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Opinion

Beyond reaction norms: the temporal dynamics of phenotypic plasticity

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Phenotypic plasticity can allow organisms to cope with environmental changes. Although reaction norms are commonly used to quantify plasticity along gradients of environmental conditions, they often miss the temporal dynamics of phenotypic change, especially the speed at which it occurs. Here, we argue that studying the rate of phenotypic plasticity is a crucial step to quantify and understand its adaptiveness. Iteratively measuring plastic traits allows us to describe the actual dynamics of phenotypic changes and avoid quantifying reaction norms at times that do not truly reflect the organism's capacity for plasticity. Integrating the temporal component in how we describe, quantify, and conceptualise phenotypic plasticity can change our understanding of its diversity, evolution, and consequences.

Highlights

Changes in environmental conditions can lead to variation in the traits expressed by a given genotype within the lifetime of an individual. This phenomenon, referred to as phenotypic plasticity, has been extensively studied in previous decades.

The majority of plasticity studies rely solely on reaction norms, which describe the amplitude of changes in traits across gradients of environmental conditions. Reaction norms, however, miss the temporal dynamics of plasticity, especially the speed at which it occurs.

We highlight why iteratively sampling phenotypic traits over time can help us understand the adaptiveness of plasticity relative to environmental change. We illustrate how this endeavour complements the reaction norm approach and triggers a series of unanswered questions of high interest.

From reaction norms to the temporal dynamics of plasticity

Phenotypic plasticity (see Glossary) can be defined as a genotype's ability to produce different phenotypes across a range of environmental conditions [1]. Plastic organisms are expected to transduce meaningful environmental cues into interpreted internal signals that can ultimately lead to directional changes in their phenotype. In the case of **adaptive plasticity**, their ability to produce environmental-dependent phenotypic traits is thought to buffer some fitness costs resulting from environmental changes [2] and allow organisms to thrive in spatially heterogeneous and temporally fluctuating environments [3,4]. Within populations, these plastic changes in trait distributions have been identified as potential pivots for eco-evolutionary dynamics [2,5–7]. Typically, we distinguish between **reversible plasticity** and **irreversible plasticity**; the former is thought to provide benefits under rapid fluctuations relative to generation time [3], whereas the latter refers to irreversible developmental switches within the timeframe of ontogeny [3,8]. In order to quantify phenotypic plasticity in either of these cases, trait values are commonly measured along gradients of environmental conditions to derive **reaction norms**. When reaction norms take the form of a linear relationship between phenotype and environment, the steepness of the slope represents the **plastic capacity** and the direction of phenotypic changes [9].

Despite being widely used to quantify phenotypic plasticity, reaction norms usually miss a key aspect of plastic responses: the speed at which phenotypic changes occur [10]. When organisms are faced with changes in their local environment, informative cues concerning the environmental change have to be detected and transduced before eventually leading to phenotypic changes. Developmental plasticity in crustaceans from the *Daphnia* genus, which includes the production of cuticular expansions when growing in the presence of a predator [1,11], can be used to illustrate this chain of events. *Daphnia* individuals first detect dissolved kairomones produced by fish predators through chemoreceptors of olfactive neurons [1,11,12], with a possibly evolved detection threshold or cue specificity [13]. Information is then transduced through neurohormonal communication and transcriptional changes, ultimately leading to the production of helmets or spines

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[12,14]. Each of these steps takes an incompressible amount of time, which forcibly translates into a lag between the onset of the environmental change and that of the plastic response.

So far, most studies have concentrated on characterising the plastic capacity and its variation within or between species at a fixed time after the onset of the perturbation, thus assuming that this is representative of the global plastic response [1,4,15]. However, the speed at which plasticity occurs, referred to as the **rate of plasticity**, is rarely considered [10]. This omission could be caused by the usual reasoning behind reaction norms, which assumes that phenotypic changes reach asymptotic values specific to each novel environment. In this setting, sampling the temporal shape of plastic changes may seem unnecessary, provided that the experimenters wait long enough to extract the new phenotypic values expressed in the alternative conditions. It may also seem unnecessary if the question is only to ask if a trait is plastic at all, especially given the technical cost of implementing protocols allowing us to measure the dynamics of plasticity over time.

Here, we argue that neglecting the **temporal dynamics** of plastic responses may lead to a truncated comprehension of phenotypic plasticity by preventing us from assessing if it is adaptive and accurately estimating the plastic capacity. In particular, we discuss how the rate of plasticity directly relates to the adaptiveness of plastic responses and why retracing their temporal dynamics is a crucial step to obtain informative reaction norms. We put forward a series of questions which could indicate key prospects for future investigations in the field of phenotypic plasticity.

The rate of plasticity and the adaptiveness of plastic responses

It is generally expected that both reversible and irreversible phenotypic plasticity are positively selected under conditions of relatively fast and predictable fluctuations [3,16,17]. However, the adaptiveness of plastic traits appears inconsistent across empirical studies, questioning the conditions favouring the evolution of plasticity or its consequences [18–21]. These inconsistencies have been often – but not only – attributed to differences in characteristics of environmental fluctuations between studies [20]. The crucial aspect of the temporal dynamics of plasticity may be involved in these discrepancies as well, but has, until now, been relatively overlooked [10,22–25]. This is especially the case in empirical studies, despite exceptions ([26] and other references listed in [10]) which often concern traits that intrinsically include time (e.g., growth rate, metabolic rate [27–29]) or have historically acknowledged a temporal component (e.g., changes in gene expression, because of the initiation of transcription or the lifetime of RNA [30]). Here, we propose that some of the apparent contradictions surrounding the adaptiveness of phenotypic plasticity might result from the reaction norm approach, in which the temporal dynamics of plastic responses and their interactions with the characteristics of environmental fluctuations are ignored.

Investigating whether phenotypic plasticity is adaptive has been a recurrent focus, yet compelling evidence is often hard to gather, either because costs are neglected [22], or because the benefits are only assessed in a single, constant environmental condition, concealing possible adaptive tradeoffs in other contexts [19,31]. For instance, reaction norms and **tolerance curves** in constant environments have sometimes turned out to be poor predictors of performance in fluctuating environments [28,32–34]. A key element that may improve the predictive power to explain fitness under fluctuating conditions could be to consider non-zero lag times in adaptive phenotypic plasticity [27,28,35]. Although the plastic capacity is key when facing environmental changes, it is probably not sufficient if plasticity is too slow to reach the optimal phenotype in time, and depends on associated costs, as hinted at by models confronting reversible and irreversible plasticity (e.g., [3,22,23]). For a given distance between the average fitness of a plastic population and its closest local adaptive peak (see 'lag-load' in [20]), the kinetics of plasticity

Glossary

Activation threshold: refers here to the minimum amount of environmental change needed for a plastic pathway to be activated. This threshold can contribute to a time lag in the plastic response.
Adaptive plasticity: phenotypic plasticity is considered adaptive when the fitness benefits drawn from this strategy compensate for its maintenance and production costs. Costs set aside, however, all plastic changes do not forcibly bring the organism closer to a new phenotypic optimum; plasticity can be neutral or maladaptive.
Irreversible plasticity: some plastic responses occur during ontogeny (e.g., winged offspring in aphids, cuticular spines in *Daphnia*) and are irreversible within the lifetime of individuals. In this sense, irreversible plasticity refers to a type of developmental plasticity acting through evolutionary switches.
Low-level/high-level phenotypic traits: sometimes referred to as primary and secondary phenotypic traits. High-level phenotypic traits are plastic traits (e.g., behavioural change) resulting from plasticity happening at lower spatial and temporal scales within the individual (e.g., transcription of new genes). Performance proxies are closest to the highest phenotypic level, contrary to molecular responses.
Phenotypic plasticity: the ability of a genotype to produce a variety of phenotypes depending on environmental conditions.
Plastic capacity (or capacity for plasticity): for a trait in a given organism, this describes the absolute amount of phenotypic change that is observed between two environmental conditions.
Rate of plasticity: following an abrupt environmental change, the plasticity rate is the speed at which a plastic trait changes towards its new value.
Reaction norm: a continuous function describing changes in a trait's values along an environmental gradient. Most of the time, a linear function is fitted to a series of phenotypic values sampled under distinct environmental conditions.
Reversible plasticity (also known as phenotypic flexibility): phenotypic changes that can be reversed back and forth if the environment shifts back to the initial state within the lifetime of the individual.

will define how much time the organisms spend in a novel environment with a suboptimal phenotype, while the capacity will constrain the maximal distance travelled in the fitness space. A necessary condition for phenotypic plasticity to be adaptive is for a sufficient portion of the capacity to be mounted for some benefits to be obtained within the timeframe of environmental change [10,23,24]. Therefore, the rate and the capacity are intertwined properties of phenotypic plasticity. The adaptiveness of a plastic response can be expected to result from their combined interaction with the environmental context (Box 1).

Given the possible role of the plasticity rate in the adaptiveness of a plastic response, it is likely to have evolved together with the plastic capacity. For instance, relatively fast fluctuations (i.e., shorter than the generation time) are classically expected to select for reversible phenotypic plasticity [3]. However, this prediction assumes an almost immediate plastic response. If phenotypic plasticity incurs some delay, its adaptiveness and the resulting probability of being positively selected with a given capacity will depend on the match between the plasticity rate and the rate of fluctuations. This match need not be perfect, as even partial phenotypic plasticity [e.g., moderate amounts of heat shock proteins (HSPs) due to too slow a plasticity rate] could still provide sufficient fitness benefits for plastic genotypes to outperform nonplastic strategies. Importantly, whether plastic responses can be fast enough to be adaptive will depend on the constraints or costs specific to the mechanisms that underlie plasticity for the trait of interest. Most of the phenotypic traits we have looked at are underlaid by a series of lower-scale phenomena, which are themselves kinetically limited and potentially costly. In the case of thermal stress, for instance, the speed of producing HSPs after thermal transduction will never exceed that of the preceding transcription of HSP mRNAs, which are of the order of 30 min for the killifish (*Fundulus heteroclitus*) in [36]. Knowing the maximum speed of phenotypic changes and how much it differs between traits and species are therefore important but usually underrated questions.

Nonplastic strategies might be favoured at the expense of plastic ones in cases where the mechanisms underlying plastic responses are too slow compared with the speed of environmental fluctuations, making the balance between the costs and benefits of plasticity unfavourable. Whether fast plastic responses incur higher costs than slower ones, and how these costs balance with the effect of fluctuations on fitness are key unsolved questions (see Outstanding questions). We should also keep in mind that even plasticity rates matching the speed of environmental change may still be of limited adaptiveness. Under rapidly fluctuating conditions, phenomena such as cue–response mismatches may emerge (i.e., increasing environmental noise, resulting in fast and costly plasticity in the wrong direction [3,37,38]) and may lead to maladaptive plasticity, despite its sufficient speed and capacity. Overall, estimating the contextual adaptiveness of phenotypic plasticity better will require us to (i) effectively measure plasticity rates and plastic capacities across traits and organisms, and (ii) compare them with the speed of the environmental fluctuations the organisms are facing [20,39] while (iii) accounting for their potential costs [22,37,38,40], and (iv) for the organism's performance in fluctuating conditions [32,34] (Box 1).

Accurately describing the temporal dynamics of phenotypic plasticity

Measuring the speed at which phenotypic plasticity occurs requires datasets in which the traits are measured iteratively across time after the onset of an environmental change. Below we expose how this objective should allow us to quantify a set of kinetic parameters that all contribute to gradual phenotypic plasticity. We explain how this should, in turn, help derive more compelling reaction norms by adjusting the timing of sampling and revise some expectations about the shape of plastic responses throughout their time course. As stated earlier, accurately describing the temporal dynamics of plasticity is a pivotal step towards understanding its adaptiveness.

Temporal dynamics: the temporal dynamics of plasticity is the combination of the activation threshold, lag time, and rate which eventually lead to the plastic capacity.

Tolerance curve: a particular case of a reaction norm for a trait considered as a proxy of performance (typically the growth rate for microorganisms, the maximum speed in a sprint test for lizards, etc.). If the environmental gradient is made up of temperature values, we may use the 'thermal performance curve' label. Most tolerance curves are derived from a trait's values measured across a gradient of constant conditions.

Box 1. Exploring the adaptiveness of plasticity by linking the rate of plasticity to changes in performance under fluctuating conditions

Phenotypic plasticity is expected to underlie some degree of ecological generalism by broadening the range of environmental conditions under which the organism's performance is maintained [51–54]. Under environmental fluctuations, this adaptiveness can only emerge if the plastic capacity is mounted fast enough relative to the environmental change [17,23,24,48,55] and if the resulting benefits compensate for the costs of plasticity [22]. Here, we explain why focussing on the temporal dynamics of plasticity is crucial to understand tolerance curves and their underlying adaptive plasticity in fluctuating environments [32]. We use a simple framework, where the plasticity of a phenotypic trait contributes to the breadth of environmental tolerance (Figure I). Let τ be the rate of adaptive plasticity associated with implementation of the plastic capacity (e.g., $\tau_{forward}$, see Figure 1 in the main text), represented in the frequency space in Figure I (red dot, broken line). In (1), we consider a classic tolerance curve, describing the changes in performance across a gradient of constant conditions ($f_1 = 0$). This tolerance curve is described by the breadth [(1), grey area below the curve], according to which generalism is usually defined. In (2), the mean values of the environmental conditions are the same as before but the environment fluctuates at a slow frequency (f_2) around these means. The fluctuating environment may lead to a slight decrease in performance at the optimum, but since $f_2 < \tau$, the speed of environmental change is low enough for plasticity to be fully implemented. Therefore, the plastic generalist is able to buffer environmental changes and maintain the breadth of its environmental tolerance by matching its phenotype to the conditions in time ($f < \tau$). As fluctuations become faster than the rate of adaptive plasticity (e.g., $f_3 > \tau$), traits change too slowly for the plastic capacity to be wholly implemented. The expected benefits of plasticity are mitigated, and so are the associated breadth of tolerance and performance (3). We may expect the decrease in environmental generalism to vary in amplitude, as plasticity of the trait of interest may underlie a large degree (3a, green path) or a more limited degree of the observed generalism under constant conditions (3b, orange path). We can especially expect 3b to emerge if several traits, each with a different rate, contribute to generalism along the environmental axis of interest. This reasoning (Figure I) could be used to design protocols comparing threshold frequency values (i.e., breaking points in the breadth of tolerance) to the rates of plasticity acquired by other experiments [32]. We would expect adaptive plasticity to result in a correlation between the rate of plasticity and the tipping points in achieved generalism across the fluctuation gradient.

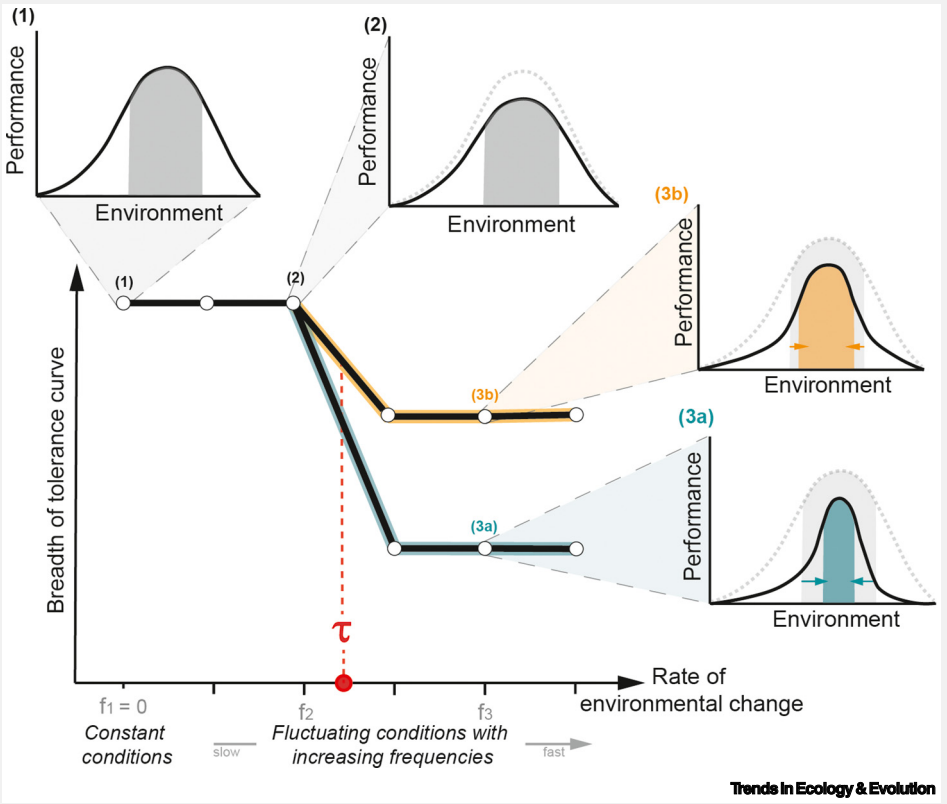

Figure I. The interaction between the rate of adaptive plasticity and the rate of environmental change can result in changes in the degree of generalism achieved.

Going back to the underlying mechanisms of plastic responses, the temporal dynamics of phenotypic plasticity and its resulting rate may be decomposed into three key parameters (Figure 1). First, a minimum amount of environmental change, that is, an **activation threshold**, as described in cases of developmental plasticity [8], may play an important role in the kinetics of reversible plasticity. Thresholds would result in slower plastic responses in face of gradual environmental changes, increasing the measured lag time. Activation thresholds are likely to result from the balance of the fitness costs of fluctuations an organism is exposed to, the costs of activating a plastic pathway (e.g., polymerising more chitin and precipitating $CaCO_3$ to form cuticular expansions in *Daphnia*), and the mechanistic constraints inherent to detecting the environmental change (e.g., the affinity constant of kairomone receptors in *Daphnia*).

Plastic changes can be characterised by a **lag time** before the phenotypic changes take place (t_{lag} in Figure 1). This lag may result from lower-scale mechanisms that underlie the changes in the trait of interest, such as transcriptional activity leading to behavioural changes [41]. We can expect to see strong variance in the duration of this lag depending on the mechanisms underlying the plastic response for the chosen trait, which may actually be information of interest [34]. In particular, **low-level phenotypic traits** such as gene transcription levels may only depend on a few transduction steps and transcription factors, and this activity can quickly be modified covalently. In these cases, the lag time (t_{lag}) is likely to be relatively low (e.g., the order of hours for transcription). However, these low-level plastic mechanisms may themselves contribute to the plasticity of **high-level phenotypic traits**, spanning higher biological scales in both time and space (i.e., within the organism). In this regard, the accumulation of low-level time delays could lead to increased high-level lag times before the onset of plasticity. The interweaving of mechanisms at different scales can result in some high-level phenotypic changes that are almost instantaneous (e.g., no delay for a fight-or-flight behavioural response, which is directly based on endocrine secretion of adrenaline [42]), whereas others are much slower (e.g., spine production in response to predators in *Daphnia,* which requires cuticular polymerisation [1,11,12]) or even involve mechanisms that take place across multiple generations (i.e., transgenerational plasticity, from one generation in [43–45] to tens of generations in [46]).

Finally, the time course of plastic changes can be described by a characteristic time, the reciprocal of the **plasticity rate** *per se* ($\tau_{forward/backward}$ in Figure 1), which relates to a delay in acclimation [23,27,28,47,48]. The acclimation time for temperature tolerance may range from less than a day (20 h for amphibians) to almost a week (6 days for crustaceans) among ectothermic animals [49]. Both the lag and rate of plasticity are probably simultaneously at play in most cases of phenotypic plasticity and contribute to an overall delay, leading to more or less gradual plastic responses. Deciphering the relative importance of the lag and the rate in the dynamics of plastic responses, and whether their importance differs between traits and organisms are important questions to answer.

Overall, the kinetics of plasticity probably result from a combination of different phenotypic traits, which play distinct roles in the temporal dynamics of plasticity. The activation threshold and the lag may stem from limiting the cellular transduction steps, whereas the rate of plasticity should be related to the mechanisms of ongoing phenotypic change, including other low-level phenotypic traits that contribute to the plasticity of the high-level phenotypic trait of interest. Being able to quantify these parameters will require experimental designs with iterative and sufficiently frequent phenotypic sampling (Box 2). Beyond the importance of adequately quantifying plastic changes, knowing whether the kinetics underlying the capacity are fast or slow is a crucial step

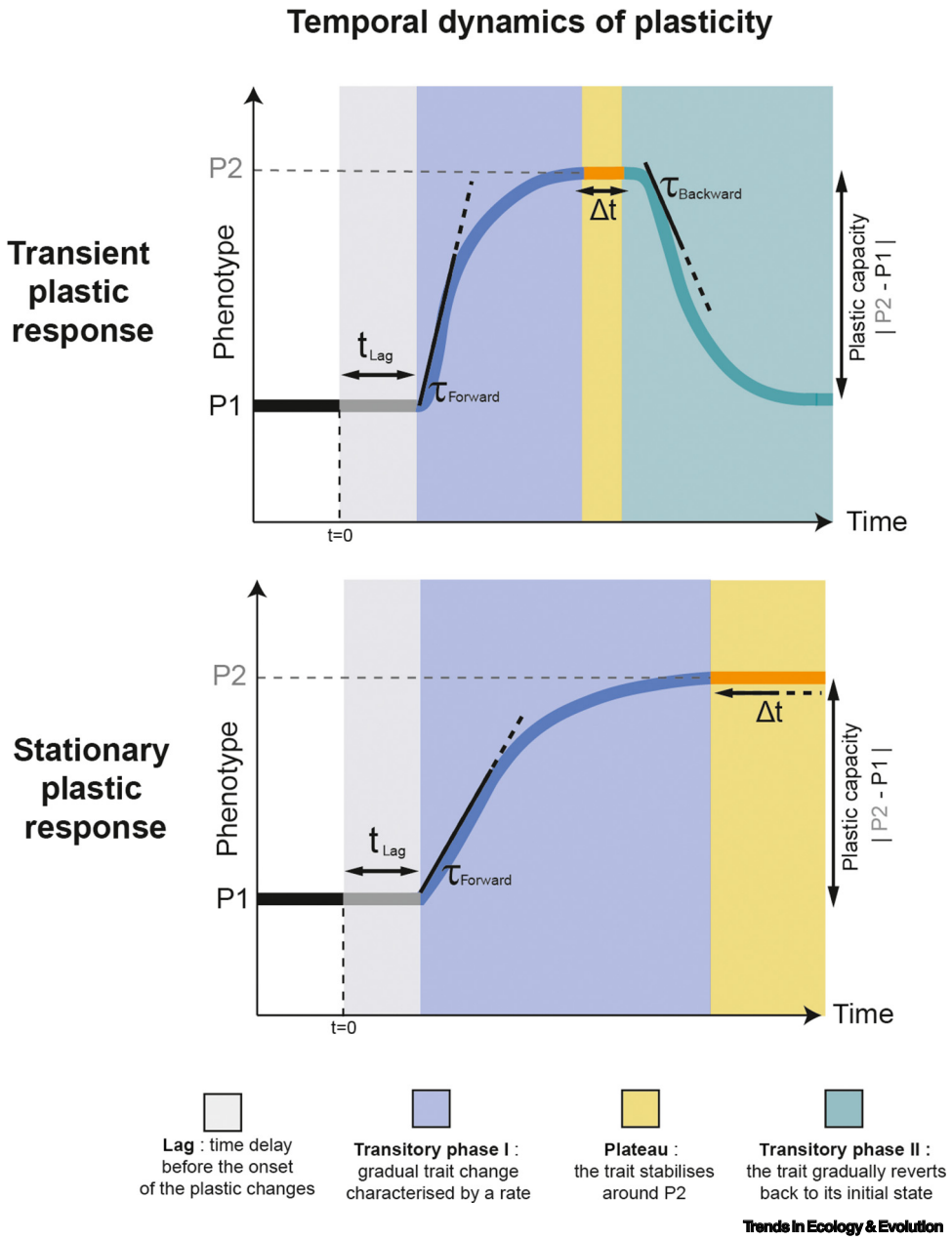

Figure 1. Beyond reaction norms, the temporal dynamics of phenotypic plasticity can be characterised by a series of kinetic parameters. Following an acute environmental change happening at t = 0, the temporal dynamics of plastic traits may follow a transient response [upper panel; e.g., the heat shock protein (HSP) response [36]] or a stationary plastic response (lower panel; e.g., developmental plasticity in *Daphnia*, cell velocity in Box 3 in the main text). A lag time (t_{lag}) may precede any change in phenotypic traits and could result from both activation thresholds and lower-level plastic mechanisms [e.g., transduction steps, early small-scale responses such as protein (de)phosphorylation, etc.]. In both the transient and the stationary scenarios, the forward rate ($\tau_{forward}$) describes the speed at which the phenotype shifts from the initial state (P1) towards a new value (P2) through a transitory phase. For a stationary plastic response, the phenotype is stable once the plateau (yellow area) is reached. For the transient plastic response, a plateau can be derived, but this is transitory (duration = Δt): the trait eventually reverses back to its initial state following a backward rate of plasticity ($\tau_{backward}$). In the stationary case, this reversibility rate can be experimentally accessed by shifting the environment back to the initial conditions. Note that in the transient case, the trait could also reverse to a value differing from the initial

(Figure legend continued at the bottom of the next page.)

Box 2. Experiments to measure the temporal dynamics of plastic responses

Designing experiments aiming to decipher the temporal dynamics of various plastic responses is key to investigating the adaptiveness of plasticity (see Box 1 in the main text), its consequences, and evolution. Here, we highlight crucial points to consider when designing protocols targeting the kinetics of plasticity.

- Technical and logistic limitations. Iteratively sampling the phenotype to trace the temporal dynamics of plasticity trades off with other dimensions. The number of experimental replicates, the number of environmental conditions, and the number of studied traits will all compromise the experimental sampling frequency.
- Constraints caused by the speed of sampling. Information theory [56] states that only plasticity rates that are at least two times slower that the sampling frequency will be correctly estimated. This limitation may not be a major issue for high-level phenotypes (e.g., morphological traits), for which the temporal dynamics are usually long enough to allow for multiple measurements throughout the time course of plasticity. However, it might be more problematic for low-level traits (e.g., reflex behavioural responses, molecular processes). For instance, Bukhari and colleagues [41] examined the plasticity of gene expression underlying a fixed-action pattern of behaviour in the three-spined stickleback (*Gasterosteus aculeatus*). Despite pinpointing the strong temporal structure of transcriptional responses, their sampling rate was too low to derive plasticity rates.
- Choosing the sampling speed. Although sufficient knowledge of the life history traits of the studied species will be key to designing such experiments, the specifics of the investigated traits and environments should be considered to choose the properly sampling frequency. Although one would not define the predictability of fluctuation to be at the same temporal scale for a mouse compared with an elephant, both exhibit rapid metabolic plasticity in response to brutal heat or drought stress through highly conserved mammalian pathways [57].
- Constraints caused by methodology. Our ability to measure plasticity rates is likely to be trait-dependent. Destructive or invasive measurements (e.g., RNAseq of a brain region or retro-orbital blood collection, respectively) are technically more difficult to implement at a high temporal resolution than noninvasive phenotypic sampling (e.g., video recordings of behaviour). In this sense, targeting the kinetics of plasticity is likely to favour the development of less invasive techniques that can be repeated across time more easily (e.g., using images to estimate melanin levels instead of clipping skin samples, or using portable thermal loggers instead of cloacal probe measurements, etc.). These trends may themselves lead to a bias in how we measure phenotypic traits and, more importantly, which phenotypic traits we choose to study.

Overall, having these points in mind should help us to design efficient protocols to quantify the temporal dynamics of plasticity. The output parameters may be further used in experiments aimed at understanding the adaptiveness of plastic changes (see Box 1 in the main text).

towards better understanding of plasticity and its consequences. Determining if the combination of the kinetics and capacity of plasticity is adaptive will require links with the organism's fitness in fluctuating environments (Box 1, [32]).

Experiments with increased temporal resolution will also allow us to depart from the widespread but simplified scheme of linear or logistic phenotypic changes up to an asymptotic state (Figure 1B). Although this may be valid for many plastic traits (e.g., helmet length in the *Daphnia* genus, [1,11,12]), we can rarely assert that the phenotypic value measured at a chosen time to describe the capacity for plasticity was indeed that of the plateau (Figure 1). In cases of reversible plasticity, the duration of the plateau (Δt) could be variable, and reverse dynamics may occur when the new environmental conditions remain unchanged (Figure 1A). This is, for instance, the case of the expression of HSPs [30,36,50], where the phenotypic response peaks temporarily (Figure 1A).

This refined knowledge of the temporal dynamics of plasticity should also help us answer the question of 'when' to measure the plastic capacity. As exposed earlier, without a precise understanding of if and when traits reach their asymptotic value, we are exposed to the risk of deriving

state. In both scenarios, it is critical to consider acute sampling times when aiming to estimate the plastic capacity in order to capture the plateau (see Box 3 in the main text). Interestingly, a transient dynamic may wrongly be interpreted as a case of a stationary response if the duration of the experiment is smaller than Δt.

unsteady reaction norms. This may not be an issue if rankings between conditions (e.g., genotypes, organisms, treatments) stay the same throughout the experiment (as in Box 3), but such temporal correlations can rarely be asserted *a priori.* For instance, characterising the dynamics of phenotypic plasticity in a ciliate revealed that the shape of reaction norms can change through time across environmental conditions, even for a trait that follows an asymptotic trajectory (Box 3). If the aim of a study is to determine the capacity of plasticity, then it requires a combination of reaction norms and iterative phenotypic measurements across time to assess when the traits stabilize following a change in the environment (Figure 1 and Box 3). In addition to expanding our description of phenotypic plasticity beyond reaction norms to their temporal dynamics, which we believe is key to understanding its adaptiveness (Box 1), approaching plasticity with the proposed mindset can produce higher confidence in the quantification of plastic capacity.

Concluding remarks

Despite extended knowledge on the amount of phenotypic plasticity organisms can express across a gradient of constant conditions, a fundamental aspect of plastic responses has been largely understudied: the speed at which organisms can change their phenotype. In this opinion piece, we indicate the necessity of going beyond reaction norms alone, as their explanatory and predictive power under fluctuating conditions may be limited. Although putting the temporal dynamics of plasticity in the spotlight may not always be an easy task from a methodological viewpoint (Box 2), it should enable us to refine our expectations regarding the shape of plastic responses across time (Box 3), which is expected to have major implications for our understanding of the adaptiveness (Box 1), evolution, and consequences of phenotypic plasticity, including in eco-evolutionary dynamics. Previous work has emphasised the need to test the adaptiveness of plasticity in fluctuating environments [32] or has shown that the properties of environmental change are a determining factor in plasticity's role in adaptive evolution [20], especially given its limits and costs [22]. Measuring the temporal dynamics of plasticity may help to fill a longstanding yet discrete gap by shedding light on key parameters which interact and evolve with the properties of environmental change.

Box 3. Measuring the temporal dynamics of phenotypic plasticity can help us ascertain the right time to derive reaction norms

Measuring the temporal dynamics of phenotypic plasticity is not only of interest for deriving kinetic parameters; it also helps us to estimate the variance in reaction norms through time and, therefore, to choose the right time to measure the phenotypic response. As an illustration, the temporal dynamics of a phenotypic trait (cell velocity) in an isogenic population of the ciliate *Tetrahymena thermophila* exposed to a thermal gradient (T = 19, 23, 27, 31, and 35°C) are presented here (Figure I; main graph). For each temperature, an exponential decay model was used to fit the phenotypic changes as a function of time. Although the trait of interest followed a classic logistic dynamic, the rate of plasticity was revealed to be temperature-dependent, leading to reaction norms which shape changes through time (Graphs a–d). In this example, the reaction norms are presented at 0, 15, 30 and 120 minutes. Initially (Graph a, t = 0), no plasticity was observed, as expected from the typical conditions at 23°C. After 15 minutes (Graph b), a linear reaction norm was observed as a result of the faster rates at T = 19°C and T = 35°C. Thirty minutes after exposition (Graph c), the reaction norm had a logarithmic shape. It then stabilised from 2 hours onwards into a sigmoid shape (Graph d, t = 120 min; the transparent datapoints are the mean values of velocity between t = 2 h and t = 4 h). Such insights reveal the importance of knowing the dynamics of plasticity to extract a reaction norm that is representative of the stable state. In this example, the ranking of plastic capacities between conditions did not change, but such trends can rarely be asserted before tracing the temporal dynamics. This may be even more essential when plasticity is transient and when the 'correct' measurement window has a lower (i.e., plasticity has not happened yet) and an upper (i.e., plasticity is not expressed any longer) time margin (e.g., the transient plastic response in Figure 1 in the main text), which may not be known *a priori*. This reasoning applies both to intra- or transgenerational reversible plasticity and to irreversible plasticity. Sampling the trait iteratively, at a sensible frequency, given the trait's identity and timescale (see Box 2 in the main text), is a robust way to ascertain when to extract the capacity and to further question how the temporal dynamics of plasticity interact with environmental fluctuations (see Box 1 in the main text). Notice that here, we can assert that the response is stationary within a 4-h period, but we cannot rule out the possibility that cell-speed plasticity is a transient response over a longer timescale.

Outstanding questions

The rate of plasticity combines the ability to detect environmental changes and the speed of the mechanisms underlying phenotypic changes. Both could incur costs, constraining the optimal rate expressed in a given environmental condition. Do the temporal dynamics of plasticity depend on the environmental conditions, either within an environmental gradient or between different environmental axes?

The rate of plasticity will determine the duration of the phenotype–environment mismatch and the subsequent fitness costs. Fluctuations with a wide amplitude may therefore require both strong and fast plastic responses. Should we expect the rate of plasticity and the plastic capacity to be positively correlated, or do interactions with costs affect this relationship?

Determining if plasticity is reversible is often a key question, which is experimentally tested by placing the organisms back into their original environment. Is the speed at which these changes occur the same forward and backward (the relationship between $\tau_{forward}$ and $\tau_{backward}$, see Figure 1 in the main text) and how can this inform us about the mechanisms underlying plasticity?

How variable are the temporal dynamics of plasticity intra- and interspecifically for a given trait? Measuring how variable rates are between organisms may be a good lead to explore the interactions of life history traits, phenotypic plasticity, and environmental fluctuations.

Limiting factors surround the ideal of high plastic capacity occurring quickly, such as the maintenance and production costs for both the rate and the capacity, and environmental noise leading to cue–response mismatches. How do these constraints interact and how can they shape the evolution of plasticity under environmental fluctuations with varying characteristics?

Phenotypic plasticity is sometimes considered to play a key role in the dynamics of colonisation or adaptation to new environmental conditions. Can the rate of plasticity provide explanations for the unfolding of key eco-evolutionary

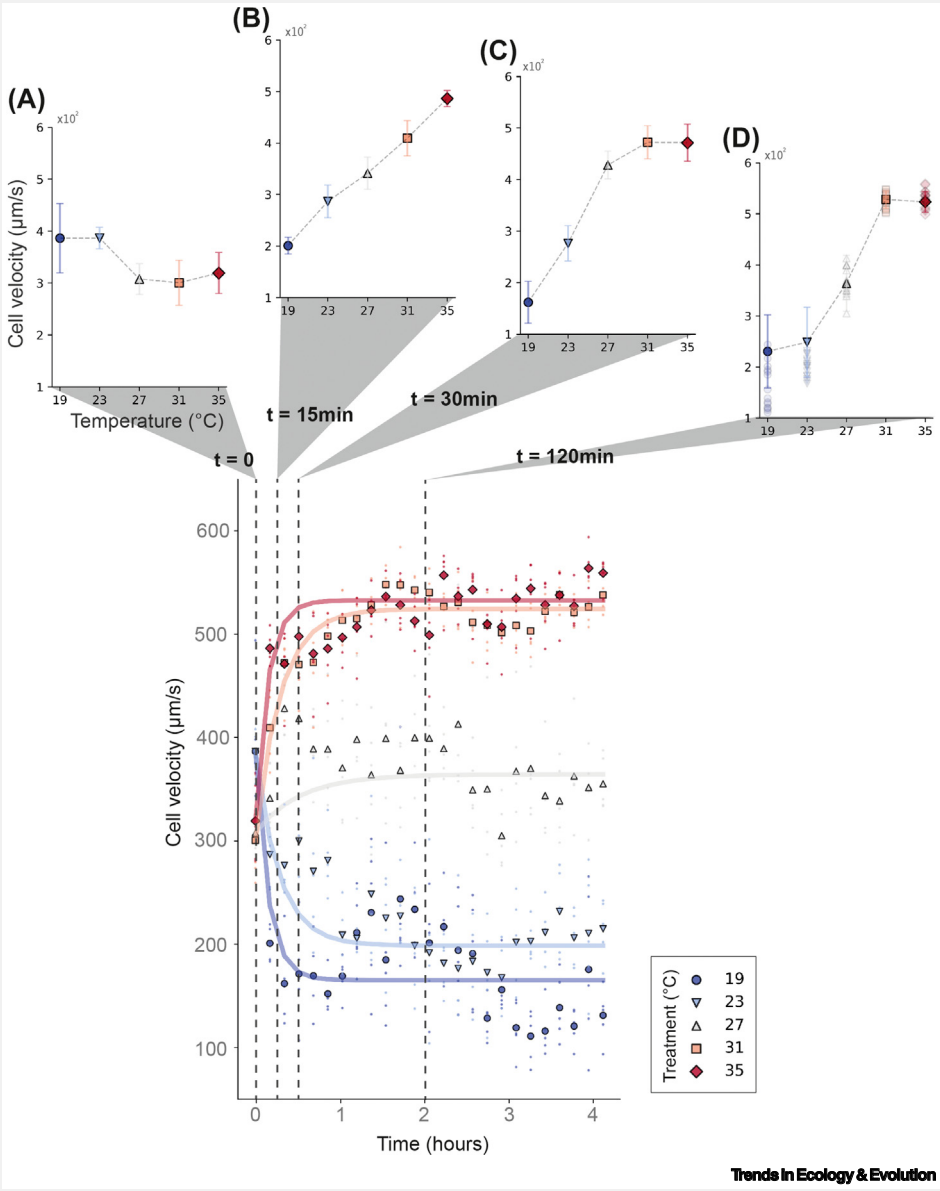

Figure I. The context-dependency of plasticity's temporal dynamics can result in unstable reaction norms.

processes relative to environmental change? The rate at which organisms react to spatial heterogeneity may play an important role in range expansion, depending especially on the interaction between the rates of plasticity and dispersal.

The capacity of phenotypic plasticity is known to influence ecological and evolutionary dynamics. How can the rate of plasticity itself or its interaction with plastic capacity affect eco-evolutionary processes such as metapopulation dynamics, local adaptation, or even speciation? For instance, the adaptiveness of phenotypic plasticity should depend on the match between the rate of plasticity and the speed of the environmental fluctuations, leading the buffering effect of plasticity on selection (e.g., the Bogert effect) to vary accordingly.

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Declaration of interests

There are no interests to declare.

References

1. Scheiner, S.M. (1993) Genetics and evolution of phenotypic plasticity. *Annu. Rev. Ecol. Syst.* 24, 35–68
2. Chevin, L.-M. *et al.* (2010) Adaptation, plasticity, and extinction in a changing environment: towards a predictive theory. *PLoS Biol.* 8, e1000357
3. Botero, C.A. *et al.* (2015) Evolutionary tipping points in the capacity to adapt to environmental change. *Proc. Natl. Acad. Sci. U. S. A.* 112, 184–189
4. West-Eberhard, M.J. (2003) *Developmental Plasticity and Evolution* (1st edn), Oxford University Press
5. Kremer, C.T. and Klausmeier, C.A. (2013) Coexistence in a variable environment: eco-evolutionary perspectives. *J. Theor. Biol.* 339, 14–25
6. Miner, B.G. *et al.* (2005) Ecological consequences of phenotypic plasticity. *Trends Ecol. Evol.* 20, 685–692
7. Smallegange, I.M. (2022) Integrating developmental plasticity into eco-evolutionary population dynamics. *Trends Ecol. Evol.* 37, 129–137
8. Snell-Rood, E.C. *et al.* (2018) Mechanisms of plastic rescue in novel environments. *Annu. Rev. Ecol. Evol. Syst.* 49, 331–354
9. Callahan, H.S. *et al.* (2008) Phenotypic plasticity, costs of phenotypes, and costs of plasticity. *Ann. N. Y. Acad. Sci.* 1133, 44–66
10. Burton, T. *et al.* (2022) Environmental change and the rate of phenotypic plasticity. *Glob. Change. Biol.* 28, 5337–5345
11. Lüning, J. (1992) Phenotypic plasticity of *Daphnia pulex* in the presence of invertebrate predators: morphological and life history responses. *Oecologia* 92, 383–390
12. Weiss, L.C. (2019) Sensory ecology of predator-induced phenotypic plasticity. *Front. Behav. Neurosci.* 12, 330
13. Weiss, L.C. *et al.* (2018) Identification of *Chaoborus* kairomone chemicals that induce defences in *Daphnia*. *Nat. Chem. Biol.* 14, 1133–1139
14. Rozenberg, A. *et al.* (2015) Transcriptional profiling of predator-induced phenotypic plasticity in Daphnia pulex. *Front. Zool.* 12, 18
15. Schlichting, C. and Pigliucci, M. (1998) *Phenotypic Evolution: a Reaction Norm Perspective*, Sinauer
16. Leung, C. *et al.* (2020) Reduced phenotypic plasticity evolves in less predictable environments. *Ecol. Lett.* 23, 1664–1672
17. Gabriel, W. (2005) How stress selects for reversible phenotypic plasticity. *J. Evol. Biol.* 18, 873–883
18. Fox, R.J. *et al.* (2019) Beyond buying time: the role of plasticity in phenotypic adaptation to rapid environmental change. *Philos. Trans. R. Soc. B* 374, 20180174
19. Hendry, A.P. (2016) Key questions on the role of phenotypic plasticity in eco-evolutionary dynamics. *J. Hered.* 107, 25–41
20. Vinton, A.C. *et al.* (2022) Plasticity's role in adaptive evolution depends on environmental change components. *Trends Ecol. Evol.* 37, 1067–1078
21. Westneat, D.F. *et al.* (2019) Causes and consequences of phenotypic plasticity in complex environments. *Trends Ecol. Evol.* 34, 555–568
22. DeWitt, T.J. *et al.* (1998) Costs and limits of phenotypic plasticity. *Trends Ecol. Evol.* 13, 77–81
23. Siljestam, M. and Östman, Ö. (2017) The combined effects of temporal autocorrelation and the costs of plasticity on the evolution of plasticity. *J. Evol. Biol.* 30, 1361–1371
24. Padilla, D.K. and Adolph, S.C. (1996) Plastic inducible morphologies are not always adaptive: the importance of time delays in a stochastic environment. *Evol. Ecol.* 10, 105–117
25. Gabriel, W. *et al.* (2005) Environmental tolerance, heterogeneity, and the evolution of reversible plastic responses. *Am. Nat.* 166, 339–353
26. Turriago, J.L. *et al.* (2023) The time course of acclimation of critical thermal maxima is modulated by the magnitude of temperature change and thermal daily fluctuations. *J. Therm. Biol.* 114, 103545
27. Fey, S.B. *et al.* (2021) Resolving the consequences of gradual phenotypic plasticity for populations in variable environments. *Ecol. Monogr.* 91, e01478
28. Kremer, C.T. *et al.* (2018) Gradual plasticity alters population dynamics in variable environments: thermal acclimation in the green alga *Chlamydomonas reinhartdii*. *Proc. R. Soc. B* 285, 20171942
29. Sandblom, E. *et al.* (2014) Temperature acclimation rate of aerobic scope and feeding metabolism in fishes: implications in a thermally extreme future. *Proc. R. Soc. B* 281, 20141490
30. Quan, Y. *et al.* (2022) Transcription dynamics of heat shock proteins in response to thermal acclimation in *Ostrinia furnacalis*. *Front. Physiol.* 13, 992293
31. DeWitt, T.J. *et al.* (2000) Functional diversity among predators of a freshwater snail imposes an adaptive trade-off for shell morphology. *Evol. Ecol. Res.* 2, 129–148
32. Ketola, T. and Kristensen, T.N. (2017) Experimental approaches for testing if tolerance curves are useful for predicting fitness in fluctuating environments. *Front. Ecol. Evol.* 5, 129
33. Ketola, T. and Saarinen, K. (2015) Experimental evolution in fluctuating environments: tolerance measurements at constant temperatures incorrectly predict the ability to tolerate fluctuating temperatures. *J. Evol. Biol.* 28, 800–806
34. Kronholm, I. and Ketola, T. (2018) Effects of acclimation time and epigenetic mechanisms on growth of *Neurospora* in fluctuating environments. *Heredity* 121, 327–341
35. Stomp, M. *et al.* (2008) The timescale of phenotypic plasticity and its impact on competition in fluctuating environments. *Am. Nat.* 172, E169–E185
36. Schulte, P.M. *et al.* (2011) Thermal performance curves, phenotypic plasticity, and the time scales of temperature exposure. *Integr. Comp. Biol.* 51, 691–702
37. Tonsor, S.J. *et al.* (2013) Developmental instability is genetically correlated with phenotypic plasticity, constraining heritability and fitness. *Evolution* 67, 2923–2935
38. Ketola, T. and Kronholm, I. (2023) Experimental evolution of evolutionary potential in fluctuating environments. *J. Evol. Biol.* 36, 945–949
39. Ruokolainen, L. *et al.* (2009) Ecological and evolutionary dynamics under coloured environmental variation. *Trends Ecol. Evol.* 24, 555–563
40. Murren, C.J. *et al.* (2015) Constraints on the evolution of phenotypic plasticity: limits and costs of phenotype and plasticity. *Heredity* 115, 293–301
41. Bukhari, S.A. *et al.* (2017) Temporal dynamics of neurogenomic plasticity in response to social interactions in male threespined sticklebacks. *PLoS Genet.* 13, e1006840
42. McCarty, R. (2016) The fight-or-flight response. In *Stress: Concepts, Cognition, Emotion, and Behavior*, pp. 33–37, Elsevier
43. Bestion, E. *et al.* (2014) Maternal exposure to predator scents: offspring phenotypic adjustment and dispersal. *Proc. R. Soc. B Biol. Sci.* 281, 20140701
44. Dantzer, B. *et al.* (2013) Density triggers maternal hormones that increase adaptive offspring growth in a wild mammal. *Science* 340, 1215–1217
45. Galloway, L.F. and Etterson, J.R. (2007) Transgenerational plasticity is adaptive in the wild. *Science* 318, 1134–1136
46. Cayuela, H. *et al.* (2022) Transgenerational plasticity of dispersal-related traits in a ciliate: genotype-dependency and fitness consequences. *Oikos* 2022, e08846
47. Havird, J.C. *et al.* (2020) Distinguishing between active plasticity due to thermal acclimation and passive plasticity due to Q_{10} effects: why methodology matters. *Funct. Ecol.* 34, 1015–1028
48. Burton, T. *et al.* (2020) Measuring phenotypes in fluctuating environments. *Funct. Ecol.* 34, 606–615
49. Einum, S. and Burton, T. (2022) Divergence in rates of phenotypic plasticity among ectotherms. *Ecol. Lett.* 26, 147–156
50. Foster, N.L. *et al.* (2015) Time-related expression profiles for heat shock protein gene transcripts (HSP40, HSP70) in the central nervous system of *Lymnaea stagnalis* exposed to thermal stress. *Commun. Integr. Biol.* 8, e1040954
51. Lande, R. (2014) Evolution of phenotypic plasticity and environmental tolerance of a labile quantitative character in a fluctuating environment. *J. Evol. Biol.* 27, 866–875
52. Meilhac, J. *et al.* (2019) Both selection and plasticity drive niche differentiation in experimental grasslands. *Nat. Plants* 6, 28–33
53. Svanbäck, R. and Schluter, D. (2012) Niche specialization influences adaptive phenotypic plasticity in the threespine stickleback. *Am. Nat.* 180, 50–59

54. Turner, K.G. *et al.* (2015) Adaptive plasticity and niche expansion in an invasive thistle. *Ecol. Evol.* 5, 3183–3197
55. Clark, C.W. and Harvell, C.D. (1992) Inducible defenses and the allocation of resources: a minimal model. *Am. Nat.* 139, 521–539
56. Shannon, C.E. (1998) Communication in the presence of noise. *Proc. IEEE* 86, 447–457
57. Richter, K. *et al.* (2010) The heat shock response: life on the verge of death. *Mol. Cell* 40, 253–266