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Ecological and evolutionary consequences of metabolic rate plasticity in response to environmental change

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Summary

Basal or standard metabolic rate reflects the minimum amount of energy required to maintain body processes, while the maximum metabolic rate sets the ceiling for aerobic work. There is typically up to three-fold intraspecific variation in both minimal and maximal rates of metabolism, even after controlling for size, sex and age; these differences are consistent over time within a given context, but both minimal and maximal metabolic rates are plastic and can vary in response to changing environments. Here we explore the causes of intraspecific and phenotypic variation at the organ, tissue and mitochondrial level. We highlight the growing evidence that individuals differ predictably in the flexibility of their metabolic rates and in the extent to which they can suppress minimal metabolism when food is limiting but increase capacity for aerobic metabolism when a high work rate is beneficial. It is unclear why this intraspecific variation in metabolic flexibility persists – possibly because of trade-offs with the flexibility of other traits – but it has consequences for the ability of populations to respond to a changing world. It is clear that metabolic rates are targets of selection, but more research is needed on the fitness consequences of rates of metabolism and their plasticity at different life stages, especially in natural conditions.

1. Introduction

A fundamental animal function is the metabolic conversion of food into a form of energy – ATP – that is usable by the body's cells; the rate of metabolism thus forms a nexus between environmental resources and animal fitness [1]. An animal's metabolic rate is usually recorded in terms of whole-animal oxygen consumption. This is really a proxy for the underlying process of cellular respiration, in which ATP is generated from nutrient molecules. While it is possible to produce ATP in the absence of oxygen (through glycolysis), this yields less ATP per molecule of energetic substrate than the alternative of oxidative phosphorylation, and so most multicellular organisms produce the majority of their ATP through a process that consumes oxygen. As a consequence, measurements of oxygen uptake by the body (which are relatively easy to make) can give a relative measure of the animal's overall rate of cellular respiration (which is more difficult to quantify).

The minimum level of sustainable metabolism occurs when an animal is non-reproductive, unstressed, inactive and not digesting food; this is termed the standard metabolic rate (SMR) in ectotherms and the basal metabolic rate (BMR) in endotherms if they are within their thermoneutral zone. The highest rate of aerobic metabolism that can be achieved is termed the maximum metabolic rate (MMR), although endotherms have a second form of maximal metabolism, summit metabolism (M-sum), which is the maximum aerobic rate induced by exposure to cold. Relatively little time may be spent operating at these minimal or maximal extremes, but they nonetheless have biological significance: BMR or SMR defines the minimal 'cost of living' that cannot be avoided, while maximum rates are measures of the individual's capacity for work (MMR) or heat generation (M-sum). The difference between the minimal and maximal rates (termed the aerobic scope) defines the maximum amount of oxygen available for activities such as muscular work or digestion.

All species appear to show significant among-individual variation in both minimal and maximal rates of metabolism even under standardised conditions [2-5]. The scale of this variation is at first sight puzzling since metabolic rates have fitness consequences [6], but is likely due to the optimal metabolic rate being context-dependent [2, 7]. It should be noted that while the different

forms of metabolic rates are often found to be correlated, especially when comparing among species [8, 9], minimal and maximal metabolic rates are best treated as independent traits since they are under different selection pressures that may vary in parallel but can be uncoupled [10-12].

Metabolic rates can appear to be (and are often treated as) consistent and repeatable traits of an individual. However, the repeatability of metabolic rates declines over time [13-15] and is weaker in more variable environments [16]. Moreover metabolic rates are known to show plasticity in response to environmental conditions [17]. This is a topical point given the increasing rate of environmental change especially in terms of temperature; temperature-induced changes in metabolic rates (principally of ectotherms) have been suggested to be a key likely cause of population failure and local extinction in a warming world [18, 19]. Greater plasticity is likely to increase resilience – a concept that has been tested among species [20] but is lacking empirical evidence among individuals of a species. Linked to plasticity is the phenomenon of acclimation – physiological traits such as metabolic rate may show an acute change in response to an environmental perturbation (e.g. in temperature) but this change can reduce with exposure time [21, 22].

In this review, we evaluate the evidence that metabolic rates are plastic traits and discuss the environmental features which drive changes in metabolism. We highlight increasing evidence that both populations and individuals within populations differ in the plasticity of their metabolic rates, and consider the physiological and cellular drivers of both intraspecific variation in metabolism and its plasticity. Through consideration of the costs and benefits of having flexible rates of metabolism we then evaluate how this will influence the capacity of species to cope with and adapt to environmental change, highlighting the gaps in knowledge that prevent a full understanding of this important subject.

2. Evidence for plasticity in metabolic rates

Metabolic rate, along with most physiological traits, exhibits phenotypic plasticity in response to changes in either the animal's internal state or its environment [17, 23, 24]. Alterations to metabolism

can be programmed so as to allow the animal to cope with predictable changes in its energetic state or demands, as when BMR is reduced when animals hibernate or aestivate [25], when M-sum is increased prior to migration in birds [17, 26], or when SMR is reduced in intertidal animals that shut down when the tide goes out [27]. Different aspects of metabolism can exhibit separate temporal rhythms, indicating independent controlling mechanisms: while both BMR and M-sum may be elevated over winter in small birds, the increase in M-sum has been found to precede that in BMR and it may last longer into the spring [28]. On top of these programmed changes, metabolism can vary in response to a stochastic change in the environment. As an example, metabolic rates of ectotherms increase after an acute rise in temperature but then usually drop again as the animal becomes acclimated to the new temperature in order to reduce maintenance costs [22]. Not all aspects of metabolism respond to the same extent: it has been proposed that the metabolic floor of ectothermic organisms (i.e. their SMR) is more plastic in response to increasing temperature than is their ceiling (MMR) [29], although this is not always the case [30]. Similarly, the BMR of endotherms generally increases after cold-acclimation and decreases after warm-acclimation [17, 31], and has been found to be much more flexible in response to temperature than either MMR [32] or M-sum [33]. Metabolic rate also exhibits plasticity in response to changes in food availability, with food restrictions leading to a reduction in SMR or BMR [34-38], but not necessarily in MMR [37].

Although a great deal of attention has been paid to individual variation in whole-animal (body-mass-adjusted) metabolic rate [2, 4], few studies have investigated variation in metabolic plasticity, but these are revealing significant variation in the extent to which animals can vary their metabolism. Plasticity varies across time within individuals (the BMR of rodents is more responsive to temperature in summer than in winter [31]), but it also varies among individuals and populations. At the population level, variation in metabolic rate plasticity occurs in response to temperature, diet quality and season: rufous-collared sparrows (*Zonotrichia capensis*) from Mediterranean ecosystems exhibit greater BMR flexibility in response to environmental temperature than do members of the same species from desert ecosystems, which have a largely inflexible BMR [39]. However, the BMR

of the desert sparrows show greater flexibility in response to diet than that of sparrows from the Mediterranean [40], emphasising how conclusions about metabolic rate flexibility depend on the environmental context. Greater plasticity in resting metabolic rate in response to temperature in high- *versus* low-latitude populations of cane toads (*Rhinella marina*) has been proposed to facilitate this species' invasion into higher latitude regions of Australia, due to an enhanced capacity to maintain critical physiological functions in the colder climate [41]. Similarly, cane toads from colder climates also exhibit greater plasticity in their lower temperature tolerance [42]. Across seasons, different subspecies of the stonechat (*Saxicola torquata*) exhibit differential plasticity in the annual cycle of their BMR when kept in a common environment with annually varying day length but constant temperature, indicating a genetic difference in programmed seasonal change in metabolic rate [43].

Evidence for population differences in metabolic rate plasticity in response to temperature has also been shown to exist at the cellular level: members of a high-latitude subspecies of the Atlantic killifish (*Fundulus heteroclitus*) increase their mitochondrial volume density and surface area relatively more than their low-latitude counterparts in response to cold acclimation [44], and these differences are reflected in high-latitude fish having a higher whole-animal metabolic rate [45]. These findings indicate that variation in plasticity may be important for adaptation to a seasonally more variable environment, and possibly to a climatically more variable future as well (keeping in mind that plasticity in killifish has been shown to differ in response to the cold but not the warm [45]). Plasticity of mitochondrial respiration in response to temperature also differs between clones of *Daphnia pulex* from temperate and subarctic environments, but without showing a clear latitudinal pattern, although clonal differences in mitochondrial function is again more pronounced when assayed in cold conditions [46].

Among individuals, differential flexibility in metabolic rate among fish is linked to their growth rates: the brown trout (*Salmo trutta*) that either increase or decrease their SMR the most in response to increased or decreased food availability, respectively, grow the fastest relative to their less flexible conspecifics [35]. A similar pattern is seen in a different fish species (qingbo,

123 *Spinibarbus sinensis*) where individuals with the most flexible SMR in response to changing food
124 levels grow the most when food availability is high [47]. Moreover, in response to food scarcity,
125 individual brown trout with the greatest reduction in SMR lose the least amount of fat in a simulated
126 overwintering scenario [36]. If lowering metabolic rate is an adaptive response to food shortage, then
127 this suggests that resource-poor conditions do not preclude an appropriate plastic response (i.e. the
128 cost of plasticity is not too great, despite energetic limitations), as otherwise suggested (reviewed in
129 [48, 49]). Phenotypes with more flexible metabolic rates may therefore be at a competitive advantage
130 in an environmentally unstable future with more fluctuating food supplies, as also suggested by
131 Canale & Henry [50].
132 Individual animals also differ in their metabolic rate flexibility in response to temperature
133 changes, with potentially important life history consequences. Siberian hamsters (*Phodopus*
134 *sungorus*) showed relatively consistent among-individual differences in the plasticity of their BMR
135 (repeatability of BMR plasticity = 0.31), and those hamsters that maintained their BMR level and
136 showed no sign of plasticity in response to cold exposure spent less time in torpor, compared to
137 individuals that increased their BMR in the cold [51]. The fitness consequences of not entering torpor
138 are hard to assess in a laboratory study in which food was readily available. Similarly, while
139 individual zebra finches (*Taeniopygia guttata*) that exhibited a larger increase in their resting
140 metabolic rate when exposed to the cold were better able to defend their body temperature [52] –
141 suggesting a lower risk of hypothermia-induced mortality in more flexible phenotypes – this also
142 assumes that there is sufficient food available to cover the increased energetic demand. In fish
143 (barramundi, *Lates calcarifer*), individuals with low SMR, MMR and aerobic scope exhibited a
144 larger increase in these metabolic rates relative to their high-metabolic-rate conspecifics when they
145 were faced with elevated temperatures but, when challenged with hypoxia, the metabolic rates of the
146 same individuals hardly changed [11]. Since both rapidly fluctuating temperatures and hypoxia occur
147 in the barramundi's natural environment in tropical Australia, and may worsen in a climatically more
148 extreme future, the low sensitivity of MMR and aerobic scope to hypoxia of low-metabolic-rate fish

149 could be advantageous as it allows these individuals to maintain their maximum aerobic capacity.
150 However, reduced hypoxia sensitivity may trade off with a larger increase in the 'cost of living'
151 (SMR) at warmer temperatures.
152
153 **3. Physiological/cellular mechanisms underlying (changes in) metabolic rates**
154 In order to interpret variation in metabolism we need to understand the underlying processes that are
155 responsible for oxygen consumption rates. Not all tissues or organs within the body have the same
156 energy demand: for instance, cells in the brain, liver and kidney are likely to use more ATP per unit
157 mass than skin or connective tissue [53], as will the intestines when digesting food [54]. A number of
158 studies have therefore attempted (with mixed success) to test whether individual variation in whole-
159 animal oxygen consumption can arise from differences in the relative size or activity of these body
160 components. As an example, Vézina *et al*. [55] found that variation in both BMR and M-sum of red
161 knots (*Calidris canutus*) was explained by variation in the residual mass of key organs after
162 correction for body mass. Thus, individuals with relatively large hearts and muscles for their size
163 tended to have higher BMRs, while a high M-sum was associated with proportionally large muscles,
164 heart and stomach. A similar study of eels (*Anguilla anguilla*) showed that the combined relative
165 mass of the heart, liver, spleen and intestine explained 38% of the among-individual variation in
166 SMR, despite these organs only comprising 1.6% of the total body mass [56]. This both highlights
167 the metabolic demands of these organs but also the potential difficulty in detecting their influence on
168 whole-animal metabolic rate, since they can make up a surprisingly small percentage of the total cells
169 (and hence mitochondria) in the body. If there is little variation among individuals in body
170 composition and in relative organ size (as may be the case in laboratory studies if all animals have
171 been kept in the same standardised benign conditions), then it is unlikely that measurements of organ
172 size will explain variation in their whole-organism metabolic rates (but see [57]). This may help
173 explain why other attempts to explain intraspecific variation in minimal or maximal metabolic rates
174 have found weak or no correlations with relative organ size (e.g. [58, 59]).

An alternative approach to determining the drivers of whole-animal metabolic rates is to examine variation in the functioning of key organs and tissues, rather than their size. One relevant measure is 'metabolic intensity', conceptually defined as the energy consumption per unit mass of tissue but in practice usually measured indirectly as either mitochondrial density or the activity of key rate-limiting mitochondrial enzymes [55]. Variation in both minimal and maximal metabolic rate amongst individuals has been found to correlate with differences in cytochrome c oxidase and/or citrate synthase activity in their mitochondria [55, 59], although these correlations are not always evident [56]. An alternative approach to quantifying mitochondrial function is to measure oxygen consumption rates of either isolated mitochondria or the mitochondria within samples of permeabilised tissue. Whilst care must be taken in the interpretation of these *in vitro* measurements, they can nonetheless reveal variation in mitochondrial performance that relates to variation in the metabolism of the animal from which they came. Thus, Salin *et al.* [60] showed that the SMR of individual brown trout was correlated with variation in the 'leak' respiration rate of their liver mitochondria, while MMR correlated with variation in the leak respiration of their muscle mitochondria. This leak respiration occurs when the mitochondria actively pump leaked protons back across the inner membrane in order to re-establish the proton gradient necessary for ATP production. Leak respiration is therefore a measure of the inefficiency of the mitochondria in producing ATP. The tissue-specificity of the correlations of mitochondrial leak respiration with SMR and MMR makes functional sense, since the liver is among the most metabolically active tissues under the conditions in which SMR is measured [53], while the muscles may contribute most to MMR [60]. One of the messages of that study is that a high metabolic rate can indicate inefficiency at producing ATP, although there may be a benefit of producing fewer damaging reactive oxygen species (ROS) [61]. There may also be more general cellular drivers of metabolic rate, such as the lipid composition of cellular membranes: comparisons between endotherms and ectotherms, and among species of endotherms, have found that BMR or SMR (after correction for body mass) increases with the degree

of poly-unsaturation of cellular membranes (the ‘membrane pacemaker’ hypothesis of metabolism [62]).

Clearly, there are a number of traits that can apparently co-vary with metabolic rate, but relationships amongst them can be complex and they do not always vary in parallel: as an example, among-individual variation in the size of energy-demanding organs does not always correlate with variation in their metabolic intensity [55, 63]. As a consequence, while these cross-sectional correlational studies can suggest cellular drivers of metabolic rate, they are rarely conclusive. A more convincing approach is to explore relationships in animals whose metabolic rates have been either up- or down-regulated (as a result of artificial selection, experimental manipulations or natural changes in environmental conditions). This approach has shown that the link between membrane lipids and metabolism is unlikely to be causal, since artificial selection experiments that caused a significant shift in minimal metabolism also altered membrane lipid composition, but in the opposite direction to that predicted by the membrane pacemaker hypothesis [64]. Causality can also be tested through manipulations of supposed cellular drivers of metabolic rate: while dietary manipulations that alter membrane lipid composition have not resulted in changes in minimal metabolism – again contrary to the predictions of the membrane pacemaker hypothesis [65] – manipulations of mitochondrial leak respiration (through use of uncoupling agents) have led to changes in whole-animal metabolic rate, indicating a causal link between mitochondrial and whole-animal respiration rates [66].

While it has usually not been possible to track within-individual changes in the underlying traits (such as organ size, mitochondrial function, etc.) since measurement often requires the animal to be sacrificed, changes in whole-animal metabolic traits can nonetheless prove informative. For example, although there is often a correlation between an individual’s BMR and its M-sum [8], Barceló *et al*. [12] were able to demonstrate through environmental manipulations that BMR and M-sum are under independent control: while cold exposure led to an increase in both the BMR and the M-sum of white-throated sparrows (*Zonotrichia albicollis*), a diet shift only altered their BMR and

had no effect on their M-sum. Exploration of the body composition of these birds showed that in both experimental manipulations the increase in BMR was related to increases in the relative size of digestive and excretory organs, whereas the increase in M-sum after cold exposure was presumed to be due to changes in the metabolic intensity of the muscles (since there was no increase in their size) [12], a response that is thought to be, at least in part, driven by changes in gene expression of several key metabolic pathways [67]. BMR was also found to change faster than either M-sum or MMR in birds exposed to an abrupt shift in ambient temperature, possibly because of differences in the relative rates at which organs can change their size *versus* their metabolic intensity [68].

Within-individual changes in organ size can happen during ontogeny, with consequences for metabolic rate: there is a shift in endotherms from BMR being driven by the fastest-growing organs early in life (when growth is fastest) to it being more influenced by organs with high metabolic intensity later in development [69]. There are also reversible changes in relative organ size (and hence metabolic rate) when animals are faced with major energetic challenges such as long-distance migrations [70] or infrequent but large meals [71, 72]. Components of mitochondrial structure and function can also shift in response to changes in ATP requirement [1, 73] and/or resource availability [74-76], with the typical response being an increase in the efficiency of ATP production (measured as ATP produced per unit consumption of oxygen) when conditions are more challenging [74, 75]. However, mitochondrial responses can differ between organs (and even between muscle types) of the same individual [74, 77], and increases in mitochondrial efficiency can come at a cost of increased rates of ROS production, which may explain why ATP production efficiency is not always maximised [76].

The process of acclimation can to some extent provide a buffer against the adverse effects on physiological processes of environmental change: several weeks' exposure to a higher ambient temperature reduces the thermal sensitivity of a range of physiological processes in ectotherms [22]. Temperature acclimation in whole-animal metabolic rate is matched by acclimation in mitochondrial function [78, 79] through alterations to mitochondrial membrane fluidity, and cytochrome c oxidase

and/or citrate synthase activity, but this capacity for full acclimation may only be over a limited temperature range that corresponds to expected temperatures within the geographical range of the species or population [44, 79, 80]. This has implications for the ability of organisms to cope with climate change, since the thermal range over which full acclimation can occur may need to evolve in parallel with rises in ambient temperatures.

4. Costs/benefits of metabolic plasticity in response to environmental change

It is possible to identify clear benefits to plasticity in metabolic rates: it has short-term benefits in terms of energy savings when food is short and enhances growth when food is plentiful [35, 36, 47]. Metabolic plasticity is also likely to increase resilience to climate change [22]. Given these benefits, there must be either costs or limitations that prevent metabolic plasticity from being greater than it is. There has been much discussion and speculation on the limits to plasticity in phenotypic traits in general [48, 81] and modelling exercises that explore how costs might influence the persistence of plasticity [82], but as yet there is little clear evidence of how significant the costs might be [48, 49].

These putative costs can be divided into two types: those of maintaining the potential to adjust metabolism, and those associated with actually undergoing a change in metabolism. Maintaining the capacity for plasticity might be expensive in terms of the machinery needed to monitor the environment (or the organism's state) and to then adjust/regulate the phenotype accordingly [81]. In the case of metabolic rate, it seems unlikely that the monitoring required for adjustment of metabolism would be greater than that needed to regulate other aspects of nutritional state, suggesting that this cost may not be significant. But the capacity to alter metabolism may be traded off against other traits important for fitness. This has been found in other contexts where behavioural flexibility traded off with foraging performance [83], and there is circumstantial evidence that selection for greater plasticity in metabolic rates in animals invading colder environments has led to reduced burst locomotor performance, suggesting that such trade-offs may exist [41], although there appears not to be any experimental evidence of this to date. It has also been suggested that plasticity in

physiological traits may come at the expense of the ability to express an extreme phenotype [84], but this has yet to be demonstrated in the context of metabolic flexibility.

It is possible that there might be pleiotropic effects that link metabolism and other traits, so constraining or imposing indirect costs on metabolic flexibility. However, while metabolic rate is clearly the result of many complex interacting factors that link mitochondria, tissues and organs, there is little clear evidence of how they individually or collectively may constrain the capacity of the animal to alter its metabolism. Indeed, the extent to which different physiological traits can vary apparently independently of one another is surprising [85], although links between different metabolic attributes within individuals – such as SMR and MMR – may be masked under benign conditions and only revealed in environments where constraints on trait variation are more pronounced [11]. Nonetheless, there may be limits to flexibility – for example, the extent to which mitochondria can ramp up ATP generation due to the risk of greatly increased ROS production [1]. The costs of altering metabolic processes to cope with a changed environment must also be balanced against the benefits: maintaining the means for extensive up- or down-regulation of metabolic physiology is only likely to be beneficial if the animal is likely to encounter significant fluctuations in energy supply or demand (e.g. if adopting a lifestyle of very irregular but large meals [86]). The cost of these metabolic adjustments may be reduced where they are programmed to follow either seasonal or life-history changes in energy demand [43], and where appetite changes in parallel [87, 88].

5. Possible evolutionary responses of metabolism to environmental change

How might we expect rates of metabolism to respond to long-term changes in the environment? A recent review found that metabolic rates had an overall average narrow-sense heritability of 0.19 (reported range: 0–0.72), so have the potential to evolve [6]. Indeed, evolutionary changes in metabolic rates have been demonstrated experimentally through selection experiments that have caused marked changes in BMR in 10-25 generations in rodents [89, 90], while the rapid evolution of

SMR has been demonstrated in natural populations of Trinidadian guppies (*Poecilia reticulata*) in response to changes in predation pressure [91] and selection on resting metabolic rate has been suggested to drive the evolution of metabolic rate plasticity in this species [92]. SMR has also been found to be under selection in a wild population of snails (*Helix aspersa*) where individuals with low to intermediate SMR had higher survival, independent of other performance traits (locomotion speed and dislodgement force) [93]. It is not only resting metabolism that may be under selection: low-food environments were recently found to select for higher maximum metabolic rates in juvenile Atlantic salmon (*Salmo salar*), presumably because of the positive association between metabolic rate and competitive ability in that species [94].

Given this scope for rapid evolutionary changes in metabolic rates, what responses are likely in a warming world? Since minimum levels of metabolism (SMR) have been found to be more plastic than maximum levels (MMR) in ectotherms, and exhibit greater thermal compensation (reduction) in response to chronic warming [29], it seems likely that the gradual elevation of metabolic rate incurred by global climate warming will drive an evolutionary reduction of at least ectothermic SMR. On the other hand, since those individuals with a relatively low SMR are faced with a proportionally greater increase in their metabolism when faced with an acute warming event [11], the evolution of minimum metabolic rates in a warming world may depend on the relative frequency of extreme warming events (heat waves), since these may impose different (and potentially opposite) selection pressures than do gradual changes in average temperatures. Along the same lines, since measurements of narrow-sense heritabilities of active metabolic rates are significantly higher than those of resting metabolism [6], this suggests that upper limits to metabolic rate are less dependent on environmental conditions, which reflects the findings of Sandblom *et al*. that maximum levels were more fixed than resting in a population of fish faced with warming [29]. Interestingly, measured heritabilities of metabolic rate appear to be higher in endotherms than in ectotherms [6], perhaps because the environmental impacts (particularly from temperature) are greater on ectotherm metabolism than that of endotherms.

An increase in the incidence of extreme environmental conditions may also be predicted to lead to an (evolutionary) increase in the use of torpor, aestivation or hibernation, since these are all means to save energy when conditions deteriorate through a controlled reduction in metabolic costs. This is supported by the observation that the highest proportion of species utilising torpor are found in regions with the most extreme climatic events [50]. Moreover, increased use of torpor occurs after extreme changes in the landscape, such as fire [95], and species utilising torpor have lower risk of extinction from environmental stress [96, 97]. Thus, the incidence, frequency and duration of torpor events can all be predicted to increase as a result of climate change.

6. Directions for future research

The costs and limits of phenotypic plasticity are still largely unknown, despite its importance for coping with and evolving in new environments [98, 99]. Given that among-individual variation is the raw material on which natural selection can operate, more empirical research is needed to investigate how and why plasticity in metabolic rates varies among members of a population. What are the costs of having a flexible metabolic rate, which must be traded off against its more obvious benefits? One possibility is that this plasticity constrains or co-varies with plasticity in other key organismal traits [100]. For instance, while a range of physiological traits (metabolic rate, haematocrit, corticosterone and immune function) can apparently change independently of one another [85], metabolic rate may constrain plasticity in behaviour [101]. Given the importance of behaviour for responding to a changing environment [102, 103], investigations are needed of the links between metabolic and behavioural plasticity. Ideally, these should be combined with a more integrative approach by which we can determine the mechanisms responsible for (variation in) plasticity, such as variation in the thermal sensitivity of mitochondria [18, 19]. The value of understanding this kind of underlying mechanism lies in our then having a much greater ability to predict organismal responses to new environmental conditions, beyond those for which we have empirical data – which could prove invaluable in a changing world. New approaches are being developed that allow appropriate samples

to be taken repeatedly from the same animal (e.g. [104]), which can greatly aid in our understanding of within-individual changes in the mechanisms underlying metabolic rate plasticity.

We also need to consider the life stage at which plasticity is occurring. Burggren [105] recently suggested that more emphasis should be placed on the phenotypic plasticity of juvenile or developing organisms in response to the more extreme and stochastic weather events associated with climate change, since plasticity in adult individuals is irrelevant if extreme events such as heat waves would have killed off these individuals before they reached maturity. Age- and size-dependent differences in metabolic responses to warming and ocean acidification have indeed been reported for marine molluscs [106], but the majority of studies consider just a single life stage (which is usually not early-stage juvenile).

Extending our research to field conditions may also prove fruitful for a fuller understanding of how and when plasticity is important for responding to environmental change. The continuing development and miniaturisation of accelerometers and heart rate tags allows for continuous and long-term monitoring of metabolic rate proxies within individuals, which can provide important information about physiological performance and plasticity in free-roaming animals [107, 108]. Field-based studies also have the benefit of testing animal responses in a context where environmental conditions (such as temperature, humidity, salinity and food supply) fluctuate rather than remain artificially constant (as in most laboratory studies). Studies conducted at constant temperature have proved to be inaccurate at predicting responses to fluctuating conditions [109]; moreover, as well as being more natural, these fluctuations in environmental parameters can be more relevant to animal performance than long-term averages [110] and may have profound effects on the ability of animals to acclimate and evolve in a changing world.

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References

1. Seebacher F, Brand MD, Else PL, Guderley H, Hulbert AJ, Moyes CD. 2010 Plasticity of oxidative metabolism in variable climates: molecular mechanisms. *Physiol. Biochem. Zool.* **83**, 721-732.
2. Burton T, Killen SS, Armstrong JD, Metcalfe NB. 2011 What causes intraspecific variation in resting metabolic rate and what are its ecological consequences? *Proc. R. Soc. B* **278**, 3465-3473.
3. Konarzewski M, Ksiazek A. 2013 Determinants of intra-specific variation in basal metabolic rate. *J. Comp. Physiol. B* **183**, 27-41.
4. Metcalfe NB, Van Leeuwen TE, Killen SS. 2016 Does individual variation in metabolic phenotype predict fish behaviour and performance? *J. Fish Biol.* **88**, 298-321.
5. Norin T, Clark TD. 2016 Measurement and relevance of maximum metabolic rate in fishes. *J. Fish Biol.* **88**, 122-151.
6. Pettersen AK, Marshall DJ, White CR. 2018 Understanding variation in metabolic rate. *J. Exp. Biol.* **221**, jeb166876.
7. Zeng LQ, Zhang AJ, Killen SS, Cao ZD, Wang YX, Fu SJ. 2017 Standard metabolic rate predicts growth trajectory of juvenile Chinese crucian carp (*Carassius auratus*) under changing food availability. *Biology Open* **6**, 1305-1309.
8. Auer SK, Killen SS, Rezende EL. 2017 Resting vs. active: a meta-analysis of the intra- and inter-specific associations between minimum, sustained, and maximum metabolic rates in vertebrates. *Funct. Ecol.* **31**, 1728-1738.
9. Swanson DL, McKechnie AE, Vézina F. 2017 How low can you go? An adaptive energetic framework for interpreting basal metabolic rate variation in endotherms. *J. Comp. Physiol. B* **187**, 1039-1056.
10. Wone BWM, Madsen P, Donovan ER, Labocha MK, Sears MW, Downs CJ, Sorensen DA, Hayes JP. 2015 A strong response to selection on mass-independent maximal metabolic rate without a correlated response in basal metabolic rate. *Heredity* **114**, 419-427.
11. Norin T, Malte H, Clark TD. 2016 Differential plasticity of metabolic rate phenotypes in a tropical fish facing environmental change. *Funct. Ecol.* **30**, 369-378.
12. Barceló G, Love OP, Vézina F. 2017 Uncoupling basal and summit metabolic rates in White-throated sparrows: Digestive demand drives maintenance costs, but changes in muscle mass are not needed to improve thermogenic capacity. *Physiol. Biochem. Zool.* **90**, 153-165.
13. Norin T, Malte H. 2011 Repeatability of standard metabolic rate, active metabolic rate and aerobic scope in young brown trout during a period of moderate food availability. *J. Exp. Biol.* **214**, 1668-1675.
14. White CR, Schimpf NG, Cassey P. 2013 The repeatability of metabolic rate declines with time. *J. Exp. Biol.* **216**, 1763-1765.

15. Cortés PA, Petit M, Lewden A, Milbergue M, Vézina F. 2015 Individual inconsistencies in basal and summit metabolic rate highlight flexibility of metabolic performance in a wintering passerine. *J. Exp. Zool. Part A Ecol. Genet. Physiol.* **323**, 179-190.
16. Auer SK, Bassar RD, Salin K, Metcalfe NB. 2016 Repeatability of metabolic rate is lower for animals living under field versus laboratory conditions. *J. Exp. Biol.* **219**, 631-634.
17. McKechnie AE. 2008 Phenotypic flexibility in basal metabolic rate and the changing view of avian physiological diversity: a review. *J. Comp. Physiol. B* **178**, 235-247.
18. Dillon ME, Wang G, Huey RB. 2010 Global metabolic impacts of recent climate warming. *Nature* **467**, 704-706.
19. Blier PU, Lemieux H, Pichaud N. 2014 Holding our breath in our modern world: will mitochondria keep the pace with climate changes? *Can. J. Zool.* **92**, 591-601.
20. Magozzi S, Calosi P. 2015 Integrating metabolic performance, thermal tolerance, and plasticity enables for more accurate predictions on species vulnerability to acute and chronic effects of global warming. *Glob. Change. Biol* **21**, 181-194.
21. Sandblom E, Gräns A, Axelsson M, Seth H. 2014 Temperature acclimation rate of aerobic scope and feeding metabolism in fishes: implications in a thermally extreme future. *Proc. R. Soc. B* **281**, 20141490.
22. Seebacher F, White CR, Franklin CE. 2015 Physiological plasticity increases resilience of ectothermic animals to climate change. *Nature Climate Change* **5**, 61-66.
23. Seebacher F. 2005 A review of thermoregulation and physiological performance in reptiles: what is the role of phenotypic flexibility? *J. Comp. Physiol. B* **175**, 453-461.
24. Hofmann GE, Todgham AE. 2010 Living in the now: Physiological mechanisms to tolerate a rapidly changing environment. *Annu. Rev. Physiol.* **72**, 127-145.
25. Staples JF. 2016 Metabolic flexibility: hibernation, torpor, and estivation. *Compr. Physiol.* **6**, 737-771.
26. Corder KR, Schaeffer PJ. 2015 Summit metabolic rate exhibits phenotypic flexibility with migration, but not latitude in a neotropical migrant, *Parkesia noveboracensis*. *J. Ornithol.* **156**, 547-550.
27. Hill AD, Taylor AC, Strang RHC. 1991 Physiological and metabolic responses of the shore crab *Carcinus maenas* (L.) during environmental anoxia and subsequent recovery. *J. Exp. Mar. Biol. Ecol.* **150**, 31-50.
28. Petit M, Lewden A, Vézina F. 2013 Intra-seasonal flexibility in avian metabolic performance highlights the uncoupling of basal metabolic rate and thermogenic capacity. *PLoS ONE* **8**, e68292.
29. Sandblom E, Clark TD, Gräns A, Ekström A, Brijs J, Sundström LF, Odelström A, Adill A, Aho T, Jutfelt F. 2016 Physiological constraints to climate warming in fish follow principles of plastic floors and concrete ceilings. *Nat. Commun.* **7**, 11447.
30. Norin T, Malte H, Clark TD. 2014 Aerobic scope does not predict the performance of a tropical eurythermal fish at elevated temperatures. *J. Exp. Biol.* **217**, 244-251.
31. Boratyński JS, Jefimow M, Wojciechowski MS. 2016 Phenotypic flexibility of energetics in acclimated Siberian hamsters has a narrower scope in winter than in summer. *J. Comp. Physiol. B* **186**, 387-402.
32. Nespolo RF, Bacigalupe LD, Rezende EL, Bozinovic F. 2001 When nonshivering thermogenesis equals maximum metabolic rate: Thermal acclimation and phenotypic plasticity of fossorial *Spalacopus cyanus* (Rodentia). *Physiol. Biochem. Zool.* **74**, 325-332.
33. van de Ven TMFN, Mzilikazi N, McKechnie AE. 2013 Phenotypic flexibility in body mass, basal metabolic rate and summit metabolism in southern red bishops (*Euplectes orix*): Responses to short term thermal acclimation. *Comp. Biochem. Physiol. A* **165**, 319-327.
34. Naya DE, Veloso C, Sabat P, Bozinovic F. 2009 The effect of short- and long-term fasting on digestive and metabolic flexibility in the Andean toad, *Bufo spinulosus*. *J. Exp. Biol.* **212**, 2167-2175.

35. Auer SK, Salin K, Rudolf AM, Anderson GJ, Metcalfe NB. 2015 Flexibility in metabolic rate confers a growth advantage under changing food availability. *J. Anim. Ecol.* **84**, 1405-1411.
36. Auer SK, Salin K, Anderson GJ, Metcalfe NB. 2016 Flexibility in metabolic rate and activity level determines individual variation in overwinter performance. *Oecologia* **182**, 703-712.
37. Zeng LQ, Fu C, Fu SJ. 2018 The effects of temperature and food availability on growth, flexibility in metabolic rates and their relationships in juvenile common carp. *Comp. Biochem. Physiol. A* **217**, 26-34.
38. Langer F, Havenstein N, Fietz J. 2018 Flexibility is the key: metabolic and thermoregulatory behaviour in a small endotherm. *J. Comp. Physiol. B* **188**, 553-563.
39. Cavieres G, Sabat P. 2008 Geographic variation in the response to thermal acclimation in rufous-collared sparrows: are physiological flexibility and environmental heterogeneity correlated? *Funct. Ecol.* **22**, 509-515.
40. Maldonado K, Bozinovic F, Cavieres G, Fuentes CA, Cortés A, Sabat P. 2012 Phenotypic flexibility in basal metabolic rate is associated with rainfall variability among populations of rufous-collared sparrow. *Zoology* **115**, 128-133.
41. Winwood-Smith HS, Alton LA, Franklin CE, White CR. 2015 Does greater thermal plasticity facilitate range expansion of an invasive terrestrial anuran into higher latitudes? *Conserv. Physiol.* **3**, cov010.
42. McCann SM, Kosmala GK, Greenlees MJ, Shine R. 2018 Physiological plasticity in a successful invader: rapid acclimation to cold occurs only in cool-climate populations of cane toads (*Rhinella marina*). *Conserv. Physiol.* **6**, cox072.
43. Versteegh MA, Helm B, Gwinner E, Tieleman BI. 2012 Annual cycles of metabolic rate are genetically determined but can be shifted by phenotypic flexibility. *J. Exp. Biol.* **215**, 3459-3466.
44. Dhillon RS, Schulte PM. 2011 Intraspecific variation in the thermal plasticity of mitochondria in killifish. *J. Exp. Biol.* **214**, 3639-3648.
45. Fangue NA, Richards JG, Schulte PM. 2009 Do mitochondrial properties explain intraspecific variation in thermal tolerance? *J. Exp. Biol.* **212**, 514-522.
46. Kake-Guena SA, Touisse K, Warren BE, Scott KY, Dufresne F, Blier PU, Lemieux H. 2017 Temperature-related differences in mitochondrial function among clones of the cladoceran *Daphnia pulex*. *J. Therm. Biol.* **69**, 23-31.
47. Zeng L-Q, Wang L, Wang G-N, Zeng Y, Fu S-J. 2017 The relationship between growth performance and metabolic rate flexibility varies with food availability in juvenile qingbo (*Spinibarbus sinensis*). *Comp. Biochem. Physiol. A* **212**, 56-63.
48. Van Buskirk J, Steiner UK. 2009 The fitness costs of developmental canalization and plasticity. *J. Evol. Biol.* **22**, 852-860.
49. Auld JR, Agrawal AA, Relyea RA. 2010 Re-evaluating the costs and limits of adaptive phenotypic plasticity. *Proc. R. Soc. B* **277**, 503-511.
50. Canale CI, Henry PY. 2010 Adaptive phenotypic plasticity and resilience of vertebrates to increasing climatic unpredictability. *Clim. Res.* **43**, 135-147.
51. Boratyński JS, Jefimow M, Wojciechowski MS. 2017 Individual differences in the phenotypic flexibility of basal metabolic rate in Siberian hamsters are consistent on short- and long-term timescales. *Physiol. Biochem. Zool.* **90**, 139-152.
52. Briga M, Verhulst S. 2017 Individual variation in metabolic reaction norms over ambient temperature causes low correlation between basal and standard metabolic rate. *J. Exp. Biol.* **220**, 3280-3289.
53. Rolfe DFS, Brown GC. 1997 Cellular energy utilization and molecular origin of standard metabolic rate in mammals. *Physiol. Rev.* **77**, 731-758.
54. Daan S, Masman D, Groenewold A. 1990 Avian basal metabolic rates - their association with body-composition and energy-expenditure in nature. *Am. J. Physiol.* **259**, R333-R340.

55. Vézina F, Gerson AR, Guglielmo CG, Piersma T. 2017 The performing animal: causes and consequences of body remodeling and metabolic adjustments in red knots facing contrasting thermal environments. *Am. J. Physiol.* **313**, R120-R131.
56. Boldsen MM, Norin T, Malte H. 2013 Temporal repeatability of metabolic rate and the effect of organ mass and enzyme activity on metabolism in European eel (*Anguilla anguilla*). *Comp. Biochem. Physiol. A* **165**, 22-29.
57. Konarzewski M, Diamond J. 1995 Evolution of basal metabolic rate and organ masses in laboratory mice. *Evolution* **49**, 1239-1248.
58. Chappell MA, Garland T, Robertson GF, Saltzman W. 2007 Relationships among running performance, aerobic physiology and organ mass in male Mongolian gerbils. *J. Exp. Biol.* **210**, 4179-4197.
59. Norin T, Malte H. 2012 Intraspecific variation in aerobic metabolic rate of fish: relations with organ size and enzyme activity in Brown trout. *Physiol. Biochem. Zool.* **85**, 645-656.
60. Salin K, Auer SK, Rudolf AM, Anderson GJ, Selman C, Metcalfe NB. 2016 Variation in metabolic rate among individuals is related to tissue-specific differences in mitochondrial leak respiration. *Physiol. Biochem. Zool.* **89**, 511-523.
61. Salin K, Auer SK, Rudolf AM, Anderson GJ, Cairns AG, Mullen W, Hartley RC, Selman C, Metcalfe NB. 2015 Individuals with higher metabolic rates have lower levels of reactive oxygen species in vivo. *Biol. Lett.* **11**, 20150538.
62. Hulbert AJ, Else PL. 1999 Membranes as possible pacemakers of metabolism. *J. Theor. Biol.* **199**, 257-274.
63. Vézina F, Williams TD. 2005 Interaction between organ mass and citrate synthase activity as an indicator of tissue maximal oxidative capacity in breeding European Starlings: implications for metabolic rate and organ mass relationships. *Funct. Ecol.* **19**, 119-128.
64. Brzek P, Bielawska K, Ksiazek A, Konarzewski M. 2007 Anatomic and molecular correlates of divergent selection for basal metabolic rate in laboratory mice. *Physiol. Biochem. Zool.* **80**, 491-499.
65. Price ER, Sirsat TS, Sirsat SKG, Curran T, Venables BJ, Dzialowski EM. 2018 The membrane pacemaker hypothesis: novel tests during the ontogeny of endothermy. *J. Exp. Biol.* **221**, 174466.
66. Salin K, Luquet E, Rey B, Roussel D, Voituron Y. 2012 Alteration of mitochondrial efficiency affects oxidative balance, development and growth in frog (*Rana temporaria*) tadpoles. *J. Exp. Biol.* **215**, 863-869.
67. Cheviron ZA, Swanson DL. 2017 Comparative transcriptomics of seasonal phenotypic flexibility in two North American songbirds. *Integr. Comp. Biol* **57**, 1040-1054.
68. Dubois K, Hallot F, Vézina F. 2016 Basal and maximal metabolic rates differ in their response to rapid temperature change among avian species. *J. Comp. Physiol. B* **186**, 919-935.
69. Vézina F, Love OP, Lessard M, Williams TD. 2009 Shifts in metabolic demands in growing altricial nestlings illustrate context-specific relationships between basal metabolic rate and body composition. *Physiol. Biochem. Zool.* **82**, 248-257.
70. Piersma T, Drent J. 2003 Phenotypic flexibilty and the evolution of organismal design. *Trends Ecol.Evol.* **18**, 228-233.
71. Wang T, Hung CCY, Randall DJ. 2006 The comparative physiology of food deprivation: From feast to famine. *Annu. Rev. Physiol.* **68**, 223-251.
72. Secor SM. 2008 Digestive physiology of the Burmese python: broad regulation of integrated performance. *J. Exp. Biol.* **211**, 3767-3774.
73. Nielsen J, Gejl KD, Hey-Mogensen M, Holmberg HC, Suetta C, Krustrup P, Elemans CPH, Ørtenblad N. 2017 Plasticity in mitochondrial cristae density allows metabolic capacity modulation in human skeletal muscle. *J. Physiol.* **595**, 2839-2847.
74. Monternier PA, Fongy A, Hervant F, Drai J, Collin-Chavagnac D, Rouanet JL, Roussel D. 2015 Skeletal muscle phenotype affects fasting-induced mitochondrial oxidative phosphorylation flexibility in cold-acclimated ducklings. *J. Exp. Biol.* **218**, 2427-2434.

75. Bourguignon A, Rameau A, Toullec G, Romestaing C, Roussel D. 2017 Increased mitochondrial energy efficiency in skeletal muscle after long-term fasting: its relevance to animal performance. *J. Exp. Biol.* **220**, 2445-2451.
76. Salin K, Villasevil EM, Anderson GJ, Auer SK, Selman C, Hartley RC, Mullen W, Chinopoulos C, Metcalfe NB. in press Decreased mitochondrial metabolic requirements in fasting animals carry an oxidative cost. *Funct. Ecol.*
77. Cortés PA, Bozinovic F, Blier PU. 2018 Mitochondrial phenotype during torpor: Modulation of mitochondrial electron transport system in the Chilean mouse-opossum *Thylamys elegans*. *Comp. Biochem. Physiol. A* **221**, 7-14.
78. Chung DJ, Schulte PM. 2015 Mechanisms and costs of mitochondrial thermal acclimation in a eurythermal killifish (*Fundulus heteroclitus*). *J. Exp. Biol.* **218**, 1621-1631.
79. Chung DJ, Bryant HJ, Schulte PM. 2017 Thermal acclimation and subspecies-specific effects on heart and brain mitochondrial performance in a eurythermal teleost (*Fundulus heteroclitus*). *J. Exp. Biol.* **220**, 1459-1471.
80. Dahlhoff E, Somero GN. 1993 Effects of temperature on mitochondria from abalone (genus *Haliotis*) - adaptive plasticity and its limits. *J. Exp. Biol.* **185**, 151-168.
81. DeWitt TJ, Sih A, Wilson DS. 1998 Costs and limits of phenotypic plasticity. *Trends Ecol.Evol.* **13**, 77-81.
82. Beaman JE, White CR, Seebacher F. 2016 Evolution of plasticity: Mechanistic link between development and reversible acclimation. *Trends Ecol. Evol* **31**, 237-249.
83. Pintor LM, McGhee KE, Roche DP, Bell AM. 2014 Individual variation in foraging behavior reveals a trade-off between flexibility and performance of a top predator. *Behav. Ecol. Sociobiol.* **68**, 1711-1722.
84. Stillman JH. 2003 Acclimation capacity underlies susceptibility to climate change. *Science* **301**, 65-65.
85. Buehler DM, Vézina F, Goymann W, Schwabl I, Versteegh M, Tieleman BI, Piersma T. 2012 Independence among physiological traits suggests flexibility in the face of ecological demands on phenotypes. *J. Evol. Biol.* **25**, 1600-1613.
86. Secor SM. 2001 Regulation of digestive performance: a proposed adaptive response. *Comp. Biochem. Physiol. A* **128**, 565-577.
87. Mrosovsky N, Sherry DF. 1980 Animal anorexias. *Science* **207**, 837-842.
88. Bull CD, Metcalfe NB, Mangel M. 1996 Seasonal matching of foraging to anticipated energy requirements in anorexic juvenile salmon. *Proc. R. Soc. Lond. B* **263**, 13-18.
89. Sadowska ET, Stawski C, Rudolf A, Dheyongera G, Chrzascik KM, Baliga-Klimczyk K, Koteja P. 2015 Evolution of basal metabolic rate in bank voles from a multidirectional selection experiment. *Proc. R. Soc. B* **282**, 20150025.
90. Ksiazek A, Czerniecki J, Konarzewski M. 2009 Phenotypic flexibility of traits related to energy acquisition in mice divergently selected for basal metabolic rate (BMR). *J. Exp. Biol.* **212**, 808-814.
91. Auer SK, Dick CA, Metcalfe NB, Reznick DN. 2018 Metabolic rate evolves rapidly and in parallel with the pace of life history. *Nat. Commun.* **9**, 14.
92. Handelsman CA, Broder ED, Dalton CM, Ruell EW, Myrick CA, Reznick DN, Ghalambor CK. 2013 Predator-induced phenotypic plasticity in metabolism and rate of growth: rapid adaptation to a novel environment. *Integr. Comp. Biol* **53**, 975-988.
93. Artacho P, Nespolo RF. 2009 Natural selection reduces energy metabolism in the Garden snail, *Helix aspersa* (Cornu Aspersum). *Evolution* **63**, 1044-1050.
94. Auer SK, Anderson GJ, McKelvey S, Bassar RD, McLennan D, Armstrong JD, Nislow KH, Downie HK, McKelvey L, Morgan TAJ, et al. 2018 Nutrients from salmon parents alter selection pressures on their offspring. *Ecol. Lett.* **21**, 287-295.
95. Stawski C, Körtner G, Nowack J, Geiser F. 2015 The importance of mammalian torpor for survival in a post-fire landscape. *Biol. Lett.* **11**, 20150134.

96. Liow LH, Fortelius M, Lintulaakso K, Mannila H, Stenseth NC. 2009 Lower extinction risk in sleep-or-hide mammals. *Am. Nat.* **173**, 264-272.
97. Geiser F, Turbill C. 2009 Hibernation and daily torpor minimize mammalian extinctions. *Naturwissenschaften* **96**, 1235-1240.
98. Chevin L-M, Lande R, Mace GM. 2010 Adaptation, plasticity, and extinction in a changing environment: Towards a predictive theory. *PLoS Biol.* **8**, e1000357.
99. Ghalambor CK, Hoke KL, Ruell EW, Fischer EK, Reznick DN, Hughes KA. 2015 Non-adaptive plasticity potentiates rapid adaptive evolution of gene expression in nature. *Nature* **525**, 372-375.
100. Fischer EK, Ghalambor CK, Hoke KL. 2016 Plasticity and evolution in correlated suites of traits. *J. Evol. Biol.* **29**, 991-1002.
101. Biro PA, Garland T, Beckmann C, Ujvari B, Thomas F, Post JR. 2018 Metabolic scope as a proximate constraint on individual behavioral variation: Effects on personality, plasticity, and predictability. *Am. Nat.* **192**, 142-154.
102. Sih A. 2013 Understanding variation in behavioural responses to human-induced rapid environmental change: a conceptual overview. *Anim. Behav.* **85**, 1077-1088.
103. Guzzo MM, Blanchfield PJ, Rennie MD. 2017 Behavioral responses to annual temperature variation alter the dominant energy pathway, growth, and condition of a cold-water predator. *Proc. Natl. Acad. Sci. U. S. A* **114**, 9912-9917.
104. Stier A, Romestaing C, Schull Q, Lefol E, Robin JP, Roussel D, Bize P. 2017 How to measure mitochondrial function in birds using red blood cells: a case study in the king penguin and perspectives in ecology and evolution. *Methods Ecol. Evol.* **8**, 1172-1182.
105. Burggren WW. 2018 Developmental phenotypic plasticity helps bridge stochastic weather events associated with climate change. *J. Exp. Biol.* **221**, jeb161984.
106. Carey N, Sigwart JD. 2014 Size matters: plasticity in metabolic scaling shows body-size may modulate responses to climate change. *Biol. Lett.* **10**, 20140408.
107. Hussey NE, Kessel ST, Aarestrup K, Cooke SJ, Cowley PD, Fisk AT, Harcourt RG, Holland KN, Iverson SJ, Kocik JF, et al. 2015 Aquatic animal telemetry: A panoramic window into the underwater world. *Science* **348**, 1255642.
108. Payne NL, Smith JA, van der Meulen DE, Taylor MD, Watanabe YY, Takahashi A, Marzullo TA, Gray CA, Cadiou G, Suthers IM. 2016 Temperature dependence of fish performance in the wild: links with species biogeography and physiological thermal tolerance. *Funct. Ecol.* **30**, 903-912.
109. Niehaus AC, Angilletta MJ, Sears MW, Franklin CE, Wilson RS. 2012 Predicting the physiological performance of ectotherms in fluctuating thermal environments. *J. Exp. Biol.* **215**, 694-701.
110. Clusella-Trullas S, Blackburn TM, Chown SL. 2011 Climatic predictors of temperature performance curve parameters in ectotherms imply complex responses to climate change. *Am. Nat.* **177**, 738-751.