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Pfab, Ferdinand; Gabriel, Wilfried; Utz, Margarete

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Reversible phenotypic plasticity with continuous adaptation

Ferdinand Pfab[1,2] · **Wilfried Gabriel**[1] · **Margarete Utz**[1,3]

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Abstract We introduce a novel model for continuous reversible phenotypic plasticity. The model includes a one-dimensional environmental gradient, and we describe performance of an organism as a function of the environmental state by a Gaussian tolerance curve. Organisms are assumed to adapt their tolerance curve after a change of the environmental state. We present a general framework for calculating the genotype fitness if such adaptations happen in a continuous manner and apply the model to a periodically changing environment. Significant differences of our model with previous models for plasticity are the continuity of adaptation, the presence of intermediate phenotypes, that the duration of transformations depends on their extent, fewer restrictions on the distribution of the environment, and a higher robustness with respect to assumptions about environmental fluctuations. Further, we show that continuous reversible plasticity is beneficial mainly when environmental changes occur slow enough so that fully developed phenotypes can be exhibited. Finally we discuss

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✉ Ferdinand Pfab
ferdinand.pfab@gmail.com

Wilfried Gabriel
wilfried.gabriel@lmu.de

Margarete Utz
m.b.utz@rug.nl

[1] Department of Biology II, Ludwig Maximilian University of Munich, Großhaderner Straße 2, 82152 Martinsried-Planegg, Germany

[2] Department of Mathematics, University of Trento, Via Sommarive 14, 38123 Povo, TN, Italy

[3] Center for Ecological and Evolutionary Studies, University of Groningen, Nijenborgh 7, 9747 AG Groningen, The Netherlands

how the model framework can be generalized to a wide variety of biological scenarios from areas that include population dynamics, evolution of environmental tolerance and physiology.

Keywords Continuous adaptation · Environmental stress · Environmental tolerance · Reversible phenotypic plasticity · Phenotypic flexibility

Mathematics Subject Classification 92B05

1 Introduction

Changing environmental conditions are ubiquitous, both on small and large temporal and spatial scales. Daily, tidal, seasonal or annual rhythms affect the way of life of individuals, and most mobile organisms encounter diverse habitat during the course of their lives. Individuals are impelled to adapt to these changes lest they face fitness loss or death. (Adaptive) phenotypic plasticity is the ability of an organism to adjust to changing environmental conditions on the phenotypic level. If such adaptations occur repeatedly and reversibly we speak of 'reversible phenotypic plasticity' or 'phenotypic flexibility' and the underlying trait is often referred to as being 'labile'. Most organisms are in fact phenotypically plastic in one or several traits, and reversible plasticity is common in all kingdoms of life.

The manner of adaptation of a reversibly plastic organism varies among organisms and traits. Behavioural modifications occur often instantaneously, such as the attempt of prey to escape as soon as they recognize approaching predators. However, morphological transformations develop or degenerate rather continuously over a period of time. To name but a few examples, our human skin tans when exposed to sun light and pales when covered; muscle parts grow or reduce with regular activity or passivity; the digestive system of animals is generally quite flexible [e.g. in snakes (Starck and Beese 2001), migratory birds (Piersma and Lindström 1997) or lactating mammals (Diamond and Hammond 1992)]; marine iguanas can switch repeatedly between growth and shrinkage, depending on environmental conditions (Wikelski and Thom 2000); *Kosciuscola tristis* grasshoppers can repeatedly alter their body colour due to the surrounding temperature (Key and Day 1954); barnacles show reversible phenotypic plasticity in the length of their cirri depending on how exposed their habitat is to waves (Marchinko 2003; Neufeld 2012), and deciduous trees hibernate without leaves to minimize water loss during winter.

Phenotypic plasticity has been studied for over a century and continues to be in the focus of many investigations—not least because of its implications for current burning ecological issues. Invasive species are believed to be often highly plastic (Davidson et al. 2011; Engel et al. 2011) and plastic species are thought to be favoured by global change (Matesanz et al. 2010; Nicotra et al. 2010). Mathematical models are essential for the understanding of mechanisms underlying phenotypic plasticity and for estimating its impact on ecological and evolutionary processes. Particularly the comparison of irreversible and reversible plasticity was formalized by Gabriel and co-workers (Gabriel 1999, 2005, 2006; Gabriel et al. 2005; Utz et al. 2014). These

authors developed models where temporal maladaptation (reduced fitness) during lag times is explicitly implemented by a time delay that precedes instantaneous adaptation to a changed environment. Here, we present a framework for continuous adaptations. We employ the theory of environmental tolerance curves and assume that the fitness of an organism expressing a given phenotype follows a Gaussian function along an environmental gradient (Lynch and Gabriel 1987). Phenotypic plasticity is regarded as changing the parameters of the tolerance curve in response to a changed environment.

We introduce the general idea of continuously changing tolerance curves in Sect. 2 of this paper. In Sect. 3, we confine this framework to a specific scenario. One of our specific assumptions is the normalization of the tolerance curve in order to model a trade-off between being well adapted at the mode of the curve and being able to cope with a wide range of environmental conditions. As a consequence continuous reversible plasticity can be seen as the ability to shift the peak of the tolerance curve (cf. Fig. 1). We assume that such shifts occur along a straight line with constant speed. Concerning the environment we assume periodic changes (as illustrated in Fig. 2). (Although in the online appendix P we briefly discuss a stochastically changing environment). We distinguish between stressful and stress-free periods and assume that stress intensities vary between stress events. Although we mainly have changing external conditions in mind (e.g., predator threat, variation in temperature, droughts or floods), 'stress' may also refer to changing internal conditions such as parasitic infections. The calculation for the specific scenario is worked out in Appendix A. The results of this calculation are discussed in Sect. 4 of the main text, together with a comparison of the current model with previous models for reversible plasticity with instantaneous but delayed adaptation, irreversible plasticity and non-plasticity.

2 The model framework

We characterise a phenotype by the mode m, breadth b and enclosed area c of a Gaussian tolerance curve that defines performance as a function of the environmental state ϕ,

$$f(m, b, c, \phi) = \frac{c}{\sqrt{2\pi}b} \mathrm{e}^{-\frac{1}{2}\frac{(m-\phi)^2}{b^2}}, \tag{1}$$

and we define phenotypic plasticity as the ability of an organism to adapt its phenotype by smoothly transforming this tolerance curve (see Fig. 1).

We assume that the tolerance curve directly gives the (life-time) fitness of an individual that experiences no environmental or phenotypic changes during life. For individuals that encounter different environmental conditions or have a changing phenotype, we assume that the performance during different phases during life affect the fitness multiplicatively (like a scenario with a single reproductive event at the end of life and several predation events that determine fitness; for a comparison of multiplicative and additive fitness effects, see Utz et al. 2014). We therefore calculate the fitness of an individual by dividing lifetime into phases with constant phenotype and environmental state

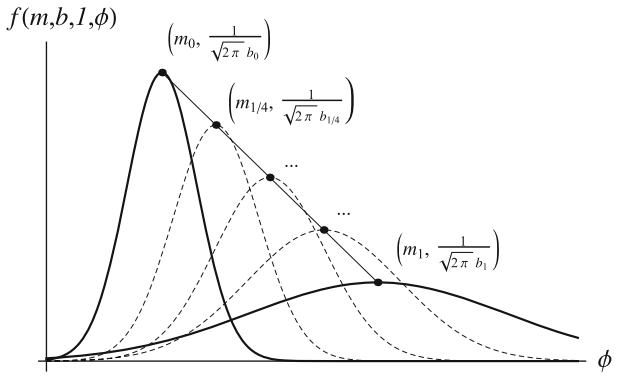

Fig. 1 Transformation between a non-induced tolerance curve $f(m_0, b_0, 1, \phi)$ and an induced tolerance curve $f(m_1, b_1, 1, \phi)$. Here the area under the tolerance curve is normalized and its peak shifts along a straight line

$$(m_\mathrm{I}, b_\mathrm{I}, c_\mathrm{I}, \phi_\mathrm{I}), (m_\mathrm{II}, b_\mathrm{II}, c_\mathrm{II}, \phi_\mathrm{II}), \ldots \tag{2}$$

and then multiplying the performances during these phases [see formula (1)], each raised to the power of its duration $t_\mathrm{I}, t_\mathrm{II}, \ldots$ (these are measured relative to lifetime)

$$w_i(m_\mathrm{I}, b_\mathrm{I}, c_\mathrm{I}, \phi_\mathrm{I}; m_\mathrm{II}, b_\mathrm{II}, c_\mathrm{II}, \phi_\mathrm{II}; \ldots) = \prod_{k=\mathrm{I},\mathrm{II},\ldots} f(m_k, b_k, c_k, \phi_k)^{t_k}. \tag{3}$$

We assume that during a transformation from phenotype (m_a, b_a, c_a) to phenotype (m_z, b_z, c_z), the parameters m, $\frac{1}{b}$ and c adapt simultaneously and linear with time. If such a transformation lasts for a time δ and if the environmental state changes linearly from ϕ_a to ϕ_z, the fitness contribution of this phase is

$$\begin{aligned}\lim_{n\to\infty} \prod_{r=1}^{n} f\left(\frac{n-r}{n} m_a + \frac{r}{n} m_z, \frac{1}{\frac{n-r}{n}\frac{1}{b_a} + \frac{r}{n}\frac{1}{b_z}}, \frac{n-r}{n} c_a + \frac{r}{n} c_z, \frac{n-r}{n}\phi_a + \frac{r}{n}\phi_z\right)^{\frac{\delta}{n}} \\ = g(m_a, b_a, c_a, \phi_a, m_z, b_z, c_z, \phi_z)^{\delta}. \end{aligned} \tag{4}$$

If either m, $\frac{1}{b}$ or c is constant, the peak of the tolerance curve shifts along a straight line. An expression for formula g is derived in Appendix A.4.

The genotype fitness is, according to Haldane and Jayakar (1963), given by the geometric mean of the fitness of an infinite number of generations. If we assume equal fitness for all offspring of a genotype within one generation, this is equivalent to calculating the geometric mean of the fitness of $n \to \infty$ independent individuals,

$$w = \lim_{n\to\infty} \prod_{i=1}^{n} w_i{}^{\frac{1}{n}}. \tag{5}$$

3 Application of the general model to a specific scenario

To imply a trade-off between being well adapted at the mode of the tolerance curve and having a wider breadth of adaptation, we now normalize the area c under the tolerance curve to 1 and we no longer include the parameter c in the formulas. We consider a genotype with two fully developed phenotypes: the stress-induced phenotype (m_1, b_1) and the non-induced phenotype (m_0, b_0). Due to the linearity of the adaptation, intermediate phenotypes can be described by the phenotypic state $s \in]0, 1[$

$$\begin{aligned} m_s &= (1-s)m_0 + sm_1, \\ b_s &= \frac{1}{\frac{1-s}{b_0} + \frac{s}{b_1}}. \end{aligned} \tag{6}$$

Because of the normalization of the area under the tolerance curve, a phenotype is equivalently defined by the peak of its tolerance curve with coordinates $(m, \frac{1}{\sqrt{2\pi}b})$, and the peaks of intermediate phenotypes lie on a straight line between the peak of the induced and the peak of the non-induced tolerance curve (cf. Fig. 1). Concerning the speed of adaptation, we assume that during stress-full times the peak of the tolerance curve shifts with constant speed v_1 towards the peak of the induced tolerance curve $f(m_1, b_1, \phi)$, and in the absence of stress it shifts with constant speed v_0 towards the peak of the non-induced tolerance curve $f(m_0, b_0, \phi)$. We assume that the peak does not shift anymore when the target phenotype is reached.

Further, we adopt a temporal distribution of environmental states that was used in previous models for phenotypic plasticity by Gabriel et al. (2005) and Utz et al. (2014). A stress-free environmental state (to which we assign the environmental state 0) is interrupted by stress events with frequency p (see Fig. 2) and the environmental state does not change continuously but suddenly. We assume that the stress events persist for a constant time $t \leq \frac{1}{p}$ and do hence not overlap. The environmental state (stress intensity) during a stress event is assumed to be constant and the stress intensities of different stress events are independently and identically distributed with mean $\overline{\phi}$ and standard deviation σ_ϕ. We assume that a change of the phenotype depends only on whether stress is present or absent, and the organisms cannot detect the actual stress intensity (the genotype, of course, can be adapted to the model parameters such as $\overline{\phi}$ and σ_ϕ). This assumption is referred to as 'incomplete information' by Gabriel et al. (2005). In order to keep the model simple and to facilitate its comparison to previous models of Gabriel and others, we furthermore assume that organisms are

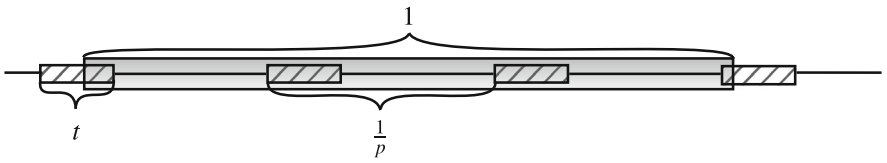

Fig. 2 Time line. Stress events (*striped boxes*) occur with frequency p and persist for a time t. The *shaded area* indicates the lifetime of an organism

born with the induced or non-induced phenotype, depending on whether stress is present or absent at the time of birth. In the Discussion we consider the phenomenon of early anticipation of environmental changes due to different environmental cues and in periodically changing environments. The lifetime of an organism is assumed to be 1, and p and t are measured relative to it. We let the time of birth of an organism be uniformly distributed in the sequence of stress events and stress-free times.

In Appendix A we show how the genotype fitness can be calculated in this specific scenario. In the following section we discuss the results of this fitness calculation.

4 Results and comparison with other models of plasticity

In this section we compare the introduced model for *reversible plasticity with continuous adaptation* (continuous reversible plasticity) with other models for phenotypic plasticity that inspired the modelling approach presented here (see references below). The comparison is based on the scenario described in Sect. 3 which implies a normalized tolerance curve, a constant adaptation speed and a periodically changing environment. Based on the assumption that evolution adapts the genotype parameters m_0, m_1, b_0 and b_1 for any given set of model parameters (p, t, $\overline{\phi}$, σ_ϕ, v_0, v_1) such that the fitness w is maximized, we numerically maximized the fitness derived in the appendix [formula (45)].

4.1 Other kinds of plasticity

Non-plasticity (Gabriel et al. 2005): A non-plastic organism can exhibit only a single phenotype with constant parameters (m, b) of the tolerance curve.
Irreversible plasticity (Gabriel 2006; Utz et al. 2014): An irreversibly plastic organism fixes its phenotype at birth. If organisms have only information about the presence or absence of stress but not about the actual intensity (which is assumed to be randomly distributed), only two phenotypes are exhibited: the induced phenotype with parameters (m_1, b_1) if life starts during stress and the non-induced phenotype with parameters (m_0, b_0) otherwise.
Reversible plasticity with delayed instantaneous adaptation (instantaneous reversible plasticity) (Gabriel et al. 2005; Gabriel 2005, 2006; Utz et al. 2014): An instantaneously reversibly plastic organism can exhibit two phenotypes: the non-induced phenotype with parameters (m_0, b_0) and the induced phenotype with parameters (m_1, b_1). The phenotype at birth is chosen according to the prevalent environment. Again, organisms have only information about the presence or absence of stress. Throughout life, stress induces the expression of the induced phenotype instantaneously but after a delay d_1 during which the phenotype stays unchanged. Likewise, the cessation of stress causes the opposite transformation instantaneously but after a delay d_0. Since these delay times are assumed to be constant, the time needed for a transformation is independent of how much the two phenotypes differ. This is different in the present model for continuous reversible plasticity because there the time needed for a phenotypic transformation depends on the distance of the peaks of the tolerance

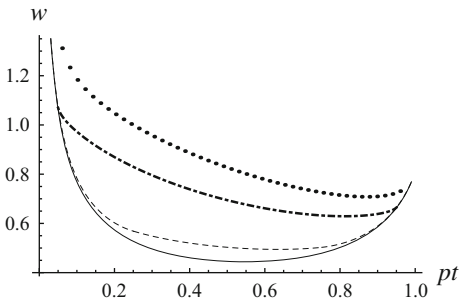

Fig. 3 Effect of the stress proportion pt on the maximal fitness w for non-plasticity (*solid*) irreversible plasticity (*dashed*), instantaneous reversible plasticity (*dash-dotted*) and continuous reversible plasticity (*dotted*). Parameter values are: $p = 0.25$ ($t = \frac{pt}{p}$), $\overline{\phi} = 1$, $\sigma_\phi = 0.3$, $v_0 = v_1 = 3$, $d_0 = d_1 = 0.2$

curves. In addition, as opposed to the model for continuous reversible plasticity, the model for instantaneous reversible plasticity can only be applied when phenotypic transformations are always completed before the environment changes again because the model does not include in-between phenotypes. That means that the time needed to transform to the induced phenotype must be shorter than or equal to the length t of a stress event, and the time needed to transform to the non-induced phenotype must be shorter than or equal to the length $\frac{1}{p} - t$ of a gap between two stress events, where p is the frequency of stress events,

$$d_1 \leq t \quad \text{and} \quad d_0 \leq \frac{1}{p} - t. \tag{7}$$

The optimal genotype parameters for these three types of plasticity have been obtained analytically in the cited articles. Plots that show how the maximal fitness and optimal genotype parameters depend on the model parameters for the different kinds of plasticity are included in the online appendix S of this article. Several excerpts are shown in this section and, based on them, we investigate differences and similarities between the models.

4.2 Comparison of plasticity and non-plasticity

It is obvious that plasticity is generally only beneficial in a changing environment. In the case that stress never occurs (the stress proportion $pt \to 0$) or is omnipresent ($pt \to 1$) the maximal fitness of an irreversibly plastic genotype and of a continuously reversibly plastic genotype equals the maximal fitness of a non-plastic genotype (see Fig. 3 at $pt = 0$ and $pt = 1$ and also Figures S.3, S.7, S.10, S.13 and S.14 in the online appendix S). [Note that the model for instantaneous reversible plasticity is not applied in this case due to the constraints (7)]. For $pt \to 0$ plastic organisms express only the non-induced tolerance curve which adopts the same values as the tolerance curve of non-plastic organisms: $m_0 = 0$ and $b_0 = 0$. That implies that the maximal fitness approaches infinity. (Note that for biological reasons the fitness could be prevented to approach infinity in such cases by including additional constraints on the height of the tolerance curve.) For $pt \to 1$ plastic organisms always express the induced tolerance curve and this tolerance curve adopts the same values as the tolerance curve

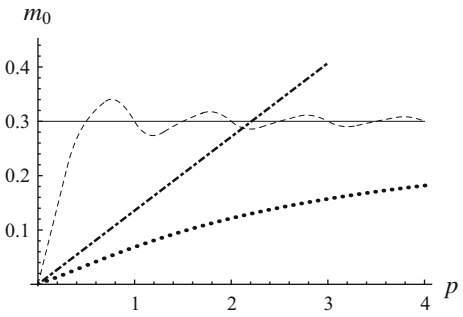

Fig. 4 Effect of the stress frequency p on the optimal mode m of the tolerance curve for non-plasticity (*solid*) and the optimal mode m_0 of the non-induced tolerance curve for irreversible plasticity (*dashed*), instantaneous reversible plasticity (*dash-dotted*) and continuous reversible plasticity (*dotted*). Parameter values are: $pt = 0.3$ ($t = \frac{pt}{p}$), $\overline{\phi} = 1, \sigma_\phi = 0.3, v_0 = v_1 = 8, d_0 = d_1 = 0.1$

of non-plastic organisms: $m_1 = \overline{\phi}$ and $b_1 = \sigma_\phi$ (see the formulas in Gabriel et al. 2005). All organisms then have the same maximal fitness (see Fig. 3) but the fitness only approaches infinity when the stress intensities are not stochastic ($\sigma_\phi = 0$).
When the environment changes very fast ($p \to \infty$), irreversible plasticity as well as continuous reversible plasticity loose its benefits, and the model for instantaneous reversible plasticity exceeds its limit of applicability due to the constraints (7). For irreversible plasticity and for continuous reversible plasticity, the optimal values for the parameters of the induced and the non-induced tolerance curve approach the optimal values of the tolerance curve for non-plasticity, $m_0 = m_1 = m$ and $b_0 = b_1 = b$ (see Fig. 4 and also Figures S.1, S.6, S.7, S.8 and S.9 in the online appendix S). For irreversible plasticity the optimal parameter values and the maximal fitness approach the respective values for non-plasticity in an oscillating fashion with increasing stress frequency p. The reason is that these optimal parameter values depend on the expected time of stress during a lifetime, given that stress is prevalent at birth, and this expectation value approaches the stress proportion pt in an oscillating fashion with increasing p (see the online appendix P for an investigation of this behaviour and an alternative environmental distribution without resulting fluctuations). In Fig. 4 these oscillations do not affect the model for continuous reversible plasticity but they obviously will have an effect if the adaptation speed is reduced because continuous reversible plasticity with adaptation speed $v_0 = v_1 = 0$ resembles irreversible plasticity. When the environment changes very rarely ($p \to 0$), organisms virtually experience no environmental change during their lives and reversible plasticity is the same as irreversible plasticity. Organisms born at a stressful time experience stress during their whole life and therefore the optimal parameter values of the induced tolerance curves for the plastic organisms equal the optimal parameter values of the tolerance curve of a non-plastic organism if the stress proportion pt was 1: $m_1 = \overline{\phi}$ and $b_1 = \sigma_\phi$. Analogously, organisms born during a stress-free time experience no stress during their whole life and the optimal parameter values of the non-induced tolerance curve of a plastic organism equal the optimal parameter values of the tolerance curve of a non-plastic organism if pt was 0: $m_0 = 0$ and $b_0 = 0$. Since organisms born during stress-free times are hence perfectly adapted in this case, all kinds of plasticity lead to infinitely high maximal fitness when $p \to 0$.

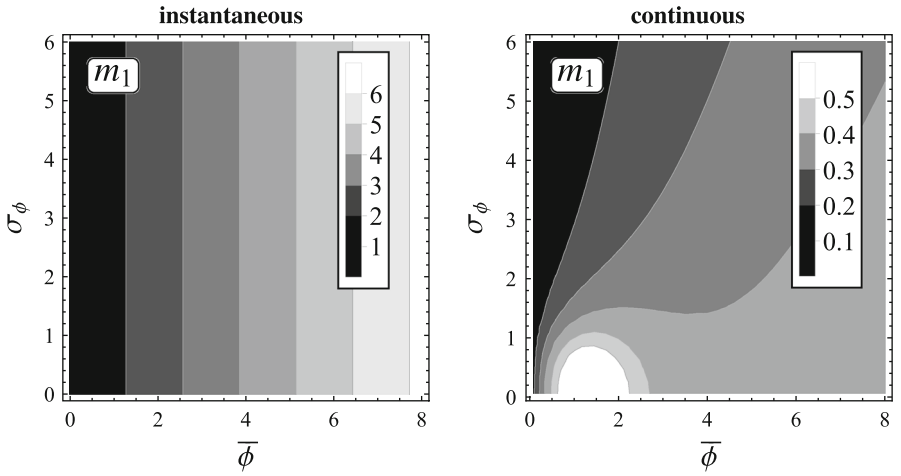

Fig. 5 Effects of the expected stress intensity $\overline{\phi}$ and the standard deviation σ_ϕ of the stress intensities on the optimal mode m_1 of the induced tolerance curve for instantaneous (*left*) and continuous reversible plasticity (*right*). Note that the values of the isoclines differ. Parameter values are: $p = 0.7$, $pt = 0.3$ ($t = \frac{pt}{p}$), $v_0 = v_1 = 6$, $d_0 = d_1 = 0.1$

4.3 Comparison of continuous and instantaneous reversible plasticity

We focus now on differences between the two models for reversible plasticity. The models include different kinds of parameters: For continuous reversible plasticity, the parameters v_0 and v_1 determine the speed with which the peak of the tolerance curve can be shifted, whereas for instantaneous reversible plasticity, the parameters d_0 and d_1 determine the delay of adaptation after environmental change. This difference makes not only quantitative comparison difficult and limits the applicability of the model for instantaneous reversible plasticity [see the constraints (7)], it also affects how fitness depends on the model parameters and therefore the mechanism of how the fitness is optimized.

One important consequence of the different approaches is that for instantaneous reversible plasticity the transformation delays are independent of how much the induced and the non-induced phenotype differ, whereas for continuous reversible plasticity the time needed for a phenotypic transformation depends on the Euclidean distance between the peaks of the two tolerance curves. Since this distance depends on modi and breadths of the tolerance curves, continuous reversible plasticity can lead to an additional trade-off between changing the mode and the breadth of adaptation. An example of how this trade-off affects the outcome of optimization in the two models is depicted in Fig. 5. The figure shows that, for instantaneous reversible plasticity, the optimal mode m_1 of the induced tolerance curve depends linearly on the expected stress intensity $\overline{\phi}$ and is independent of the standard deviation σ_ϕ of the stress intensities. [This is also shown analytically in Gabriel et al. (2005)]. In contrast, for continuous reversible plasticity, m_1 first increases and then decreases with $\overline{\phi}$ when σ_ϕ is low. To understand this behaviour, it is useful to see how the optimal values

of the other genotype parameters depend on $\overline{\phi}$ and σ_ϕ. In Figure S.11 in the online appendix S in the panels for continuous plasticity, when $\overline{\phi}$ or σ_ϕ is high, m_1 is close to the optimal mode m_0 of the non-induced tolerance curve (note that m_0 itself increases slightly with $\overline{\phi}$) and adaptation happens more via the breadth of the tolerance curve [the difference between b_0 and b_1 is larger for continuous reversible plasticity than for instantaneous reversible plasticity (left panels)]. This effect is also observed in Figures S.2, S.6 and S.12 in the online appendix S.

Another mechanism is at work only in the model for instantaneous reversible plasticity in which adaptations to environmental changes occur after constant delays: When these delays are almost as long as the stress events or stress-free times, then a phenotypic transformation occurs shortly before the next change of the environment. Hence, fitness is maximized when the induced tolerance curve is adapted to stress-free times and the non-induced tolerance curve is adapted to stressful times. In Figure S.1 in the online appendix S, for instantaneous reversible plasticity (dash-dotted lines) the optimal mode m_1 of the induced phenotype is smaller than the optimal mode m_0 of the non-plastic tolerance curve when the value of p is close to 3 where the model is close to the limit of applicability [cf. constraints (7)]. This behaviour results from the periodicity of the environment. A similar conclusion has been found by other authors including Lande (2009), who states, in the jargon of quantitative genetics, that "a negative environmental autocorrelation over a developmental time lag can select a norm of reaction with slope of opposite sign to that of the optimum phenotype as a function of the environment". Such 'optimal maladaptations' could not be observed in the present model for continuous reversible plasticity since phenotypic adaptation begins immediately after environmental change.

4.4 Continuous reversible plasticity with non-optimized genotype parameters

A closer look at the model for continuous reversible plasticity reveals an interesting behaviour for the case that the environment changes so quickly that phenotypic transformations cannot be completed. [Note that the model for instantaneous reversible plasticity cannot be applied at high stress frequencies because of the constraints (7).] For the graphs in Fig. 6, we chose constant (non-optimized) but tendentiously realistic genotype parameter values—i.e. the induced mode m_1 is closer to the expected stress intensity $\overline{\phi}$ than the non-induced mode m_0, and the induced breadth b_1 is greater than the non-induced breadth b_0. With constant transformation speeds, this implies that for each direction of adaptation the time needed for a full phenotypic transformation is constant. The lower plot of Fig. 6 shows these times as horizontal lines. The decreasing curves represent the durations of the stress events (solid curve) and of the gaps between the stress events (dashed curve) as functions of the stress frequency p. The upper plot shows the fitness as a function of p. In both plots, the stress proportion pt is kept constant. We can see that the fitness first decreases rapidly with p until the stress events last just as long as an organism needs to completely transform from the non-induced to the induced phenotype. After that the organism can no longer entirely adapt to stress but instead is in a transient state of phenotypic change during stress events (intersection point of the two solid lines in the lower plot). At this breaking point, the

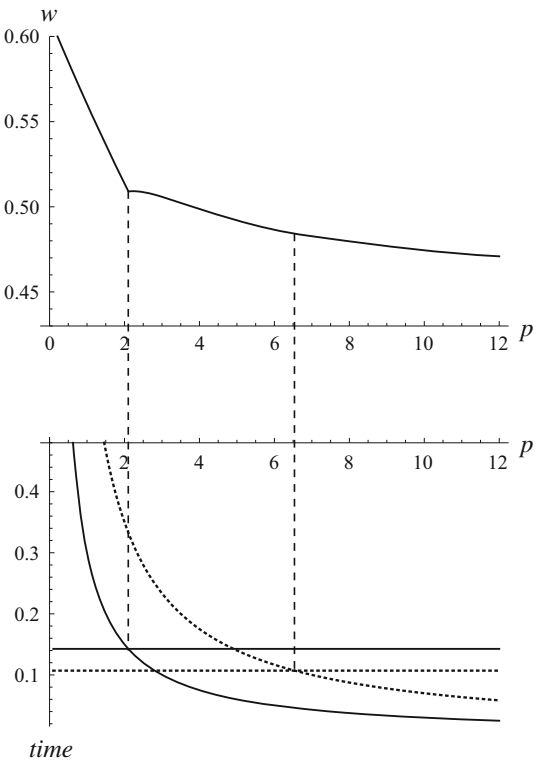

Fig. 6 *Upper plot* Effect of the stress frequency p on the fitness w for continuous reversible plasticity with constant, not optimized, genotype parameters. *Lower plot* Length t of the stress events (*solid curve*) and length $\frac{1}{p} - t$ of the stress-free times (*dotted curve*) and the durations of full transformation from the non-induced to the induced phenotype (*solid horizontal*) and full transformation from the induced to the non-induced phenotype (*dotted horizontal*), all as a function of stress frequency p. Parameter values are: $pt = 0.3$ ($t = \frac{pt}{p}$), $\bar{\phi} = 1$, $\sigma_\phi = 0.3$, $v_0 = 8$, $v_1 = 6$, $m_0 = 0.1$, $m_1 = 0.95$, $b_0 = 0.6$, $b_1 = 0.7$

slope of the fitness curve is discontinuous and afterwards the fitness decreases much slower. Presumably, the reason for the discontinuity of the slope is that plasticity is mainly beneficial in phases where the phenotype is fully adapted. The benefit of an incomplete transformation seems to be compensated by the elongated distance for the transformation back in the other direction. Further plots concerning this behaviour can be found in the online appendix S. Interestingly, plot S.17 suggests that this discontinuity of the slope does not occur in such a strong manner when only the breadth of adaptation is changed. That may have to do with the fact that a changing breadth of adaptation usually does not change the fitness as abruptly as a shift of the mode of adaptation.

5 Discussion

As much as phenotypic plasticity is a universal phenomenon as diverse are the ways in which organisms exhibit it across different biological scenarios. We presented a new model for reversible plasticity where adaptations occur in a continuous way. In this section, we discuss the assumptions and limitations of the model and its relevance for real situations, and we present potential further investigations and possibilities how it can be embedded into other modelling frameworks.

A central aspect of our theory is the assumption that the performance of a phenotype along an environmental gradient can be described by a Gaussian tolerance curve. Although all one-dimensional unimodal tolerance curves that imply lethality on both ends of the gradient can assume a Gaussian shape via appropriate scale transformation, the Gauss property and the normalization become real assumptions because the same scale transformation does not necessarily result in a Gauss curve for different original tolerance curves. In reality, tolerance curves may be shaped differently. For example, it is known that heat tolerance curves can be strongly skewed (see Huey and Kingsolver 1989). Such considerations are therefore important when applying the model framework to a specific scenario. Preliminary tests showed that analytic formulas for the mean performance during phases of transformation can be obtained for some differently shaped tolerance curves.

For the specific scenario we analysed, a trade-off between being well adapted at the mode of the tolerance curve and having a larger breadth of adaptation has been implemented by normalizing the area under the tolerance curve (note that the actual value for the normalization is arbitrary when fitness is thought of as a relative measure). Such trade-offs may be widespread in biology (e.g. for a stress-induced phenotype with increased metabolism) but our realization may seem somewhat arbitrary. Therefore, when applying the model framework to a different scenario it might be important to include a flexible area under the tolerance curve. (Note that also deterministic processes like ageing can change the phenotype and hence this area.) For this reason we already include the possibility of a linear change of the area under the tolerance curve in the formulas presented in this paper. A natural way of obtaining such a trade-off between breadth and height of adaptation has recently been presented by Lande (2014). In his quantitative genetic approach, cost and benefit of a plastic continuous trait are used to derive a (Gaussian) tolerance curve with such a trade-off. Although a similar trade-off appears in our scenario, the two approaches differ fundamentally. While Lande (2014) finally derives a tolerance curve that indicates the fitness of a plastic individual or population in a constant environment, we model phenotypic plasticity by applying tolerance curves that change during life.

The environmental gradient we considered is one-dimensional. Multiple or multi-dimensional gradients may be necessary to describe a real scenario but can only be analysed separately with the introduced techniques, and only for the generally unrealistic case that adaptations along the different gradients or dimensions are independent of each other. For scenarios where adaptations along different gradients or dimensions are not independent of each other, it seems however straightforward to generalize the model framework for multidimensional tolerance curves.

For the scenario we analysed in detail, the environment was modelled as a stress-free state that is interrupted by stress events with constant frequency and duration, but our general framework can be applied to all kinds of environmental scenarios. These include fast or stochastic fluctuations or continuous change of the environment, i.e., scenarios to which the previous model for instantaneous reversible plasticity could not be applied because it required a non-changing environment during adaptation delays. Pfab (2012) examined different stochastic distributions for the temporal distribution of stressful and stress-free states. For a wide parameter range, the different distributions did not considerably effect the results for continuous reversible plasticity. However,

note that our way of calculating the fitness for continuous reversible plasticity can readily be transferred to scenarios with periodically changing environments. For our scenario, it is straightforward to additionally distribute the stress-free environmental state in the same way as the stress intensities. Furthermore, the introduced techniques can be used to calculate the fitness in a continuously changing environment and in a predefined (e.g. via simulation) set of environmental changes.

An interesting consequence of the periodicity of the environment is that for irreversible and instantaneous reversible plasticity the function of the two fully-developed phenotypes can switch, i.e. the induced phenotype becomes less adapted to stress than the non-induced phenotype (see Fig. 4 and the discussion of this graph in Sect. 4). For irreversible plasticity, the optimal parameters and the fitness of the genotype fluctuate (i.e., repeated switches) when regarded as a function of the model parameter p that determines the frequency of environmental change, whereas for instantaneous reversible plasticity, the exchange of functionality of the two phenotypes is only expressed when the times to switch between phenotypes are virtually as long as the times during which a given phenotype is advantageous. However, both cases base on the fact that information about coming environmental changes is available and certain, due to the strict environmental periodicity. (In the online appendix P, we discuss a scenario where, for irreversible plasticity, such behaviour is prevented by an exponential distribution of the environmental changes. In the present model for continuous reversible plasticity, we did not observe such switches.) Although early anticipation of environmental changes is certainly biologically relevant (see the following paragraph), the way they are implied by the model for instantaneous reversible plasticity seems not appropriate for most scenarios since these switches only occur when the durations of environmental periods are similar to the adaptation delays. Environmental changes then trigger a phenotypic change away from the new environment and in the direction of the previous (i.e., the following 'anticipated') environment. In the present model for continuous reversible plasticity, adaptations start immediately after environmental changes, and hence this new model is more robust with respect to environmental fluctuations.

For the analysed scenario, we assumed that phenotypic transformations are directly induced by appearance or cessation of stress. However, real scenarios may not satisfy that assumption. Not only the environment often changes continuously rather than instantaneously, but also phenotypic change can be induced by triggers different from the environmental factor to which the phenotype adapts. The triggering may happen at a different time than the change of the corresponding environmental factor. Especially in periodically changing environments, early anticipation of environmental changes is probably widespread. For example, deciduous trees hibernate without leaves to minimize water loss through transpiration in winter. The fall of leaves is triggered by changing length of the days and takes place already in autumn, a season during which maintenance of water balance is easy and photosynthesis still seems attractive. To capture such scenarios, additional parameters or mechanisms that determine when the phenotype transforms could be introduced. That would presumably not only increase the maximal fitness but also change the optimal parameters. Hence, in a model with early anticipation of environmental changes, the optimal modi of the induced and non-induced tolerance curves could be separated by a greater distance since more

intermediate phenotypes would be expected at times close to environmental changes. One more assumption is that the phenotype at birth is completely induced or non-induced, depending on whether stress is prevalent at birth or not. This assumption is met in order to focus on the presentation of the framework and the comparison of the model to preceding works. On biological grounds it certainly can be more realistic to assume more intermediate phenotypes at birth. It is straightforward to include and optimize parameters that determine the initial phenotype depending on the prevalent environmental state.

The different parameters of the tolerance curve were assumed to change simultaneously and in a linear manner with constant speed. While these assumptions may be appropriate for some scenarios, they might need to be adjusted for others. For example, when we assume that the parameters of the tolerance curve can change independently but the speed with which the peak of the tolerance curve can shift is constant, it might become advantageous to start adaptation to a changed environment by first increasing the breadth of the tolerance curve, then shifting the mode of adaptation, and finally reducing the breadth of adaptation again for the sake of a higher fitness close to the mode of adaptation. In our approach not only the direction of adaptation but also the speed of adaptation was assumed to be constant during a transformation. Although a limitation of the transformation speed generally seems realistic due to physiological constraints, the mechanisms and trade-offs on which the limitation depends can vary. It may be realistic for some scenarios to assume that the speed of adaptation can change depending on the progress of transformation or on other circumstances. For instance, the speed could be maximal at the beginning of a transformation and then decrease. The model by Gabriel et al. (2005) for instantaneous reversible plasticity can be seen as the opposite case where the speed of adaptation after an environmental change is first zero and then suddenly increases to virtually infinity so that an organism adapts instantaneously after a certain delay. Altogether it remains an interesting question how transformations occur in natural scenarios. A general method to describe different courses for the change of the parameters could for instance be to introduce systems of differential equations that define the current change of the phenotype.

Scenarios to which the introduced model for continuous reversible plasticity can be readily applied include different types of irreversible plasticity. Gabriel et al. (2005) modelled irreversible plasticity such that an organism fixes its phenotype at birth according to the prevalent environment and that no further adaptations can occur during life. Our model resembles this case if the adaptation speed in both directions is zero, $v_0 = v_1 = 0$. By choosing the adaptation speed for only one direction to be zero, our model can be additionally applied to scenarios where adaptations in one direction, either towards the induced phenotype or towards the non-induced phenotype, can occur irreversibly during the life of an organism.

The quality of information that organisms possess about the current environment can differ and may strongly affect the results, especially when the undetected variance of the environment is large. For the scenario we analysed here, organisms possess information only about the momentary presence or absence of stress but not about the actual intensity (while genotypes, of course, can be adapted to the general distribution of the environment). In Gabriel et al. (2005), this was referred to as “incomplete information” and was opposed by “complete information”, where exact information

about the current environmental state is available to the organisms. A pleasant feature of the case of incomplete information is that the fitness of a genotype can be obtained analytically and that the genotype parameters can be optimized numerically without (possibly extensive) stochastic simulations. In real scenarios, the quality of information often lies between these two extremes. This fact is, for instance, captured in the model by Tufto (2000), where obtained cues are distributed unbiased with a certain variance around the environmental state. An interesting approach to modelling reversible phenotypic adaptation with uncertain cues about the environmental state that are accumulated during life has been introduced by Fischer et al. (2014). There the environment changes between two discrete states at discrete points in time with certain transition probabilities. At the same time points the phenotype can adjust according to the information gathered about the state of the environment. The fitness is then estimated via survival and fecundity rates that depend on the interaction of phenotype and environment and on direct costs of expressing plasticity. It may be interesting to combine their and our approach, e.g. by Bayesian estimation of the current environment via information collected during life (as in Fischer et al. 2014) together with continuous time, a continuous environmental gradient and a continuously adapting tolerance curve (as in our model).

In this article, performance during different phases of life was assumed to affect the fitness of an organism multiplicatively. That may be a realistic approach for e.g. survival probabilities, while other properties, like fecundity, may affect fitness in an additive way. The multiplicative and additive approach have been compared before for different models of plasticity (Utz et al. 2014) without observing qualitative differences in the results. However, it cannot be ruled out that additive fitness contributions may gain importance in our continuous model. Note that the assumption of constant life length does not necessarily affect the generality of our model because, when calculating the fitness, we do not need to literally refer to single lives but we can rather refer to an average fitness in a given sequence of environmental changes.

We optimized genotype parameters and hence focused on adaptive phenotypic plasticity. However, non-adaptive phenotypic plasticity, where phenotypic changes cannot be interpreted as an adaptation to the environment, is found in all kinds of organisms and can be modelled by our approach as well. In particular, mode and breadth of the tolerance curve and other model parameters such as the area under the tolerance curve or the adaptation speed itself can be changed in a non-adaptive way. Such phenotypic change can be caused by various external influences as well as inner processes like ageing or random events and obviously is an important factor in the life of an organism.

After these considerations, it will be interesting to compare the assumptions and expectations of our model to real scenarios and to data gained from field research or laboratory experiments. For this it seems important to carefully study the biology of the organisms of interest and modify the model according to the points mentioned, i.e. to implement additional assumptions on the shape of the tolerance curve, the way of adaptation and the cost and constraints of plasticity. See for example DeWitt et al. (1998) for a general review on costs and limitations of phenotypic plasticity. In order to experimentally investigate how environmental tolerance of organisms changes during life, one has to measure performance of individuals in changing environments

repeatedly. Although measuring fitness can be difficult in practice, it might be possible in some cases to find easily estimated fitness proxies that can be used for parameter estimation and verification and adaptation of our theory.

Most important is probably that our approach to phenotypic plasticity can be embedded into other models. Besides using the model to estimate the performance of a genotype in a certain environment, the framework can for example be implemented into ecological agent-based models where organisms have individual tolerance curves. Another interesting approach for modelling population dynamics in a changing environment may be to consider a plastic tolerance curve of a whole population and to describe properties of the population like the growth rate or carrying capacity with the values of the tolerance curve in the given environment. For a quantitative genetic approach to model the dynamics and genetic evolution of a plastic population in a changing environment see Chevin et al. (2010) where the concept of "reaction norms" is used to describe phenotypic plasticity. Chevin et al. (2013) offer a review of recent theory on the interplay of phenotypic plasticity, genetic evolution and demography in environments that change in time and space. A connection between the impact of plasticity on the fitness of a single organism and on the fitness of whole populations has been made by Lynch and Gabriel (1987) who showed that the environmental fitness dependency of a population of individuals that have Gaussian tolerance curves again has approximately Gaussian shape. The idea of continuously changing tolerance curves could also be applied to evolutionary scenarios where a population of (not necessarily plastic organisms) adapts to a changing environment over many generations. Furthermore, our theory of continuous reversible phenotypic plasticity may not only be applied to scenarios from ecology but from different fields of biology. For example the performance of single organs or cells could be modelled with plastic tolerance curves in order to implement our approach into models for physiology, cell biology, neurobiology or molecular biology.

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Appendix A

A.1 Calculating the fitness of a continuously reversibly plastic genotype

In this appendix we derive a formula for the fitness of a genotype in the scenario introduced in Sects. 2 and 3. That includes that the peak $(m, \frac{1}{\sqrt{2\pi}b})$ of a normalized tolerance curve shifts with constant speed v_1 (respectively v_0) towards the peak of an induced (respectively non-induced) tolerance curve in order to adjust the phenotype to a periodically changing environment. A stress-free environmental state is interrupted by stress events with frequency p and the stress events are assumed to have length t and their stress intensities are randomly distributed with mean $\overline{\phi}$ and standard deviation σ_ϕ. Individuals are born at uniformly distributed times and we will use the law of large numbers to average out the stochasticity in the individual lives and calculate

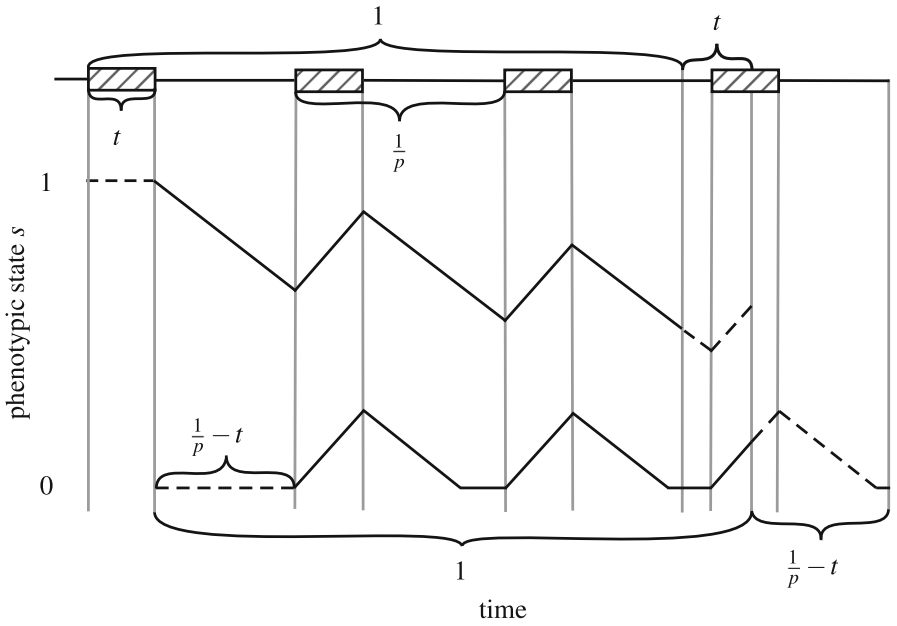

Fig. 7 Change of the phenotypic state s over time. The *striped boxes* symbolise stress events and the lines in between are stress-free times. The *upper course* describes lives that begin during a stress event, and the *lower course* describes lives that begin during a stress-free time. *Dashed lines* indicate phases that are only partly experienced by a given individual, depending on the exact time of birth, whereas *solid lines* indicate phases that are experienced completely by all individuals, depending only on whether their life starts during stress or not

the fitness of the genotype. We then maximize the fitness numerically by finding optimal parameter values for the induced and non-induced tolerance curve. To increase comprehensibility of this section, the reader is referred to a list of notations and abbreviations at the end of this appendix.

The following calculations base on the fact that—due to the assumption that phenotypes are fully developed at birth—all organisms born during stress (and likewise all organisms born during stress-free times) have a common course of the phenotypic state for part of their lives given the same sequence of stress events and given the stress frequency is high enough (see Fig. 7). Since the phenotypic state s [definition (6)] changes both the mode m_s and the breadth b_s of the tolerance curve, we introduce the following shorthand notation for the phenotype (m_s, b_s),

$$mb(s) = m_s, b_s. \tag{8}$$

A.2 Calculating the fitness in a simplified scenario

The stress intensities of all stress events are first assumed to be constant ψ, that is $\sigma_\phi = 0$. We define environmental states $\Phi(\gamma)$ by environmental settings

$$\gamma \in \{0, 1\}, \tag{9}$$

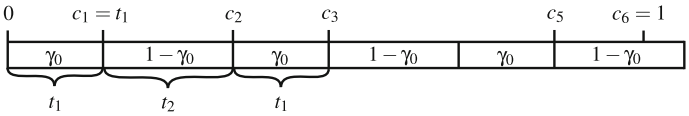

Fig. 8 The environmental course shown in Fig. 7 is abstracted into a sequence of phases with alternating environmental setting (γ_0 and $1-\gamma_0$) and length (t_1 and t_2). In this example, the interval $[t_1, 1[$ is divided into $k = 5$ intervals $A_j = [q_j, q_{j+1}[$ with alternating environmental setting [cf. definition (13)]

where stress is indicated by $\gamma = 1$ and the stress-free environment by $\gamma = 0$,

$$\Phi(\gamma) = \begin{cases} \psi & \text{if } \gamma = 1 \\ 0 & \text{if } \gamma = 0. \end{cases} \tag{10}$$

In order to avoid repeating similar calculations, we consider a sequence of phases with alternating environmental settings and lengths t_1 and t_2 (see Fig. 8). It is arbitrary whether the phases of length t_1 have environmental setting $\gamma = 1$ and the phases of length t_2 have environmental setting $\gamma = 0$ or vice versa. The environmental setting of the phases with length t_1 is denoted by γ_0 and the environmental setting of the phases with length t_2 is $1-\gamma_0$. The length of a life was set to be 1 and for the time being we additionally assume that the length of the first phase is not longer than lifetime, $t_1 \leq 1$. We assume that organisms are born at uniformly distributed points during the first phase.

With this simplification, the lives of all organisms lie within the interval $[0, 1+t_1[$. According to formula (3) in the main text the fitness of an individual is calculated by dividing the interval $[0, 1+t_1[$ into subintervals with constant phenotypic state and environmental setting, and multiplying the performance during each subinterval, raised to the power of the subintervals length (since life length is 1). Naturally, subintervals during which the individual is not alive do not contribute to the fitness. Since the fitness of an individual is calculated by the geometric mean of the contribution of the different phases of life, we can formally assume that the fitness during subintervals that are not experienced is 1. According to formula (5), the fitness of the genotype (in short: the genotype fitness) is the geometric mean of the fitness of $n \to \infty$ individuals.

This is equivalent to first dividing the interval $[0, 1+t_1[$ into a partition $\mathbb{A}$; then for each subinterval $A \in \mathbb{A}$, calculating the performance of a given individual during the subinterval; then calculating the geometric mean of such performances of $n \to \infty$ individuals (we term that the interval's contribution to the genotype fitness $\tilde{w}(A)$); and finally assembling the genotype fitness as

$$\tilde{w}([0, 1+t_1[) = \prod_{A \in \mathbb{A}} \tilde{w}(A). \tag{11}$$

We divide the interval $[0, 1+t_1[$ into three subintervals:

- $[0, t_1[$: Individuals are born at uniformly distributed points during that interval.
- $[t_1, 1[$: All individuals experience that interval completely.
- $[1, 1+t_1[$: Individuals die at uniformly distributed points during that interval.

These intervals are again divided such that each change of the environmental setting marks a division point. Once more we divide these smaller intervals into intervals during which the phenotype changes and intervals during which the phenotype stays constant. The genotype fitness contribution of these smallest intervals are the building blocks of which we will assemble the genotype fitness.

A.2.1 Interval $[0, t_1[$

The time of birth u_i of an individual is uniformly distributed on $[0, t_1[$ and independent of the time of birth of other individuals. The environmental setting at birth, and consequently the phenotypic state during this first interval, is γ_0. Therefore the contribution of the interval $[0, t_1[$ to the fitness of that individual is $f(mb(\gamma_0), \Phi(\gamma_0))^{t_1-u_i}$, and the contribution of the interval $[0, t_1[$ to the genotype fitness is

$$\begin{aligned}\tilde{w}([0, t_1[) &= \lim_{n\to\infty} \prod_{i=1}^{n} \left(f(mb(\gamma_0), \Phi(\gamma_0))^{t_1-u_i}\right)^{\frac{1}{n}} \\ &= f(mb(\gamma_0), \Phi(\gamma_0))^{\lim_{n\to\infty} \sum_{i=1}^{n} \frac{1}{n}(t_1-u_i)} \\ &\overset{(\star)}{=} f(mb(\gamma_0), \Phi(\gamma_0))^{E[t_1-u_i]} \\ &= f(mb(\gamma_0), \Phi(\gamma_0))^{\frac{1}{2}t_1}, \end{aligned} \tag{12}$$

where $(\star)$ holds by the law of large numbers.

A.2.2 Interval $[t_1, 1[$

Independently of the time of birth, all individuals experience the interval $[t_1, 1[$ completely and with the same course of the phenotypic state (consider one of the two solid courses in Fig. 7). Therefore, the genotype fitness contribution of that interval equals the contribution of the interval to the fitness of one individual. To obtain that contribution, we divide the interval $[t_1, 1[$ into subintervals

$$A_j = [q_j, q_{j+1}[\tag{13}$$

with altering environmental settings. The number k of such subintervals can be obtained by adding the number of intervals with environmental setting $1 - \gamma_0$ that start during $[t_1, 1[$ to the number of intervals with environmental setting γ_0 that start during $[t_1 + t_2, 1[$,

$$k = \min\{n \in \mathbb{N} : n(t_1 + t_2) \geq 1 - t_1\} + \min\{n \in \mathbb{N} : n(t_1 + t_2) \geq 1 - t_1 - t_2\} \tag{14}$$

The left boundary of the first interval A_1 is $q_1 = t_1$ and the right boundary of the last interval A_k is $q_{k+1} = 1$. The other boundaries q_j (for $j = 2, \ldots, k$) can be calculated recursively,

$$q_1 = t_1,$$
$$\text{for } j = 2, \ldots, k: \; q_j = q_{j-1} + \begin{cases} t_2 & \text{if } j \text{ even} \\ t_1 & \text{if } j \text{ odd,} \end{cases}$$
$$q_{k+1} = 1. \tag{15}$$

The environmental setting during the phase A_j is,

$$\gamma(A_j) = \begin{cases} \gamma_0 & \text{if } j \text{ even} \\ 1 - \gamma_0 & \text{if } j \text{ odd.} \end{cases} \tag{16}$$

We use the shorthand notations $\gamma_j = \gamma(A_j)$ and $\phi_j = \Phi(\gamma_j)$.

During the environmental setting γ, let $\vartheta(\gamma)$ be the rate with which the phenotypic state changes until the target state γ is reached. This rate is equivalent to the signed speed with which the peak of the tolerance curve shifts, measured relative to the Euclidean distance between the peaks of the tolerance curves of the induced and the non-induced phenotype,

$$\vartheta(\gamma) = \left((m_1 - m_0)^2 + \left(\frac{1}{\sqrt{2\pi} b_1} - \frac{1}{\sqrt{2\pi} b_0} \right)^2 \right)^{-\frac{1}{2}} \cdot \begin{cases} v_1 & \text{if } \gamma = 1 \\ -v_0 & \text{if } \gamma = 0. \end{cases} \tag{17}$$

We use the shorthand notation $\vartheta_j = \vartheta(\gamma_j)$.

The phenotypic state $s(q_1)$ at time q_1 is identical to the phenotypic state γ_0 at birth. The phenotypic states $s(q_j)$ at the other boundary points can be calculated recursively by adding the product of the adaptation rate ϑ_{j-1} and the length $|A_{j-1}|$ of the preceding interval to the phenotypic state $s(q_{j-1})$ at the previous boundary point, and constraining to the interval [0, 1],

$$s(q_1) = \gamma_0$$
$$\text{for } j \neq 1: s(q_j) = \max\{0, \min\{1, s(q_{j-1}) + \vartheta_{j-1}|A_{j-1}|\}\}. \tag{18}$$

We use the shorthand notation $s_j = s(q_j)$.

With the phenotypic states s_j and s_{j+1} we can calculate the time $\delta(A_j)$ of transformation during the interval A_j,

$$\delta(A_j) = \min \left\{ |A_j|, \frac{s_{j+1} - s_j}{\vartheta_j} \right\}. \tag{19}$$

We use the shorthand notation $\delta_j = \delta(A_j)$.

The phases with constant phenotype are infinitesimally short during a transformation. We divide the transformation interval $[q_j, q_j + \delta_j[$ into $n \to \infty$ equal steps. One step has length $\frac{\delta_j}{n}$ and the phenotypic state during the q'th step is $\frac{n-q}{n} s_j + \frac{q}{n} s_{j+1}$. Hence the genotype fitness contribution of the interval is

$$\tilde{w}([q_j, q_j + \delta_j[) = \lim_{n\to\infty} \prod_{r=1}^{n} f\left(mb\left(\frac{n-r}{n}s_j + \frac{r}{n}s_{j+1}\right), \phi_j\right)^{\frac{\delta_j}{n}}$$
$$= g(mb(s_j), mb(s_{j+1}), \phi_j)^{\delta_j} \tag{20}$$

The function g is defined in a general form in formula (4) in Sect. 2 and further derived in Sect. A.4, whereby there additionally the area under the tolerance curve and the environmental state is changing linearly. For the application here those parameters are constant and hence their initial and terminal parameters are equal. Note again that the area c under the curve is not included in the formulas here because it is normalized and the environmental state is represented by a single parameter since it stays constant during the considered phase.

During the remaining part $[q_j + \delta_j, q_{j+1}[$ of the interval A_j, the phenotypic state is constant γ_j and hence the fitness contribution of the interval $[q_j + \delta_j, q_{j+1}[$ is

$$\tilde{w}([q_j + \delta_j, q_{j+1}[) = f(mb(\gamma_j), \phi_j)^{|A_j|-\delta_j}. \tag{21}$$

Finally, the contribution of an interval A_j can be assembled from its two parts,

$$\tilde{w}(A_j) = \tilde{w}([q_j, q_{j+1}[) = \tilde{w}([q_j, q_j + \delta_j[)\tilde{w}([q_j + \delta_j, q_{j+1}[) \tag{22}$$

and the contribution of the entire interval $[t_1, 1[$ is

$$\tilde{w}([t_1, 1[) = \prod_{j=1}^{k} \tilde{w}(A_j). \tag{23}$$

A.2.3 Interval $[1, 1 + t_1[$

Since the lifetime of an organism is 1 and the time of birth u_i is uniformly distributed on $[0, t_1[$, an individual lives until the time $u_i + 1$, which is uniformly distributed on $[1, 1 + t_1[$. Because both the interval $[1, 1 + t_1[$ and the phases with environmental setting γ_0 have length t_1, the interval $[1, 1 + t_1[$ can include at most three subintervals with alternating environmental settings,

$$A'_j = [q'_j, q'_{j+1}[. \tag{24}$$

The boundaries of the subintervals are:

$$\begin{aligned} q'_1 &= q_{k+1} = 1 \\ q'_2 &= \min\{1 + t_1, q_{k-1} + t_1 + t_2\} \\ q'_3 &= \min\{1 + t_1, q_k + t_1 + t_2\} \\ q'_4 &= 1 + t_1. \end{aligned} \tag{25}$$

For the case $k = 0$ (that is when $t_1 = 1$) or $k = 1$, we formally define $q_{-1} = -t_2$ and $q_0 = 0$. Note that $|A'_1| = 0$ ($q'_1 = q'_2$) if $\frac{1}{t_1+t_2} \in \mathbb{N}$ or $\frac{1-t_1}{t_1+t_2} \in \mathbb{N}_0$. The other intervals A'_j can have length 0, too, depending on the values for t_1 and t_2.

The environmental setting during the interval A'_1 is the same as the environmental setting γ_k at the end of $[t_1, 1[$, and the environmental settings of the intervals A'_j alter,

$$\gamma(A'_j) = \begin{cases} \gamma_k & \text{if } j \text{ odd} \\ 1 - \gamma_k & \text{if } j \text{ even.} \end{cases} \tag{26}$$

We use analogous shorthand notations as before, $\gamma'_j = \gamma(A'_j)$, $\vartheta'_j = \vartheta(\gamma'_j)$ and $\phi'_j = \Phi(\gamma'_j)$.

Since $q'_1 = q_{k+1}$, the phenotypic state $s(q'_1)$ is s_{k+1} and the phenotypic states at the succeeding boundaries are calculated analogous to formula (18),

$$\begin{aligned} s(q'_1) &= s_{k+1} \\ \text{for } j \neq 1: \; s(q'_j) &= \max\{0, \min\{1, s(q'_{j-1}) + \vartheta'_{j-1}|A'_{j-1}|\}\}. \end{aligned} \tag{27}$$

We use the shorthand notation $s'_j = s(q'_j)$.

Analogous to formula (19), the phenotype is transforming during the interval A'_j for a time

$$\delta(A'_j) = \min\left\{|A'_j|, \frac{s'_{j+1} - s'_j}{\vartheta'_j}\right\}. \tag{28}$$

We use the shorthand notation $\delta'_j = \delta(A'_j)$.

The probability that the point $u_i + 1$ where a given life ends lies in the interval $[q'_j, q'_j + \delta'_j[$ during which the phenotype is transforming is

$$P(u_i + 1 \in [q'_j, q'_j + \delta'_j[) = \frac{\delta'_j}{t_1}. \tag{29}$$

Given that an individual's life ends during the transformation $[q'_j, q'_j + \delta'_j[$, the exact time when the life ends is uniformly distributed on this interval. We divide the transformation into $n \to \infty$ steps. The step length is $\frac{\delta'_j}{n}$ and the contribution of the interval $[q'_j, q'_j + \delta'_j[$ to the fitness of an individual whose life ends at the i'th step is

$$\lim_{n\to\infty} \prod_{r=1}^{i} f\left(mb\left(\frac{n-r}{n}s'_j + \frac{r}{n}s'_{j+1}\right), \phi'_j\right)^{\frac{\delta'_j}{n}}. \tag{30}$$

We consider $n \to \infty$ individuals and due to the law of large numbers we can assume that during each step the life of one individual ends. Therefore the interval's genotype fitness contribution, under the condition that life ends during this interval, is

$$\begin{aligned}
&\tilde{w}([q'_j, q'_j + \delta'_j[: u_i + 1 \in [q'_j, q'_j + \delta'_j[) \\
&= \lim_{n\to\infty} \prod_{i=1}^{n} \left(\prod_{r=1}^{i} f\left(mb\left(\frac{n-r}{n} s'_j + \frac{r}{n} s'_{j+1} \right), \phi'_j \right)^{\frac{\delta'_j}{n}} \right)^{\frac{1}{n}} \\
&= h(mb(s'_j), mb(s'_{i+j}), \phi'_j)^{\delta'_j} \qquad (31)
\end{aligned}$$

The function h is derived in a generalized form in Sect. A.5. As for the general formula for g, there additionally the area under the tolerance curve and the environmental state is changing linearly. Again for the application here those parameters are constant and hence their initial and terminal parameters are equal. As before the area c under the curve is not included in the formulas because it is normalized and the environmental state is represented by a single parameter since it stays constant during the considered phase.

The probability that a given individual is alive after the interval $[q'_j, q'_j + \delta'_j[$ is

$$P(u_i + 1 \geq q'_j + \delta'_j) = \frac{1 + t_1 - (q'_j + \delta'_j)}{t_1}. \qquad (32)$$

Under the condition that all individuals are alive after the transformation $[q'_j, q'_j + \delta'_j[$, the interval's genotype fitness contribution is according to formula (20),

$$\tilde{w}([q'_j, q'_j + \delta'_j[: u_i + 1 \geq q'_j + \delta'_j) = g(mb(s'_j), mb(s'_{j+1}), \phi'_j)^{\delta'_j}. \qquad (33)$$

The unconditioned genotype fitness contribution of the interval $[q'_j, q'_j + \delta'_j[$ is then the product of the two conditioned contributions, each raised to the corresponding probability,

$$\begin{aligned}
\tilde{w}([q'_j, q'_j + \delta'_j[) = \tilde{w}([q'_j, q'_j + \delta'_j[: u_i + 1 \in [q'_j, q'_j + \delta'_j[)^{P(u_i+1\in[q'_j, q'_j+\delta'_j[)} \\
\cdot \tilde{w}([q'_j, q'_j + \delta'_j[: u_i + 1 \geq q'_j + \delta'_j)^{P(u_i+1\geq q'_j+\delta'_j)}. \qquad (34)
\end{aligned}$$

During the remaining part $[q'_j + \delta'_j, q'_{j+1}[$ of the interval A'_j the phenotypic state is constant γ'_j.

The probability that a given life ends during that part is

$$P(u_i + 1 \in [q'_j + \delta'_j, q'_{j+1}[) = \frac{q'_{j+1} - (q'_j + \delta'_j)}{t_1}. \qquad (35)$$

Under the condition that all lives end during the interval $[q'_j + \delta'_j, q'_{j+1}[$, the individual lives end at points that are uniformly distributed on this interval. The interval's genotype fitness contribution can hence be derived analogously to formula (12),

$$\tilde{w}([q'_j + \delta'_j, q'_{j+1}[: u_i + 1 \in [q'_j + \delta'_j, q'_{j+1}[) = f(mb(\gamma'_j), \phi'_j)^{\frac{1}{2}(|A'_j| - \delta'_j)}. \qquad (36)$$

The probability that a given individual is alive after the interval $[q'_j + \delta'_j, q'_{j+1}[$ is

$$P(u_i + 1 \geq q'_{j+1}) = \frac{1 + t_1 - q'_{j+1}}{t_1}. \tag{37}$$

Under the condition that individuals are alive after the interval $[q'_j + \delta'_j, q'_{j+1}[$, the interval's genotype fitness is

$$\tilde{w}([q'_j + \delta'_j, q'_{j+1}[: u_i + 1 \geq q'_{j+1}[) = f(mb(\gamma'_j), \phi'_j)^{|A'_j| - \delta'_j}. \tag{38}$$

The unconditioned genotype fitness contribution of the interval $[q'_j + \delta'_j, q'_{j+1}[$ is therefore

$$\begin{aligned} &\tilde{w}([q'_j + \delta'_j, q'_{j+1}[) \\ &\quad = \tilde{w}([q'_j + \delta'_j, q'_{j+1}[: u_i + 1 \in [q'_j + \delta'_j, q'_{j+1}[)^{P(u_i+1 \in [q'_j+\delta'_j, q'_{j+1}[)} \\ &\qquad \cdot \tilde{w}([q'_j + \delta'_j, q'_{j+1}[: u_i + 1 \geq q'_{j+1})^{P(u_i+1 \geq q'_{j+1})}. \end{aligned} \tag{39}$$

Finally, the fitness contribution of an interval A'_j can be assembled from its two parts,

$$\tilde{w}(A'_j) = \tilde{w}([q'_j, q'_j + \delta'_j[) \cdot \tilde{w}([q'_j + \delta'_j, q'_{j+1}[), \tag{40}$$

and the genotype fitness contribution of the entire interval $[1, 1 + t_1[$ is

$$\tilde{w}([1, 1 + t_1[) = \prod_{j=1}^{3} \tilde{w}(A'_j). \tag{41}$$

A.2.4 The complete interval $[0, t_1 + 1[$

We now assemble the genotype fitness contributions of the subintervals and so obtain the genotype fitness. It is a function of the lengths t_1 and t_2 of the phases with alternating environmental setting and the environmental setting γ_0 of the first phase. We still consider the case that $t_1 \leq 1$,

$$\tilde{w}_{t_1 \leq 1}(t_1, t_2, \gamma_0) = \tilde{w}([0, 1 + t_1[) = \tilde{w}([0, t_1[) \cdot \tilde{w}([t_1, 1[) \cdot \tilde{w}([1, 1 + t_1[). \tag{42}$$

A.3 Generalization to the original scenario

We now relax the assumptions of Sect. A.2 and generalize the introduced method to calculate the genotype fitness for the original scenario where the first phase can last longer then a lifetime ($t_1 > 1$) and stress intensities are distributed randomly ($\sigma_\phi > 0$), see Sect. 2.

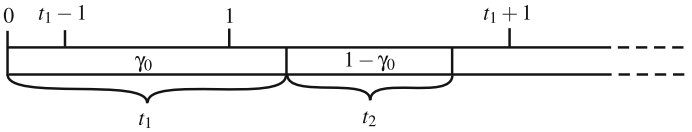

Fig. 9 Sequence of phases with alternating environmental settings (γ_0 and $1 - \gamma_0$) and lengths (t_1 and t_2). When $t_1 > 1$, individuals born during $[0, t_1 - 1[$ experience the environmental setting γ_0 during their whole life

A.3.1 Generalization for $t_1 > 1$

We excluded the case $t_1 > 1$ (see Fig. 9). In that case, organisms born during $[0, t_1 - 1[$ experience the environmental setting γ_0 during their whole life and consequently have the fitness $f(mb(\gamma_0), \Phi(\gamma_0))$. Individuals that are born during $[t_1 - 1, t_1[$ experience the same environmental distribution as if the length of the first phase with constant environmental setting was 1 instead of t_1. Under the condition that all individuals are born during that interval, the genotype fitness is therefore $\tilde{w}_{t_1 \leq 1}(1, t_2, \gamma_0)$. The general genotype fitness is then the product of the conditioned genotype fitness expressions, each raised to the power of the birth probability in the according interval, $\frac{t_1 - 1}{t_1}$ or $\frac{1}{t_1}$. For all $t_1 > 0$, the generalized formula for the genotype fitness is hence

$$\tilde{w}_{t_1 > 0}(t_1, t_2, \gamma_0) = \begin{cases} \tilde{w}_{t_1 \leq 1}(t_1, t_2, \gamma_0) & \text{if } t_1 \leq 1 \\ f(mb(\gamma_0), \Phi(\gamma_0))^{\frac{t_1 - 1}{t_1}} \tilde{w}_{t_1 \leq 1}(1, t_2, \gamma_0)^{\frac{1}{t_1}} & \text{if } t_1 > 1. \end{cases} \tag{43}$$

A.3.2 Generalization for $\sigma_\phi > 0$

We now relax the assumption that all stress events have the same stress intensity ($\sigma_\phi = 0$) and instead assume a random distribution of stress intensities with known expectation value $\overline{\phi}$ and variance σ_ϕ^2. In the formulas, we can simply replace the stress intensity ψ by $\overline{\phi}$ and ψ^2 by $\sigma_\phi^2 + \overline{\phi}^2$ after expanding all terms that are connected to ψ. This can be verified by calculating the genotype fitness contribution of a stressful interval A with constant phenotype (m, b) as the geometric mean of the contribution of A to the fitness of $n \to \infty$ individuals with independently drawn stress intensities ψ_i,

$$\begin{aligned} \tilde{w}(A) &= \lim_{n \to \infty} \prod_{i=1}^{n} f(m, b, \psi_i)^{\frac{1}{n}|A|} \\ &= \lim_{n \to \infty} \prod_{i=1}^{n} \left(\frac{1}{\sqrt{2\pi} b} \mathrm{e}^{-\frac{1}{2} \frac{m^2 - 2m\psi_i + {\psi_i}^2}{b^2}} \right)^{\frac{1}{n}|A|} \\ &= \frac{1}{\sqrt{2\pi} b} \mathrm{e}^{-\frac{1}{2} \frac{\frac{1}{n} \sum_{i=1}^{n} \left(m^2 - 2m\psi_i + {\psi_i}^2\right)}{b^2} |A|} \\ &\overset{(\star)}{=} \frac{1}{\sqrt{2\pi} b} \mathrm{e}^{-\frac{1}{2} \frac{m^2 - 2m\overline{\phi} + \sigma_\phi^2 + \overline{\phi}^2}{b^2} |A|}, \end{aligned} \tag{44}$$

where $(\star)$ holds by the law of large numbers.

Technically, the terms ψ and ψ^2 are substituted by $\overline{\phi}$ and $\sigma_\phi^2 + \overline{\phi}^2$, respectively, in formula (1) of the tolerance curve f; then the formulas for g (20) and h (31) need to be recalculated. Alternatively, the exchange of the terms can be realized directly in the formulas for the functions f, g and h. In all other formulas the terms with ψ only interact additively and hence they do not need to be changed directly. Note that stochasticity of the stress-free state could be introduced in the same way.

A.3.3 The original temporal distribution of stress

We can finally assemble the fitness of a genotype in the originally introduced environmental distribution, where stress events occur with frequency p and persist for a time t (hence the gaps between the stress events have length $\frac{1}{p} - t$), and where the individual times of birth are uniformly distributed within the sequence of stress events and stress-free times. Under the condition that all organisms are born during a stress event, the exact times of birth are uniformly distributed on the stress event. The genotype fitness under that condition is obtained by formula (43) with $t_1 = t$, $t_2 = \frac{1}{p} - t$ and $\gamma_0 = 1$. Given all organisms are born during the gap between two stress events, the exact times of birth are uniformly distributed on the gap. The genotype fitness under that condition is formula (43) with $t_1 = \frac{1}{p} - t$, $t_2 = t$ and $\gamma_0 = 0$. Altogether, the genotype fitness (here with all parameters as arguments) is then,

$$\begin{aligned} &w(p, t, \overline{\phi}, \sigma_\phi, v_0, v_1, m_0, m_1, b_0, b_1) \\ &\quad = \tilde{w}_{t_1>0}\left(t, \frac{1}{p} - t, 1\right)^{pt} \cdot \tilde{w}_{t_1>0}\left(\frac{1}{p} - t, t, 0\right)^{1-pt}. \end{aligned} \tag{45}$$

where pt and $1 - pt$ are the probabilities for a birth during stress or during a stress-free time, respectively.

A.4 Derivation of the function g

Here, we derive an expression for the function g, formula (4) in Sect. 2 in the main text. The formula is applied in the calculation (20) where the area under the tolerance curve is normalized, $c_a = c_z = 1$, and the environmental state does not change during the transformation, $\phi_a = \phi_z = \phi$. In (20) we hence use the short-hand notation $g(m_a, b_a, m_z, b_z, \phi)$ or $g(mb(a), mb(z), \phi)$. The general formula is

$$\begin{aligned} &g\,(m_a, b_a, c_a, \phi_a, m_z, b_z, c_z, \phi_z) \\ &= \lim_{n\to\infty} \prod_{r=1}^{n} f\left(\frac{n-r}{n} m_a + \frac{r}{n} m_z, \frac{1}{\frac{n-r}{n}\frac{1}{b_a} + \frac{r}{n}\frac{1}{b_z}}, \frac{n-r}{n} c_a + \frac{r}{n} c_z, \frac{n-r}{n}\phi_a + \frac{r}{n}\phi_z\right)^{\frac{1}{n}} \\ &= \lim_{n\to\infty} \prod_{r=1}^{n} \left(\frac{\left(\frac{n-r}{n} c_a + \frac{r}{n} c_z\right)\left(\frac{n-r}{n}\frac{1}{b_a} + \frac{r}{n}\frac{1}{b_z}\right)}{\sqrt{2\pi}} \mathrm{e}^{-\frac{1}{2}\left(\frac{n-r}{n} m_a + \frac{r}{n} m_z - \left(\frac{n-r}{n}\phi_a + \frac{r}{n}\phi_z\right)\right)^2 \left(\frac{n-r}{n}\frac{1}{b_a} + \frac{r}{n}\frac{1}{b_z}\right)^2}\right)^{\frac{1}{n}} \end{aligned}$$

$$= \frac{1}{\sqrt{2\pi}} \lim_{n\to\infty} \prod_{r=1}^{n} \left(\left(\frac{n-r}{n} c_a + \frac{r}{n} c_z \right)^{\frac{1}{n}} \left(\frac{n-r}{n} \frac{1}{b_a} + \frac{r}{n} \frac{1}{b_z} \right)^{\frac{1}{n}} \right) \cdot$$
$$\cdot \mathrm{e}^{-\frac{1}{2} \lim_{n\to\infty} \sum_{r=1}^{n} \frac{1}{n} \left(\frac{n-r}{n} m_a + \frac{r}{n} m_z - \left(\frac{n-r}{n} \phi_a + \frac{r}{n} \phi_z \right) \right)^2 \left(\frac{n-r}{n} \frac{1}{b_a} + \frac{r}{n} \frac{1}{b_z} \right)^2}$$
$$= \frac{1}{\sqrt{2\pi}} \zeta_{g1} \zeta_{g2} \mathrm{e}^{-\frac{1}{2}\zeta_{g3}} \tag{46}$$

where $\zeta_{g1} = c_a$ if $c_a = c_z$ and otherwise

$$\begin{aligned}
\zeta_{g1} &= \lim_{n\to\infty} \prod_{r=1}^{n} \left(\frac{n-r}{n} c_a + \frac{r}{n} c_z \right)^{\frac{1}{n}} \\
&= \exp\left(\lim_{n\to\infty} \log\left(\prod_{r=1}^{n} \left(\frac{n-r}{n} c_a + \frac{r}{n} c_z \right)^{\frac{1}{n}} \right) \right) \\
&= \exp\left(\lim_{n\to\infty} \sum_{r=1}^{n} \frac{1}{n} \log\left(\frac{n-r}{n} c_a + \frac{r}{n} c_z \right) \right) \\
&= \exp\left(\frac{1}{c_z - c_a} \int_{c_a}^{c_z} \log(x)\, \mathrm{d}x \right) \\
&= \exp\left(\frac{1}{c_z - c_a} \left(c_z \log(c_z) + c_a - c_a \log(c_a) - c_z \right) \right).
\end{aligned} \tag{47}$$

Accordingly $\zeta_{g2} = \frac{1}{b_a}$ if $b_a = b_z$ and otherwise

$$\begin{aligned}
\zeta_{g2} &= \lim_{n\to\infty} \prod_{r=1}^{n} \left(\frac{n-r}{n} \frac{1}{b_a} + \frac{r}{n} \frac{1}{b_z} \right)^{\frac{1}{n}} \\
&= \exp\left(\frac{1}{\frac{1}{b_z} - \frac{1}{b_a}} \left(\frac{1}{b_a} \log(b_a) + \frac{1}{b_a} - \frac{1}{b_z} \log(b_z) - \frac{1}{b_z} \right) \right),
\end{aligned} \tag{48}$$

and

$$\begin{aligned}
\zeta_{g3} &= \lim_{n\to\infty} \sum_{r=1}^{n} \frac{1}{n} \left(\frac{n-r}{n} m_a + \frac{r}{n} m_z - \left(\frac{n-r}{n} \phi_a + \frac{r}{n} \phi_z \right) \right)^2 \left(\frac{n-r}{n} \frac{1}{b_a} + \frac{r}{n} \frac{1}{b_z} \right)^2 \\
&= \int_0^1 \left((1-x) m_a + x m_z - \left((1-x)\phi_a + x\phi_z \right) \right)^2 \left(\frac{1-x}{b_a} + \frac{x}{b_z} \right)^2 \mathrm{d}x \\
&= \frac{1}{30 b_a^2 b_z^2} \Big[b_z^2 (6m_a^2 + m_z^2 - 3m_z\phi_a + 6\phi_a^2 + 3m_a(m_z - 4\phi_a - \phi_z) \\
&\quad -2m_z\phi_z + 3\phi_a\phi_z + \phi_z^2) + b_a b_z (3m_a^2 + 3m_z^2 - 4m_z\phi_a + 3\phi_a^2 \\
&\quad + m_a(4m_z - 6\phi_a - 4\phi_z) - 6m_z\phi_z + 4\phi_a\phi_z + 3\phi_z^2)
\end{aligned}$$

$$+ b_a^2(m_a^2 + 6m_z^2 + \phi_a^2 + m_a(3m_z - 2\phi_a - 3\phi_z) + 3\phi_a\phi_z + 6\phi_z^2 - 3m_z(\phi_a + 4\phi_z))\Big]. \tag{49}$$

A.5 Derivation of the function *h*

Here, we derive an expression for the function h which is introduced in the calculation (20). In the generalized form here, all parameters of the tolerance curve, m, $\frac{1}{b}$ and c, and the environmental state ϕ change linearly from m_a, $\frac{1}{b_a}$, c_a, ϕ_a to m_z, $\frac{1}{b_z}$, c_z, ϕ_z. In the calculation (20) the area under the tolerance curve is normalized, $c_a = c_z = 1$, and the environmental state is not changing during the transformation, $\phi_a = \phi_z = \phi$, and we hence use the short-hand notation $h(m_a, b_a, m_z, b_z, \phi)$ or $h(mb(a), mb(z), \phi)$. The general formula is given by

$$\begin{aligned}
&h\,(m_a, b_a, c_a, \phi_a, m_z, b_z, c_z, \phi_z)\\
&= \lim_{n\to\infty} \prod_{i=1}^{n} \left(\prod_{r=1}^{i} f\left(\frac{n-r}{n} m_a + \frac{r}{n} m_z,\ \frac{1}{\frac{n-r}{n}\frac{1}{b_a} + \frac{r}{n}\frac{1}{b_z}},\ \frac{n-r}{n} c_a + \frac{r}{n} c_z,\ \frac{n-r}{n}\phi_a + \frac{r}{n}\phi_z \right)^{\frac{1}{n}} \right)^{\frac{1}{n}}\\
&= \lim_{n\to\infty} \prod_{i=1}^{n} f\left(\frac{n-i}{n} m_a + \frac{i}{n} m_z,\ \frac{1}{\frac{n-i}{n}\frac{1}{b_a} + \frac{i}{n}\frac{1}{b_z}},\ \frac{n-i}{n} c_a + \frac{i}{n} c_z,\ \frac{n-i}{n}\phi_a + \frac{i}{n}\phi_z \right)^{\frac{n-i}{n^2}}\\
&= \lim_{n\to\infty} \prod_{i=1}^{n} f\left(\frac{i}{n} m_a + \frac{n-i}{n} m_z,\ \frac{1}{\frac{i}{n}\frac{1}{b_a} + \frac{n-i}{n}\frac{1}{b_z}},\ \frac{i}{n} c_a + \frac{n-i}{n} c_z,\ \frac{i}{n}\phi_a + \frac{n-i}{n}\phi_z \right)^{\frac{i}{n^2}}\\
&= \lim_{n\to\infty} \prod_{i=1}^{n} \left(\frac{\left(\frac{i}{n} c_a + \frac{n-i}{n} c_z\right)\left(\frac{i}{n}\frac{1}{b_a} + \frac{n-i}{n}\frac{1}{b_z}\right)}{\sqrt{2\pi}} \mathrm{e}^{-\frac{1}{2}\left(\frac{i}{n} m_a + \frac{n-i}{n} m_z - \left(\frac{i}{n}\phi_a + \frac{n-i}{n}\phi_z\right)\right)^2 \left(\frac{i}{n}\frac{1}{b_a} + \frac{n-i}{n}\frac{1}{b_z}\right)^2} \right)^{\frac{i}{n^2}}\\
&= \frac{1}{\sqrt[4]{2\pi}} \lim_{n\to\infty} \prod_{i=1}^{n} \left(\left(\frac{i}{n} c_a + \frac{n-i}{n} c_z \right)^{\frac{i}{n^2}} \right) \left(\frac{i}{n}\frac{1}{b_a} + \frac{n-i}{n}\frac{1}{b_z} \right)^{\frac{i}{n^2}} \cdot\\
&\quad \cdot \mathrm{e}^{-\frac{1}{2}\lim_{n\to\infty}\sum_{i=1}^{n} \frac{i}{n^2}\left(\frac{i}{n} m_a + \frac{n-i}{n} m_z - \left(\frac{i}{n}\phi_a + \frac{n-i}{n}\phi_z\right)\right)^2 \left(\frac{i}{n}\frac{1}{b_a} + \frac{n-i}{n}\frac{1}{b_z}\right)^2}\\
&= \frac{1}{\sqrt[4]{(2\pi)}} \zeta_{h1}\zeta_{h2}\mathrm{e}^{-\frac{1}{2}\zeta_{h3}}
\end{aligned} \tag{50}$$

where $\zeta_{h1} = \sqrt{c_a}$ if $c_a = c_z$ and otherwise

$$\begin{aligned}
\zeta_{h1} &= \lim_{n\to\infty} \prod_{i=1}^{n} \left(\frac{i}{n} c_a + \frac{n-i}{n} c_z \right)^{\frac{i}{n^2}}\\
&= \exp\left(\lim_{n\to\infty} \log\left(\prod_{i=1}^{n} \left(\frac{i}{n} c_a + \frac{n-i}{n} c_z \right)^{\frac{i}{n^2}} \right) \right)\\
&= \exp\left(\lim_{n\to\infty} \sum_{i=1}^{n} \frac{1}{n}\frac{i}{n} \log\left(\frac{i}{n} c_a + \frac{n-i}{n} c_z \right) \right)
\end{aligned}$$

$$= \exp\left(\frac{1}{c_a - c_z}\int_{c_z}^{c_a} \frac{x - c_z}{c_a - c_z}\log(x)\,\mathrm{d}x\right)$$
$$= \exp\left(\frac{2c_z^2\log(c_z) - (c_a - 3c_z)(c_a - c_z) + 2c_a(c_a - 2c_z)\log(c_a)}{4(c_a - c_z)^2}\right). \quad (51)$$

Accordingly $\zeta_{h2} = \sqrt{\frac{1}{b_a}}$ if $b_a = b_z$ and otherwise

$$\zeta_{h2} = \lim_{n\to\infty}\prod_{i=1}^{n}\left(\frac{i}{n}\frac{1}{b_a} + \frac{n-i}{n}\frac{1}{b_z}\right)^{\frac{i}{n^2}}$$
$$= \exp\left(\frac{4b_ab_z - 3b_a^2 - b_z^2 + 2b_z(2b_a - b_z)\log(b_a) - 2b_a^2\log(b_z)}{4(b_a - b_z)^2}\right), \quad (52)$$

and

$$\zeta_{h3} = \lim_{n\to\infty}\sum_{i=1}^{n}\frac{i}{n^2}\left(\frac{i}{n}m_a + \frac{n-i}{n}m_z - \left(\frac{i}{n}\phi_a + \frac{n-i}{n}\phi_z\right)\right)^2\left(\frac{i}{n}\frac{1}{b_a} + \frac{n-i}{n}\frac{1}{b_z}\right)^2$$
$$= \int_0^1 x\left(xm_a + (1-x)m_z - \left(\frac{i}{n}\phi_a + \frac{n-i}{n}\phi_z\right)\right)^2\left(\frac{x}{b_a} + \frac{1-x}{b_z}\right)^2\mathrm{d}x$$
$$= \frac{1}{60b_a^2b_z^2}\left[2b_ab_z(2m_a^2 + m_z^2 + 2\phi_a^2 + 2m_a(m_z - 2\phi_a - \phi_z) + 2\phi_a\phi_z + \phi_z^2 - 2m_z(\phi_a + \phi_z))\right.$$
$$+ b_z^2(10m_a^2 + m_z^2 + 10\phi_a^2 + 4m_a(m_z - 5\phi_a - \phi_z) + 4\phi_a\phi_z + \phi_z^2 - 2m_z(2\phi_a + \phi_z))$$
$$\left. + b_a^2(m_a^2 + 2m_z^2 + \phi_a^2 + 2m_a(m_z - \phi_a - \phi_z) + 2\phi_a\phi_z + 2\phi_z^2 - 2m_z(\phi_a + 2\phi_z))\right]. \quad (53)$$

A.6 Notations and abbreviations

This list contains a selection of notations and abbreviations. Notations which are only introduced and used in a short context are mostly not listed here.

Notation	Description	Introduction
		Section 2
m	Mode of the tolerance curve	
b	Breadth of the tolerance curve	
c	Area under the tolerance curve	
ϕ	Environmental state	
f	Tolerance curve	(1)
w_i	Fitness of an individual	(3)
g^τ	Fitness contribution of a phase with length τ during which the parameters change linearly	(4), (20)

Notation	Description	Introduction
w	Fitness of the genotype	(5), (45)
		Section 3
m_0, b_0	Mode and breadth of the non-induced tolerance curve	
m_1, b_1	Mode and breadth of the induced tolerance curve	
s	Phenotypic state	
v_0	Adaptation speed of the peak of the tolerance curve in the direction of the peak of the non-induced tolerance curve	
v_1	Adaptation speed of the peak of the tolerance curve in the direction of the peak of the induced tolerance curve	
p	Frequency of stress occurrence	
t	Duration of a stress event	
$\overline{\phi}$	Expected stress intensity	
σ_ϕ	Standard deviation of the stress intensities	
		Section 4
d_0	Instantaneous model: transforming delay from the induced to the non-induced phenotype	
d_1	Instantaneous model: transforming delay from the non-induced to the induced phenotype	
v	Continuous model: adaptation speed of the peak of the tolerance curve in either direction when $v_0 = v_1$	
d	Instantaneous model: transforming delay in either direction when $d_0 = d_1$	
pt	Proportion of time stress is present. Product of p and t	
		Appendix A
$mb(s) = m_s, b_s$	Mode and breadth of the tolerance curve at phenotypic state s	(8)
ψ	Stress intensity	
t_1, t_2	Lengths of phases with constant environmental state	
$\tilde{w}(A)$	Genotype fitness contribution of the interval A of the time line in the simplified scenario, with $t_1 \leq 1$	(11)
$\tilde{w}(A : E)$	Same as $\tilde{w}(A)$ but the individual times of birth are conditioned according to E	
$\tilde{w}_{t_1 \leq 1}(t_1, t_2, \gamma_0)$	Genotype fitness in the simplified scenario with $t_1 \leq 1$	(42)
$\tilde{w}_{t_1 > 0}(t_1, t_2, \gamma_0)$	Genotype fitness in the simplified scenario with arbitrary $t_1 > 0$	(43)
γ	Environmental setting	(9)
$\Phi(\gamma)$	Environmental state as a function of the environmental setting	(10)
A_j	$[q_j, q_{j+1}[$, j'th interval with alternating environmental setting in $[t_1, 1[$	(13)
k	Number of intervals with alternating environmental setting in $[t_1, 1[$	(14)
q_j	j'th boundary point in $[t_1, 1[$ between intervals with alternating environmental setting, left boundary point of A_j	(15)
$\gamma(A_j)$	Environmental setting of the interval A_j	(16)
γ_j	Abbreviation for $\gamma(A_j)$	

Notation	Description	Introduction
ϕ_j	Abbreviation for $\Phi(\gamma_j)$	
$\vartheta(\gamma)$	Adaptation rate of the phenotypic state in the environmental setting γ	(17)
ϑ_j	Abbreviation for $\vartheta(\gamma_j)$	
$s(q_j)$	Phenotypic state at the point q_j, abbreviation: s_j	(18)
$\delta(A_j)$	Time of phenotypic change in the interval A_j, abbreviation: δ_j	(19)
A'_j	$[q'_j, q'_{j+1}[$, j'th interval with alternating environmental setting in $[1, 1+t_1[$. Attention: the A'_j can have length zero!	(24)
q'_j	j'th boundary point in $[1, 1+t_1[$ between intervals with alternating environmental setting, left boundary of A'_j	(25)
$\gamma(A'_j)$	Environmental setting of the interval A'_j	(26)
γ'_j	Abbreviation for $\gamma(A'_j)$	
ϕ'_j	Abbreviation for $\Phi(\gamma'_j)$	
ϑ'_j	Abbreviation for $\vartheta(\gamma'_j)$	
$s(q'_j)$	Phenotypic state at the point q'_j, abbreviation: s'_j	(27)
$\delta(A'_j)$	Time of phenotypic change in the interval A'_j, abbreviation: δ'_j	(28)
h^τ	Fitness contribution of a phase with length τ during which the parameters change linearly and which is experienced up to on its length uniformly distributed points	(31)

References

Chevin LM, Lande R, Mace GM (2010) Adaptation, plasticity, and extinction in a changing environment: towards a predictive theory. PLoS Biol 8(4):e1000,357

Chevin LM, Collins S, Lefèvre F (2013) Phenotypic plasticity and evolutionary demographic responses to climate change: taking theory out to the field. Funct Ecol 27(4):967–979

Davidson AM, Jennions M, Nicotra AB (2011) Do invasive species show higher phenotypic plasticity than native species and if so, is it adaptive? A meta-analysis. Ecol Lett 14:419–431

DeWitt TJ, Sih A, Wilson DS (1998) Costs and limits of phenotypic plasticity. Trends Ecol Evol 13:77–81

Diamond J, Hammond K (1992) The matches, achieved by natural selection, between biological capacities and their natural loads. Experientia 48:551–557

Engel K, Tollrian R, Jeschke JM (2011) Integrating biological invasions, climate change and phenotypic plasticity. Commun Integr Biol 4:247–250

Fischer B, van Doorn GS, Dieckmann U, Taborsky B et al (2014) The evolution of age-dependent plasticity. Am Nat 183:108–125

Gabriel W (1999) Evolution of reversible plastic responses: inducible defenses and environmental tolerance. In: Harvell CD, Tollrian R (eds) The ecology and evolution of inducible defences. Princeton University Press, Princeton, pp 286–305

Gabriel W (2005) How stress selects for reversible phenotypic plasticity. J Evol Biol 18:873–883

Gabriel W (2006) Selective advantage of irreversible and reversible phenotypic plasticity. Archiv für Hydrobiologie 167:1–20

Gabriel W, Luttbeg B, Sih A, Tollrian R (2005) Environmental tolerance, heterogeneity, and the evolution of reversible plastic responses. Am Nat 166:339–353

Haldane JBS, Jayakar SD (1963) Polymorphism due to selection of varying direction. J Genet 58:237–242

Huey RB, Kingsolver JG (1989) Evolution of thermal sensitivity of ectotherm performance. Trends Ecol Evol 4(5):131–135

Key K, Day M (1954) A temperature-controlled physiological colour response in the grasshopper Kosciuscola Tristis Sjost. (Orthoptera: Acrididae). Aust J Zool 2:309–339
Lande R (2009) Adaptation to an extraordinary environment by evolution of phenotypic plasticity and genetic assimilation. J Evol Biol 22(7):1435–1446
Lande R (2014) Evolution of phenotypic plasticity and environmental tolerance of a labile quantitative character in a fluctuating environment. J Evol Biol 27(5):866–875
Lynch M, Gabriel W (1987) Environmental tolerance. Am Nat 129:283–303
Marchinko KB (2003) Dramatic phenotypic plasticity in barnacle legs (*Balanus glandula* Darwin): magnitude, age dependence, and speed of response. Evolution 57:1281–1290
Matesanz S, Gianoli E, Valladares F (2010) Global change and the evolution of phenotypic plasticity in plants. Annu N Y Acad Sci 1206:35–55
Neufeld CJ (2012) Barnacle appendage plasticity: asymmetrical response time-lags, developmental mechanics and seasonal variation. J Exp Mar Biol Ecol 429:20–27
Nicotra AB, Atkin OK, Bonser SP, Davidson AM, Finnegan EJ, Mathesius U, Poot P, Purugganan MD, Richards CL, Valladares F, van Kleunen M (2010) Plant phenotypic plasticity in a changing climate. Trends Plant Sci 15:684–692
Pfab F (2012) Erweiterungen und Modifikationen eines Modells zur phänotypischen Plastizität. Diploma thesis, Ludwig Maximilian University of Munich
Piersma T, Lindström Å (1997) Rapid reversible changes in organ size as a component of adaptive behaviour. Trends Ecol Evol 12:134–138
Starck JM, Beese K (2001) Structural flexibility of the intestine of Burmese python in response to feeding. J Exp Biol 204:323–335
Tufto J (2000) The evolution of plasticity and nonplastic spatial and temporal adaptations in the presence of imperfect environmental cues. Am Nat 156:121–130
Utz M, Jeschke JM, Loeschcke V, Gabriel W (2014) Phenotypic plasticity with instantaneous but delayed switches. J Theor Biol 340:60–72
Wikelski M, Thom C (2000) Marine iguanas shrink to survive El Niño. Nature 403:37–38