and sampling variance [7] (Box 3), of which the latter cannot be targeted by natural selection. In large populations, temporal variation in vital rates is mostly driven by environmental stochasticity and we expect this source of variation to be buffered in the most influential vital rates [7,20]. In populations of less than 100 individuals, demographic stochasticity is usually a dominant source of variation for the vital rates. Depending on the taxon and mating system, it can also be the case in populations far above 100 individuals [7,57,58]. For example, Sæther *et al.* [58] showed that demographic stochasticity was the main component of observed population fluctuations in four bird species at population sizes that exceeded 250 individuals. Whether demographic stochasticity could be targeted by natural selection and, therefore, buffered, remains debated (but see [59,60] and Outstanding Questions). However, demographic stochasticity still generates within-year variation among individuals in survival and reproduction, possibly increasing temporal variance in vital rates and thereby reducing the population growth rate at small population sizes [7]. Thus, ignoring demographic stochasticity may seriously overestimate environmental stochasticity and the amount of temporal variation in vital rates [61] and may prevent the detection of demographic buffering. Even so, demographic stochasticity is rarely accounted for (but see [62] and Table 1), and it is either assumed to be small enough compared with environmental stochasticity to be ignored, or simply not mentioned (Table 1). Importantly, many studies have reported evidence of demographic buffering without accounting for the influence of demographic stochasticity (Table 1). These results are biased because ignoring demographic stochasticity increases type 2 errors (false negative), but still support the occurrence of demographic buffering because type 1 errors are not affected.

Sampling variance is not influenced by natural selection and a proper assessment of the demographic buffering hypothesis needs to estimate and account for this potentially confounding source of variation. Most studies on demographic buffering have accounted for sampling variance (Table 1), using, for example, random effects models to estimate the variance [63]. Thanks to recent methodological advances, it is now possible to decompose the different sources of variance in studies based on capture–mark–recapture data [e.g., by using integrated population models (IPMs) [64]]. The study by Bjørkvoll *et al.* [62] remains the only one that investigated demographic buffering by including all three variance components (Table 1).

Box 2. Accounting for the Mean–Variance Relationship of Vital Rates

Pfister [15] conducted the first analysis of demographic buffering using the CV of the vital rates and their elasticities. However, using the CV to standardize variation in vital rates is not satisfactory either, because the CV also depends on the relationship between the variance and the mean [16,20]. For binomial variables, the maximum value of the CV varies

with P: $CV_{max} = \sqrt{\frac{1-P}{P}}$. As P approaches 0, CV_{max} approaches infinity, and when $P = 1$, $CV_{max} = 0$ [16]. Consequently,

the negative correlation observed between CV and elasticities (or between variance and sensitivities) across vital rates with different scales may not result from an adaptive reduction of the variation in the most influential vital rates, but rather reflects a statistical artefact arising from the mean–variance relationship of bounded variables [16]. The problem of comparing vital rates with different distributions may also affect the comparison of the CV and elasticity of apparently similar vital rates, such as fecundity in different organisms. Indeed, for organisms that only produce one offspring per reproductive event (e.g., seabirds and large mammals), fecundity is based on individual contributions of reproduction approximated by a binomial distribution, while for organisms producing many offspring (e.g., fish and plants), fecundity is well approximated by a generalized Poisson distribution [89]. Several methods have been proposed to reduce the impact of the mean–variance relationship of vital rates (see Table 1 in the main text): (i) scale the variance or CV in survival with the maximum amount of 'possible variation' [16,20]; (ii) use an arcsine square-root transformation [$\sin^{-1}(\sqrt{\mu})$] on survival data [90,91]. However, when the mean of a binary variable is close to 0 or 1, arcsine transformation of binomial variables does not work well and a logit transformation both has higher power and is simpler to interpret [92]; and (iii) randomly draw vital rate values from simulated statistical distributions (i.e., the beta-distribution for survival, and gamma- and negative binomial distribution for low and high reproductive rates, respectively) around the observed mean. Then, test the relationship between the CV and elasticity of the vital rates of the simulated distributions and compare to the relationship observed in the populations [27]. Using this method, a stronger negative correlation between CV and elasticity for the observed data compared to the simulated data would indicate demographic buffering.

Comparing the slopes of the relationship between CV and elasticity (on a log-scale) between the observed and simulated data sets, Bjørkvoll *et al.* [62] found no difference and concluded that demographic buffering was absent in their study population. Interestingly, using the relative CV, they found a negative correlation with the elasticity of the vital rates, providing support for the demographic buffering hypothesis. This last result indicates that tests of the demographic buffering hypothesis are sensitive to the method used to account for the mean–variance relationship of vital rates on different scales. Accordingly, Bjørkvoll *et al.* [62] noticed that, even though both methods satisfactorily removed the undesirable relationship between the mean and the variance for binary vital rates, the latter qualitative test did not include estimates of uncertainty in the parameters and, therefore, was less conservative than the former quantitative method.

Responses of Vital Rates to Different Sources of Variation

Covariation, Environmental Autocorrelation, and Length of Time-Series

Environmental stochasticity can affect the population growth rate via temporal variation in vital rates, covariation among vital rates, and temporal autocorrelations of a single vital rate or combinations of vital rates (e.g., [65–67]) (Box 4). Even though the covariation among vital rates provides small contributions to the population growth rate, an increase in environmental variation due to, for example, climate change, may change the structure of covariances among vital rates, and

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Box 3. Sources of Variation in Vital Rates

Temporal variation in vital rates results from: (i) environmental stochasticity; (ii) demographic stochasticity; and (iii) sampling variance, which results from variation in the estimates of population size and vital rates [7]. Ignoring sampling variance, Engen *et al.* [93] showed that the stochastic growth rate of an age-structured population can be approximated using Equation I as:

$$a(N) = r - \frac{1}{2}\sigma_e^2 - \frac{1}{2N}\sigma_d^2, \qquad\qquad [I]$$

where $r = \ln \lambda_1$, N is the population size, σ_e^2 is the environmental variance and σ_d^2 is the demographic variance. Environmental stochasticity negatively affects population growth rate [10,94,95]. In large populations, it represents the major source of variation in population growth rates [7]. Demographic stochasticity also negatively affects the population growth rate, but this effect increases with decreasing population size [23,96] and represents a form of **Allee effect** [7,11]. Sampling variance is the variance associated with the sampling procedure and increases when sample size decreases [97]. Sampling variance affects the precision of the estimates of most population parameters [98,99] and, even when sampling procedures are reasonably accurate (i.e., unbiased and relatively precise with large sample size), the observed or estimated values are associated with errors that need to be accounted for [100–102].

Box 4. Covariation and Temporal Autocorrelation

The negative effect of temporal variance in vital rates on population growth rate can either be enhanced if there is a positive correlation between vital rates or decreased if the correlation is negative. For example, including covariation between vital rates in the calculation of the sensitivity of population growth rate to vital rate variation in the desert tortoise (*Gopherus agassizi*) changed the sign of the sensitivity from negative to positive for some of the vital rates [70]. Jongejans *et al.* [27] found positive covariances between survival and reproduction in most of the plant populations they studied, with only one out of 40 species displaying a negative contribution of covariances. Even when mostly negative, covariation alone cannot counteract the negative effect of temporal variation in vital rates on the population growth rate (Box 1). However, although unlikely, a high negative covariation between vital rates can reduce the contribution of direct variation in vital rates and, thus, makes it harder to detect statistically the effect of buffering.

Autocorrelations occur when the effects of the environment on the vital rates in a specific time-step carry over to one or more subsequent time-steps, or when the environmental conditions are correlated among time-steps [103], giving a temporal correlation in vital rate variation [14,67]. For example, environmental conditions can track large-scale climatic factors, such as El Niño, which occur periodically, causing serial autocorrelation in environmental variation [104]. Local conditions, such as resources and prey abundance, can also exhibit strong temporal autocorrelation (e.g., population cycles or solar cycles), and can even be a major driving force of the population dynamics [105,106]. Environmental autocorrelation can influence the stochastic growth rate of a population positively or negatively [14]. In strongly age-structured populations, some combinations of age-specific vital rates and their covariation can cause differences between the long-term average age structure and the stable age structure of the deterministic projection matrix, leading to an increase in the stochastic long-run growth rate [107]. Therefore, we should account for environmental autocorrelation to assess reliably demographic buffering, especially for fast-living species that are more prone to be affected by environmental autocorrelation compared with slow-living species [65,108].

ultimately amplify or decrease the effects of environmental variation [68]. Thus, future studies need to account for covariation between vital rates when analyzing the effect of temporal variation in vital rates on the stochastic growth rate, including studies of demographic buffering.

The effect of environmental stochasticity on population growth rate can also be attenuated or amplified by temporal autocorrelations in vital rates (Box 4). Estimating environmental autocorrelation requires several years of data, but most studies to date are based on data sets with relatively few years of data, especially on plants (3–7 years, [27,28,63]; reviewed in [69]). Although some studies have tried to account for short time-series by testing for a negative correlation between variance in vital rates and the number of years in the time-series (e.g., [24]), this is not a satisfactory solution because the variance over a short period of time is just as likely to overestimate as underestimate the true variance of vital rates. Overall, it is unlikely that temporal variation in vital rates has been reliably assessed, because short time-series lead to more uncertain estimates [70] and the presence of positive environmental autocorrelation might have reduced (or increased in case of negative autocorrelation) the variance in the short term [23,71]. To reliably estimate the temporal variance of a vital rate, the study period should extend over at least 10 years for annually reproducing species [71,72]. The precision of the yearly vital rate estimates also depends on the number of individuals sampled each year as well as on demographic stochasticity. Simulations can be a useful tool to assess the precision of the estimates given the data available.

Age-Specific Variation in Vital Rates

The occurrence of demographic buffering is often tested by comparing temporal variation between juvenile and adult survival (Table 1) under the assumption that the higher elasticity of adult survival compared with juvenile survival in slow-living species has generated stronger selection to decrease its temporal variation [20]. However, the power of such tests may be weakened if some adults experience lower and more variable survival than others, due to, for instance, actuarial senescence (i.e., an increase in mortality with age that is widespread across vertebrate populations [73]). Even without actuarial senescence, the elasticity of survival consistently decreases with increasing age [74] and prime-aged adults should be more buffered than old adults against variation in survival. Combining all sexually mature individuals in a single 'adult' stage class might underestimate survival,

thereby overestimating its variance [75,76] and reducing the power of detecting demographic buffering in such a vital rate. It will also inflate the elasticity and decrease the sampling variance of the adult age class because it contains a relatively high proportion of the population. One solution may be to use age- or stage-classified models allowing the comparison between 'prime-aged' and older (possibly senescent) individuals. Few studies so far have accounted for the possible effect of senescence on the test of the demographic buffering hypothesis. Two noticeable exceptions are the studies by Bjørkvoll *et al.* [62] and Gaillard and Yoccoz [20], where the former included older individuals in a separate age class, whereas the latter defined adults as prime-aged individuals only.

Challenges and Recommendations

Although this review reveals a general support for demographic buffering, we also demonstrate that the validity of the conclusions made by the various studies is often challenged by the introduction of simplifying assumptions to the analyses that severely restricts the possibility for a critical evaluation of the demographic buffering hypothesis. These problems are mainly related to difficulties in obtaining unbiased estimates of temporal variation in vital rates that take into account the influence of sampling errors, demographic stochasticity, and fluctuations in age structure, and include analyses of the potential effects of density dependence. For example, several papers found evidence for both buffering and lability across a high number of plant species [27,28,77], but they all suffer from one or more of the aforementioned problems, making it hard to draw any conclusion on whether demographic buffering is happening in plants. In addition, scaling of vital rates poses a problem when comparing rates with different distributions.

Reviewing the various challenges encountered by empirical studies of the demographic buffering hypothesis enables us to suggest a general framework to test for the demographic buffering hypothesis from data collected in the field (Figure 1), and to provide some guidelines for future studies: (i) temporal variance in vital rates must be separated into variance components caused by environmental and demographic stochasticity while accounting for sampling variance. Only the effects of temporal variation in response to variation in environmental conditions should be included when evaluating demographic buffering, assuming that effects of density dependence are accounted for. Based on capture–mark–recapture data, stochastic population models with various degrees of complexity, depending on species-specific life histories, should be parameterized to decompose the variance in the population growth rate. IPMs provide a reliable approach to analyze this type of data and offer the possibility to use additional sources of data to improve the accuracy of the parameter estimates [78,79] (Figure 1); (ii) individual data collected for >10 years for annually breeding species is a recommended minimum time period to reduce the uncertainties in the estimates of temporal variance, temporal covariance, and autocorrelation in vital rates; (iii) for demographic traits measured as probabilities, the analysis should take the mean–variance relationship into account. The variance should either be corrected by its maximum value or, alternatively, the demographic traits should be modeled with a distribution that fits the observed variation; (iv) vital rates with different mean–variance relationships should either be analyzed separately or by using an appropriate statistical modeling framework, such as IPM. Applying an IPM approach [78] presents the advantage of easily implementing covariances between vital rates in the model (e.g., [80]); and (v) demographic buffering considers only the effects of environmental stochasticity using the stochastic long-run growth rate as a measure of fitness. An important challenge is now to examine the presence of demographic buffering when other quantities are maximized by evolution, such as in populations subject to density dependence [81].

Concluding Remarks

The ability of species to cope with increasing environmental variation is of great interest in management and conservation, and more generally in ecology and evolution. Our review shows that environmentally induced temporal variation in vital rates often follows the pattern expected with demographic buffering. However, more work is needed to fully understand the role of demographic buffering, along with demographic lability, in shaping population dynamics facing environmental variation. More work is also needed to unravel the mechanisms inducing those demographic patterns, such as homeostasis (i.e., the ability of the organism to maintain a constant expression of a phenotypic character in different environments) and phenotypic plasticity (i.e., the capacity of an individual to produce different phenotypic characters in different environments) [82–84]. Similarly, it remains unknown how selection on behavioral, physiological, and life-history traits can translate into demographic buffering, how fast such changes can occur, and the potential cost of maintaining them (see Outstanding Questions) [82,85]. As one of few hypotheses grounded in life-history theory that targets temporal variation in traits rather than mean values, demographic buffering is pivotal in the current context of global changes, where both the mean (i.e., trend) and the variance of climatic variables are expected to change.

Author Contributions

C.H.H., C.P., M.G., B.E.S., J.M.G., and N.G.Y. conceptualized the study. C.H.H. did the literature survey and wrote the first draft of the manuscript. All authors contributed to the final manuscript.

Supplemental Information

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Outstanding Questions

The demographic buffering hypothesis was derived using the long-run population growth rate as a measure of fitness. How does the concept fit short-term transient dynamics?

While demographic buffering is expected to minimize variation due to environmental stochasticity, can it also target variation generated by demographic stochasticity?

Can demographic buffering impact population persistence when facing increased environmental variation?

Given that the relationship between environmental variables and vital rates may be nonlinear, can the role of demographic buffering vary along the range of environmental variation?

Negative correlations between the temporal variance (CV) of vital rates and their sensitivity (elasticity) are often observed, but how many of these correlations results from selection on variation compared with selection on the mean?

How evolvable are genetic and environmental variability in vital rates?

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