

# Thermal acclimation and the dynamic response of plant respiration to temperature

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**Temperature-mediated changes in plant respiration ( $R$ ) are now accepted as an important component of the biosphere's response to global climate change. Here we discuss the underlying mechanisms responsible for the dynamic response of plant respiration to short and long-term temperature changes. The  $Q_{10}$  is often assumed to be 2.0 (i.e.  $R$  doubles per 10°C rise in temperature); however, the  $Q_{10}$  is not constant (e.g. it declines near-linearly with increasing temperature). The temperature dependence of  $Q_{10}$  is linked to shifts in the control exerted by maximum enzyme activity at low temperature and substrate limitations at high temperature. In the long term, acclimation of  $R$  to temperature is common, in effect reducing the temperature sensitivity of  $R$  to changes in thermal environment, with the temperature during plant development setting the maximal thermal acclimation of  $R$ .**

Respiration ( $R$ ) is the powerhouse that provides the driving force for biosynthesis, cellular maintenance and active transport in plants (Box 1). Respiration couples the production of ATP, reducing equivalents and carbon skeletons to the release of large amounts of CO<sub>2</sub> into the atmosphere; 30–80% of daily photosynthetic carbon gain is released into the atmosphere by plant  $R$  [1–3]. At the ecosystem level, plant  $R$  contributes 30–65% of the total CO<sub>2</sub> released into the atmosphere [4–6] with the remaining CO<sub>2</sub> release coming from heterotrophic soil  $R$ . The balance between ecosystem  $R$  (i.e. plant  $R$  plus heterotrophic soil  $R$ ) and photosynthesis determines whether an ecosystem is a net source or sink of CO<sub>2</sub> into the atmosphere. Riccardo Valentini *et al.* [7] concluded that ecosystem  $R$  is the main determinant of net ecosystem CO<sub>2</sub> exchange in northern European forests. Globally, terrestrial plant  $R$  releases ~60 gigatonnes of carbon per year (Gt C year<sup>-1</sup>) into the atmosphere [8,9]. Although terrestrial photosynthesis represents a greater carbon flux than terrestrial plant  $R$  on a global scale, plant  $R$  is a large flux compared with the relatively small release of CO<sub>2</sub> from the combustion of fossil fuels, cement production and changing land use (total 7.1 Gt C year<sup>-1</sup> [8,9]). If temperatures change as a result of greenhouse warming, understanding the effect of temperature on plant  $R$  will be

a prerequisite for predicting plant growth and performance, net CO<sub>2</sub> fluxes into the atmosphere and atmospheric CO<sub>2</sub> concentrations in the future. The role of temperature in mediating a potentially large positive feedback on respiratory flux from plants and soils to the atmosphere is of concern for its effects on global atmospheric CO<sub>2</sub> concentrations and contribution to greenhouse warming [10–12].

Although it has been long recognized that  $R$  is temperature sensitive [13–15], many questions have remained unanswered about the underlying factors responsible for the dynamic response of  $R$  to short- and long-term changes in temperature. In this review, we examine recent published reports to provide a mechanistic explanation for why the  $Q_{10}$  (see Glossary) of plant  $R$  varies with measurement temperature and respiratory substrate availability. The role of temperature-mediated changes in substrate availability and/or changes in maximum catalytic activity of respiratory enzymes (i.e.  $V_{\max}$ ) in

## Glossary

**Homeostasis:** Similar rates of  $R$  exhibited by warm and cold-acclimated plants, when compared at their respective growth temperatures.

**$Q_{10}$ :** The proportional change in  $R$  per 10°C rise in temperature. Thus, if the  $Q_{10}$  is 2.0,  $R$  will double for each 10°C increase in temperature.  $Q_{10}$  values can be calculated for temperature intervals less than 10°C using equation 1:

$$Q_{10} = (R_2/R_1)^{10/(T_2 - T_1)} \quad [\text{Eqn 1}]$$

where  $R_2$  and  $R_1$  are rates of  $R$  measured at temperatures  $T_2$  (high temperature) and  $T_1$  (low temperature), respectively.

**PUMP:** Plant uncoupling mitochondrial protein. Transports protons across the inner mitochondrial membrane (from the inter-membrane space to the matrix) via a fatty acid cycling system.

**Transition temperature ( $T_m$ ):** Temperature at which membranes undergo a conversion from a fluid state (that exists at warm-moderate temperatures) to a gel-like state (that exists in chilled membranes).  $T_m$  values as high as 15–20°C have been reported for some species, with increases in the unsaturated fatty acid content of membranes decreasing the  $T_m$  [40]. Below the  $T_m$ , the function of membrane-bound respiratory enzymes is likely to be reduced owing to the gel-like state of the membrane. The transport of substrates across all membranes and the leakage of protons across the inner mitochondrial membrane, are likely to be reduced at temperatures below the  $T_m$  [40]. The leakage of protons across the inner mitochondrial membrane might result in mitochondrial electron transport being more restricted by proton accumulation within the mitochondrial inter-membrane space (Box 1) in the cold than at higher temperatures.

**$V_{\max}$ :** Maximum rate of enzyme activity at a given temperature. In this review,  $V_{\max}$  is used to describe maximal flux through the respiratory system as a whole (or parts thereof) in the absence of other limiting factors (e.g. substrate supply and adenylates).



would restrict flux via CIII and CIV). Abbreviations: AOX: alternative oxidase;  $e^-$ : electrons; F6P: fructose 6 phosphate; F1,6bP: fructose 1,6 biphosphate; CI: complex I; CII: complex II (succinate dehydrogenase); CIII: complex III (Cytochrome *b/c*<sub>1</sub> complex); Cyt *c*: cytochrome *c*; CIV: complex IV (cytochrome *c* oxidase); CV: ATP synthase; ExtNDH:

external NAD(P)H dehydrogenase(s); PDC: pyruvate dehydrogenase complex; PEP: phosphoenolpyruvate; PFK, phosphofructokinase; PK: pyruvate kinase; Pyr: pyruvate; RIB: internal, rotenone-insensitive NADH dehydrogenase; UQ and UQ<sub>2</sub>: oxidized and reduced ubiquinone, respectively.

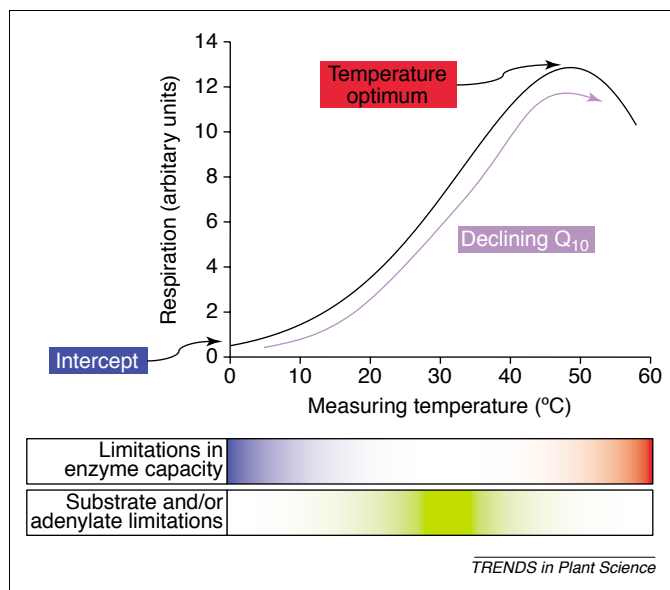
determining the degree of acclimation to long-term changes in temperature is discussed. We highlight the importance of temperature-mediated changes in leaf and root development for thermal acclimation of *R* in plants. When discussing acclimation, we focus on temperature-mediated changes in respiratory flux ( $O_2$  uptake and/or  $CO_2$  release); we do not address other metabolic and structural changes associated with acclimation to a new growth temperature. In related fields of plant biology, acclimation is used to describe the effects of long-term changes in temperature on growth and maintenance processes, membrane properties, freezing tolerance, gene expression and targeted increases in specific proteins [16–18]. Collectively, such changes contribute to the effect of growth temperature on the changes in the temperature-response of *R* that defines the scope of our use of the term acclimation.

### Dynamic responses of plant *R* to temperature

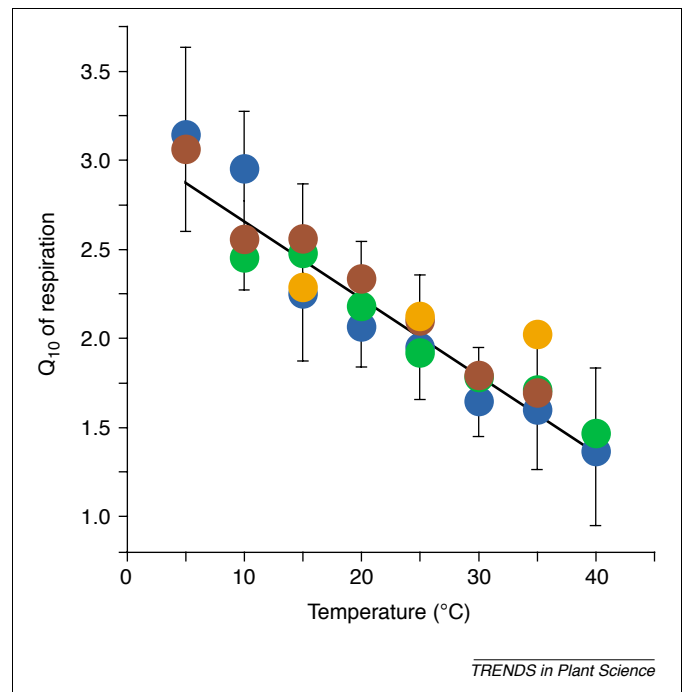
Often, temperature-mediated variations in *R* are modelled as a simple exponential function of temperature with a constant  $Q_{10}$  of near 2.0. In some respects, this is surprising because it has been long recognized that the  $Q_{10}$  is neither constant nor near 2.0 except over a limited temperature range [13–15]; rather, the  $Q_{10}$  is dependent upon the shape of the temperature-response curve and the

range of measurement temperatures used in its determination (Fig. 1). For example, the  $Q_{10}$  declines linearly with short-term (minutes to hours) increases in measurement temperature [14,15,19]. Recent work has shown that the temperature-dependence of the  $Q_{10}$  is predictable across diverse plant taxa and biomes (Fig. 2) [19]. As a result, short-term increases in temperature can have a greater potential impact on plant *R* in plants growing in cold climates, such as the Arctic (e.g. average leaf  $R$   $Q_{10}$  = 2.56), than in plants growing in warmer environments, such as the tropics (e.g. average leaf  $R$   $Q_{10}$  = 2.14) [19].

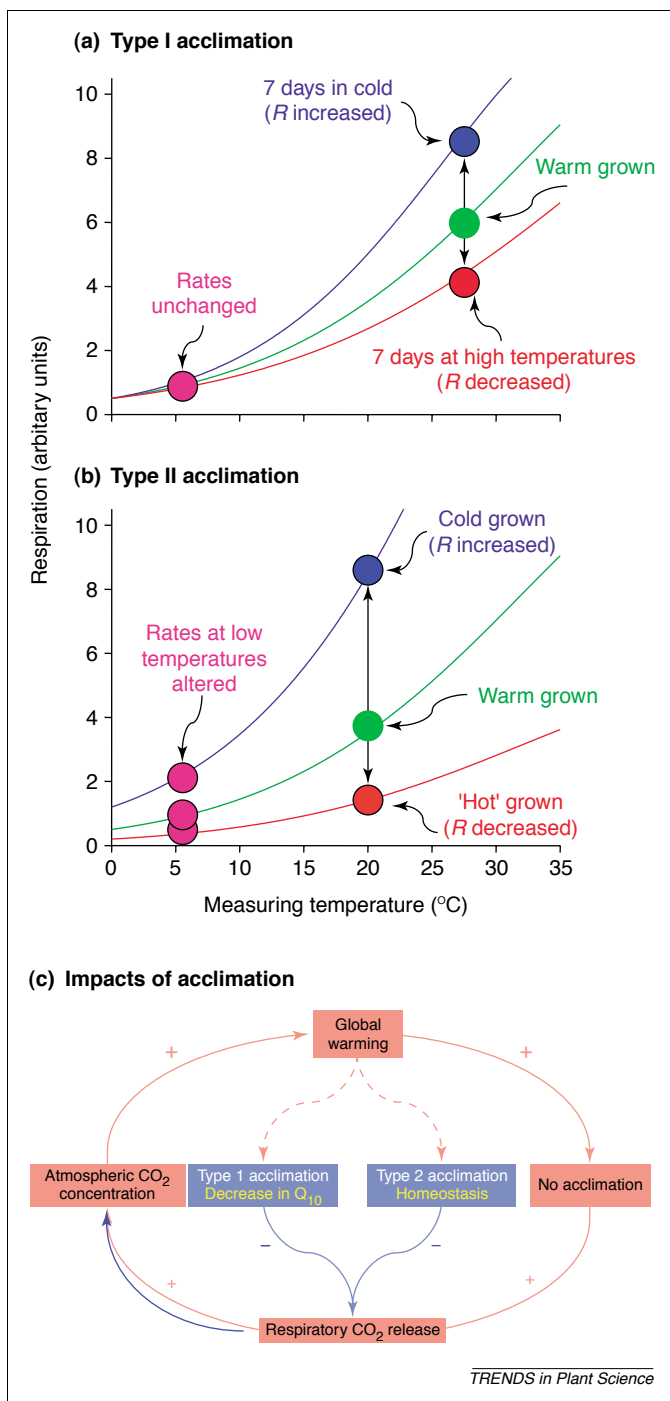
Changes in growth temperature that last several days also alter the  $Q_{10}$  of leaf and root *R* in some species [20]. Leaf  $Q_{10}$  values are higher in winter than in summer in several temperate evergreen plant species [21,22]. Moreover,  $Q_{10}$  values can be influenced by other environmental factors such as drought [23,24] or light environment [25,26]. In spite of increasing acceptance of such variability in the  $Q_{10}$  of *R*, few attempts have been made to elucidate the underlying mechanisms responsible for the variability.



**Fig. 1.** Temperature response of respiration (*R*). Assuming a rate of *R* of 0.5 at 0°C (arbitrary units), *R* at other temperatures was predicted assuming a linear decline in the  $Q_{10}$  with increasing temperature (Fig. 2). Both the intercept (i.e. *R* at 0°C) and the temperature optimum of *R* (i.e. temperature where maximal rates of *R* occur) are shown. The lower panels indicate the degree to which respiratory flux is likely to be limited by enzyme capacity versus substrate supply and adenylates. The temperatures where respiratory flux is likely to be limited by maximum catalytic enzyme activity (i.e.  $V_{max}$ ) are indicated in blue (limitations in the cold) and red (limitations at supra-optimal temperatures). At moderate temperatures, respiratory flux is more likely to be regulated by the availability of substrate and/or adenylates (i.e. the absolute concentration of ADP and the ratio of ATP:ADP).



**Fig. 2.**  $Q_{10}$  of foliar respiration rates of plants in relation to short-term measurement temperature. Symbols are the mean  $Q_{10}$  of species of arctic, indicated in blue (49 species), boreal, indicated in green (24 species), temperate, indicated in brown (50 species) and tropical, indicated in orange (3 species). Error bars indicate  $\pm 1$  sd of all observations across biomes for each 5°C temperature class. Covariance analysis indicated that slopes did not differ among the arctic, boreal, and temperate biome groups ( $P = 0.52$ ), but was less negative for tropical species. A single linear regression was fitted to all foliar data ( $R^2 = 0.46$ ,  $n = 650$ ) with the temperature dependence of  $Q_{10}$  of foliar  $R = 3.090 - 0.043 T$ . Data are of 121 species of 23 published studies. The analysis and figure is adapted from [19] to include data of [68]. A similar survey of published  $Q_{10}$  values (5–35°C) of root *R* of 21 boreal and temperate tree species (M.G. Tjoelker *et al.*, unpublished; not shown in Fig. 2) reveals the temperature dependence of  $Q_{10}$  of root  $R = 3.000 - 0.045 T$ .



**Fig. 3.** Theoretical examples of two types of acclimation. (a) Type I and (b) Type II, and (c) their effects on the positive feedback respiration ( $R$ ) might play in determining future atmospheric concentrations of  $\text{CO}_2$  and global surface temperatures via the greenhouse effect. (a) In Type I acclimation, changes in growth temperature result in changes in the  $Q_{10}$  of  $R$  with no change in the value of  $R$  at low temperatures (i.e. the intercept remains unchanged). Rather, changes in  $R$  are only observed at moderate to high temperatures. Shifting to low growth temperatures for an extended period typically results in an increase in the  $Q_{10}$ , whereas the  $Q_{10}$  decreases following a shift to high growth temperatures. (b) Type II acclimation results in changes in  $R$  at both low and high temperatures (i.e. the overall elevation of the temperature response curve is affected). No changes in the  $Q_{10}$  of  $R$  are necessary for Type II acclimation. Type II acclimation will result in a greater degree of homeostasis of  $R$  than Type I acclimation. (c) Both Types I and II acclimation could potentially reduce the positive feedback that  $R$  might play in a coupled climate-carbon cycle system [10–12]. In the absence of acclimation, climate warming would trigger an increase in plant and soil  $R$ , resulting in additional  $\text{CO}_2$  release and global warming (red lines). Acclimation would weaken this positive feedback (blue lines); the degree of weakening would be greater for Type II than for Type I acclimation owing to the greater degree of homeostasis achieved in

Over longer time periods, plant  $R$  in many species acclimates to changes in prevailing ambient temperature (Fig. 3) [22,27–32]. Acclimation of  $R$  can occur within a 1–2 day period following a change in ambient temperature [20,22,33], raising the possibility that plant  $R$  dynamically acclimates to changes in the thermal environment. Acclimation is also observed in soil  $R$  [11,34]. Acclimation is the adjustment of rates of  $R$  to compensate for a change in temperature [32]. It is associated with changes in the intercept, slope (shape of the curve), and/or temperature optimum (i.e. temperature where maximal rates of  $R$  occur) of the short-term temperature response function of  $R$  (Fig. 3). In effect, thermal acclimation results in  $R$  at a standard measuring temperature increasing upon acclimation to colder temperatures and declining upon acclimation to warmer temperatures (Fig. 3). For example, shifting warm-grown plants to a lower growth temperature (e.g. for several days) increases the rate of  $R$  measured at a moderate, common measurement temperature (Fig. 3a) [20,22]. Differences in the rate of  $R$  at common measurement temperatures are also exhibited by plants grown and developed under contrasting temperature regimes (either in the laboratory or in the field) (Fig. 3b) [27,28,30,31,35]. Overall, thermal acclimation of respiration results in a reduction in the long-term temperature sensitivity of  $R$  [28]. As a result, acclimation can play an important role in weakening positive feedback through the warming-respiration-atmospheric  $\text{CO}_2$  concentration connection (Fig. 3c) [10–12]. Moreover, acclimation can result in  $R$  being nearly identical in contrasting thermal environments [32] (although complete HOMEOSTASIS seems to be the exception rather than the rule).

#### Why is the $Q_{10}$ of plant $R$ temperature-dependent?

Recent evidence indicates that the most important factor responsible for the temperature dependence of the  $Q_{10}$  (Fig. 2) is the effect of measurement temperature on control exerted by maximum enzyme activity over respiratory  $\text{O}_2$  uptake or  $\text{CO}_2$  release [36]. At low measurement temperatures (e.g. 5°C), respiratory flux is probably limited by the  $V_{\text{max}}$  (lower panels in Fig. 1) of the respiratory apparatus (i.e. glycolysis, the TCA cycle and mitochondrial electron transport, Box 1). This is either because of the inhibitor effect of cold on potential enzyme activity *per se* (both in soluble and membrane-bound compartments) and/or limitations on the function of enzymes embedded in membranes at temperatures below the TRANSITION TEMPERATURE ( $T_m$ ). At moderately high temperatures (e.g. 25°C), respiratory flux is less limited by enzymatic capacity [37] because of increases in the  $V_{\text{max}}$  of enzymes in soluble and membrane-bound compartments; here,  $R$  is likely to be limited by substrate availability and/or adenylates (in particular the ratio of ATP to ADP and the concentration of ADP *per se*; [38]). Increased leakiness of membranes at temperatures above the transition temperature (particularly at high temperatures) could further contribute to substrate limitations

tissues that exhibit Type II acclimation. In a recent modelling exercise, dynamic thermal acclimation to daily temperature variation was shown to reduce annual  $\text{CO}_2$  release by 40% compared with a theoretical case without acclimation [32].

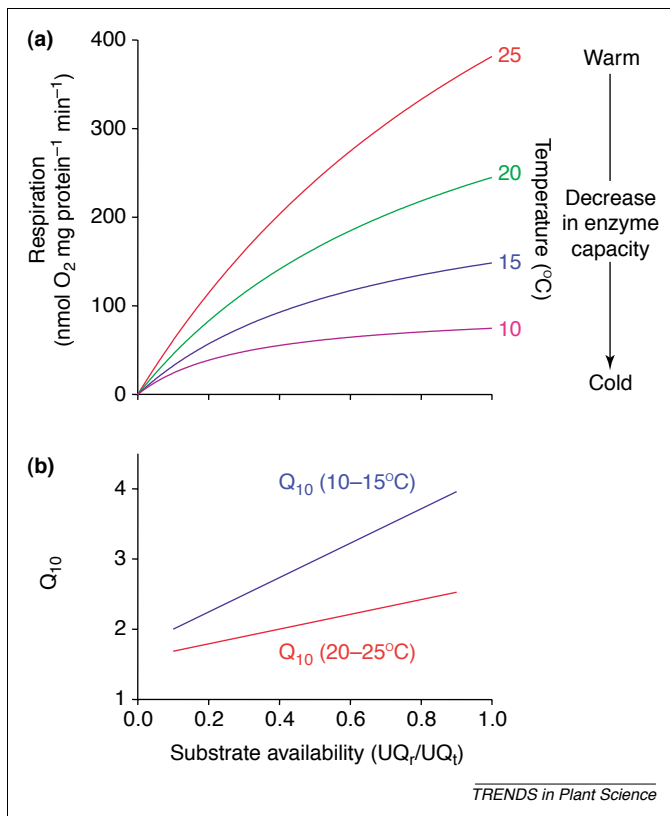
because concentration gradients (e.g. of TCA cycle intermediates) are more difficult to maintain when mitochondrial membranes are too fluid. The net result of temperature-mediated shifts in control from capacity (at low temperatures) to substrate or adenylate limitation (at moderately high temperatures) (Fig. 1) is that a rise in measurement temperature has less impact on respiratory flux at moderate-high temperatures than it does in the cold. As a result, the calculated  $Q_{10}$  is lower when calculated across a high measurement temperature range than a range of low measurement temperatures (Fig. 4).

What evidence is there that  $R$  is capacity-limited in the cold? To definitively establish whether respiratory enzyme capacity limits respiratory flux in the cold, data are needed on the maximum potential flux of the respiratory apparatus in intact tissues at low temperatures. This can be obtained via measurements of respiration in

isolated mitochondria, in the presence of saturating substrates and ADP. Mitochondrial rates can be then scaled up to represent rates per gram of root tissue using a mitochondrial marker enzyme such as fumarase [39]. Although published data are currently lacking, the  $Q_{10}$  for *in vivo* mitochondrial  $O_2$  uptake (1.9) is lower than the  $Q_{10}$  for  $O_2$  uptake in substrate-saturated mitochondria (2.4) in soybean cotyledons [36]. As a result, the respiratory temperature curves for *in vivo* rates of maximum potential respiratory flux and actual  $R$  converge as the temperature decreases. Additional evidence that  $R$  is capacity limited in the cold is as follows: first, whereas respiratory uncouplers plus exogenous glucose stimulate  $O_2$  uptake in intact roots at moderate temperatures (e.g. 25°C), they have no effect on root  $R$  at low temperatures [20]. Second, glycolytic substrates (soluble sugars) often accumulate when plants are exposed to the cold [20,22,40]. Third, whereas ADP stimulates substrate-saturated  $O_2$  uptake in isolated mitochondria at moderate temperatures, ADP has no effect on mitochondrial  $O_2$  uptake in the cold in either soybean cotyledon [36] or in rat liver [41] mitochondria. Thus, even though the potential for adenylate restriction is greater in the cold because of reduced rates of ATP turnover to ADP by growth and maintenance processes [2] and decreased proton leakage through the inner mitochondrial membrane (Box 1), particularly at temperatures below the transition temperature, the available data suggests that adenylates are not the most-limiting factor in the cold. Fourth, plots of  $O_2$  uptake versus substrate concentration in isolated soybean cotyledon mitochondria show that respiratory flux is saturated by relatively low concentrations of reduced ubiquinone ( $UQ_r$ ; the common substrate for both the cytochrome and alternative pathways) (Box 1) in the cold (Fig. 4) [36]. Taken together, these findings suggest that  $R$  is capacity limited in the cold in intact plant tissues and in isolated mitochondria. Further work is needed to establish whether cold effects the activity of all enzymes equally or whether it has a greater detrimental effect on some enzymes than on others.

#### Substrate-dependent changes in the $Q_{10}$

Although it is well established that most reactions catalysed by enzymes exhibit a higher  $Q_{10}$  when substrates are saturating [40], until recently, little data was available to demonstrate the substrate dependence of the  $Q_{10}$  of  $R$  in plant tissues. The addition of exogenous glucose increases the  $Q_{10}$  of  $R$  in intact roots [20] and there is a positive relationship between the concentration of soluble sugars and the  $Q_{10}$  of  $R$  [20,42]. Moreover, the  $Q_{10}$  of  $O_2$  uptake in isolated mitochondria increases with the increasing reduction level of the ubiquinone ( $UQ$ ) pool (Fig. 4) [36]. These findings [36] can be explained as follows. Typically, the  $V_{max}$  increases with measurement temperature (up to a maximum rate); however, to take advantage of this increased potential rate, sufficient substrate and/or ADP supply must be available. If substrate availability and/or adenylates limit  $R$ , then increases in measurement temperature will have less stimulatory effect on flux (Fig. 4). A further factor contributing to the substrate dependence of the  $Q_{10}$  of  $R$  is the effect of temperature on the affinity constant ( $K_m$ ) of



**Fig. 4.** Effect of substrate availability on (a) rates of respiration ( $R$ ) measured at four temperatures (10, 15, 20 and 25°C) and (b) the  $Q_{10}$  of  $R$  calculated over two temperature ranges (between 10–15°C and 20–25°C). Substrate availability is represented by the redox state of the ubiquinone pool ( $UQ_r/UQ_t$ ). In (a), rates of total  $O_2$  uptake (i.e. cytochrome plus alternative oxidase activity) are shown for mitochondria in the presence of ADP. At low  $UQ_r/UQ_t$  values, rates of total  $O_2$  uptake are limited by substrate availability (i.e. reduced ubiquinone), regardless of the measuring temperature. In measurements made in the cold (e.g. 10°C),  $R$  becomes substrate-saturated at high  $UQ_r/UQ_t$  values (i.e.  $R$  becomes limited by enzymatic capacity at high  $UQ_r/UQ_t$  in the cold). By contrast,  $R$  is not limited by enzymatic capacity *per se* at high temperatures (e.g. 25°C). In (b),  $Q_{10}$  values were calculated at individual  $UQ_r/UQ_t$  values using the data in (a) and equation 1:

$$Q_{10} = (R_2/R_1)^{10/(T_2-T_1)} \quad [\text{Eqn 1}]$$

where  $R_2$  and  $R_1$  are rates of  $R$  measured at temperatures  $T_2$  (high temperature) and  $T_1$  (low temperature), respectively. The lines in (b) are first order linear regressions fitted to  $Q_{10}$  values at each  $UQ_r/UQ_t$  value. An additional factor that accentuates the effect of substrate availability on the  $Q_{10}$  is the stimulatory effect of high  $UQ$ -pool reduction levels on the activation state of the alternative oxidase [69], particularly at high measuring temperatures [36].

the enzymes catalysing the reaction in question (further details in Ref. [40]). This is shown by the rate of  $R = (V_{\max} \times C)/(K_m + C)$ , where  $C$  is the concentration of the available substrate (i.e. reduced UQ when considering  $O_2$  uptake, as in Fig. 4). At low substrate availability, the temperature dependence of  $R$  will reflect the quotient of the separate temperature dependencies of the  $V_{\max}$  and the  $K_m$  [40]. Thus, if the  $Q_{10}$  of the  $V_{\max}$  and the  $K_m$  of the terminal oxidases are similar (as is the case for the  $CO_2$  fixation by Rubisco in the chloroplast [40]), then changes in temperature will have little effect on  $O_2$  uptake at low substrate concentrations but will substantially increase  $R$  at higher substrate concentrations. Thus,  $Q_{10}$  values of  $O_2$  uptake (calculated over a given measurement temperature range) increase as substrate availability increases, largely as a result of temperature-dependent changes in the  $K_m$  of the terminal oxidases and  $R$  being less limited by enzymatic capacity at high measuring temperatures than in the cold (Fig. 4). The substrate concentration that is required to saturate  $R$  therefore increases with increasing temperature; consequently, the relative difference in rates of  $R$  at low and high measuring temperatures (i.e. the  $Q_{10}$ ) increases with increasing substrate availability. Taken together, these recent studies suggest that  $Q_{10}$  of plant  $R$  is indeed higher whenever respiratory flux is limited by enzymatic capacity than when  $R$  is limited by substrate supply. Conversely, transition from enzymatic control to limitations imposed by substrate supply (or adenylates) is associated with a decline in the  $Q_{10}$ .

Are all variations in  $Q_{10}$ s (measured over a common temperature range) likely to reflect concomitant differences in the availability of respiratory substrates (e.g. soluble carbohydrates)? No. Although variations in the  $Q_{10}$  of plant  $R$  are correlated with variations in substrate availability [20,36], there are published examples where variations in the  $Q_{10}$  are not linked to variations in the concentration of soluble carbohydrates [22,24,43]. Here, factors other than substrate availability alone must be responsible for variations in the  $Q_{10}$  [e.g. the extent to which respiratory flux is limited by adenylates (i.e. the ratio of ATP to ADP and/or the concentration of ADP *per se*)]. Indeed, there is evidence that variations in adenylate concentrations affect the substrate dependence of the  $Q_{10}$ . For example, in the absence of ADP, the  $Q_{10}$  of the cytochrome pathway (in isolated mitochondria) is less sensitive to changes in substrate concentration [36]. This is probably because in the absence of ADP, control over flux through the cytochrome pathway is mediated by the leak of protons across the inner mitochondrial membrane [44]. By contrast, control over flux through the cytochrome pathway is distributed among several steps in the presence of ADP, including the dehydrogenases, proton leak, the ATPase (CV), CIII (cytochrome *b/c* complex), cytochrome *c*, and CIV (cytochrome *c* oxidase) (Box 1). The  $Q_{10}$  of the proton leak is likely to be fundamentally different from temperature dependence as electrons flux via the cytochrome pathway [36].

The extent to which the substrate dependence of the  $Q_{10}$  is affected by adenylates might depend on the partitioning of electron flux between the cytochrome and alternative oxidase (AOX) pathways of electron transport. The

cytochrome pathway (terminating at CIV) couples the reduction of  $O_2$  to water with the translocation of protons across the inner mitochondrial membrane, thereby building up a proton-motive force that drives ATP synthesis when protons pass through the ATPase (CV) (Box 1). In the absence of ADP, protons cannot move through CV, with the result that electron transport via the cytochrome path becomes adenylate restricted. By contrast, electron transport from UQ to  $O_2$  via the AOX occurs without further proton translocation; consequently, respiration is less likely to be severely limited by adenylates in tissues with substantial potential AOX activity. Given that engagement of the AOX is highly variable in plant tissues (e.g. varies with age [45], growth temperature [46], phosphate supply [47], inherent relative growth rate [48], irradiance [49] and infection [50]), AOX-mediated changes in adenylate control might, in part, explain why  $Q_{10}$  values are substrate-dependent in some cases but not in others. Further experiments are needed to establish if this is the case.

To what extent does engagement of the AOX affect the  $Q_{10}$  of  $R$  (independent of the degree of adenylate restriction)? During the 1980s and the early 1990s, several authors concluded that the  $Q_{10}$  of the AOX was lower than that of the cytochrome pathway [51,52]. As a result, switching between the AOX and cytochrome pathway could affect the  $Q_{10}$  of overall  $O_2$  consumption in intact tissues. However, more recent studies have not supported this conclusion. For example, a study using the  $^{18}O$  fractionation method, found that the  $Q_{10}$  of the AOX is similar to the  $Q_{10}$  of the cytochrome pathway in mung bean (*Vigna radiata*) leaves and hypocotyls or soybean cotyledons [46]. Similarly, the  $Q_{10}$  of the AOX was not necessarily lower than that of the cytochrome pathway in soybean cotyledon mitochondria [36]. In the presence of pyruvate (which activates the AOX) [53], the  $Q_{10}$  of the AOX is 2.61 (mean  $Q_{10}$  between 10–25°C), which is similar to or greater than the cytochrome pathway. In the absence and presence of ADP, the  $Q_{10}$  of the cytochrome pathway is 2.55 and 2.40, respectively [36]. Therefore, it seems unlikely that engaging the AOX will reduce the  $Q_{10}$  of plant  $R$  *per se*.

### Long-term thermal acclimation: the importance of development

Although acclimation of  $R$  to long-term changes in temperature is widespread, the degree of acclimation is highly variable within and among plant species [23,24,28,30,54,55]. Moreover, although the degree of acclimation differs systematically among taxa in some studies [30], many results are contradictory [28,55]. In spite of such variability and contradictory results, it appears likely that the degree of acclimation is developmentally dependent because the degree of acclimation is generally lower in fully expanded, pre-existing leaves and mature roots that are exposed to a new growth temperature [20,22] than it is in leaves and roots that develop under contrasting growth temperatures [28,30,31,35,55]. Although acclimation of  $R$  to a new growth temperature can occur in pre-existing leaves and mature roots formed at the previous growth temperature [22,55], maximal

acclimation appears to require that new leaves and roots be formed [55]. Studies on photosynthetic acclimation to temperature have shown that newly developed leaves possess the chemical composition and enzymatic machinery to acclimate to a higher degree than fully expanded, pre-existing leaves exposed to a new growth temperature [18]. Recently, we have found that acclimation of  $R$  to 5°C is also substantially greater in *Arabidopsis* leaves that develop at 5°C than in warm-grown leaves shifted to 5°C for several days (A. Armstrong and O.K. Atkin, unpublished). Development of new leaves and roots following a change in growth temperature is therefore a likely prerequisite for maximal acclimation of  $R$ . In terms of plant response to climate change, it is the long-term acclimation response and perhaps the role of temperature during development that will ultimately govern the respiratory fluxes with climate warming.

### Distinguishing between two types of acclimation

Long-term acclimation of  $R$  to temperature might occur via altered temperature sensitivity (i.e. the slope or  $Q_{10}$ ) and/or via a shift in the overall intercept (elevation) of the temperature-response function. We term a temperature acclimation response of  $R$  that is characterized predominantly by a change in  $Q_{10}$  as 'Type I acclimation' [20], whereas an acclimation response that is characterized by a change in intercept as 'Type II acclimation' [30,31] (Fig. 3). Although Type I acclimation does not result in as high a degree of acclimation as Type II acclimation (Fig. 3), it does enable the temperature response of  $R$  to adjust dynamically to changes in the ambient temperature of the growing environment. Recent findings indicate that a change in the  $Q_{10}$  of the short-term temperature response of  $R$  over a broad range of measurement temperatures (Type I acclimation) might be the more common mode of acclimation in fully developed, mature tissues (although further work is needed to test this hypothesis). For example, Type I acclimation was exhibited by fully expanded leaves of *Eucalyptus pauciflora* experiencing seasonal changes in temperature [22] and warm-developed roots shifted to a lower growth temperature for several days [20]. Underpinning the change in  $Q_{10}$  (Type I acclimation) are changes in  $R$  measured at moderate-high temperatures, with little or no change in  $R$  in the cold (because there is no measurable change in respiratory capacity [20]). Changes in  $R$  at moderate-high temperatures probably reflect, in part, changes in the availability of respiratory substrate and/or degree of adenylate restriction of  $R$ . Plants shifted from one temperature to another often exhibit large changes in the concentration of soluble sugars [18,20,22], in addition to changes in the  $Q_{10}$  of  $R$  [20,42]. Changes in the membrane fluidity (as a result of changes in the unsaturated fatty acid content of membranes [40]) following several days of exposure to a new growth temperature might also play a role, via their effects on adenylate control (as mediated by changes in proton leakage across the inner mitochondrial membrane) and substrate transport across membranes. Growth-temperature dependent changes in PUMP [56,57] are also likely to contribute to changes in the degree of adenylate restriction of  $R$ . The extent to which maintenance processes

(e.g. protein turnover and maintenance of cellular ion-gradients [2]) acclimate to several days' exposure to a new growth temperature could also contribute to changes in adenylate restriction of  $R$  (via changes in demand for ATP) and the  $Q_{10}$  (and thus Type I acclimation). Further experiments are needed to assess the extent to which Type I acclimation is common among plant species, and the extent to which it is linked to substrate-dependent versus adenylate-dependent changes in the  $Q_{10}$  of  $R$ .

In contrast to its Type I counterpart (Fig. 3a), Type II acclimation (Fig. 3b) is associated with a change in rates of  $R$  at high and low measuring temperatures, with rates of  $R$ , both at low (e.g. 0–5°C) and moderately high (e.g. 20–25°C) temperatures, being lower in warm-acclimated plants than their cold-acclimated counterparts. Although the mechanisms responsible for Type II acclimation have not been fully elucidated, it seems likely that changes in respiratory capacity are responsible for the differences in  $R$  at low measuring temperatures exhibited by warm and cold-developed leaves and roots (either via differences in capacity per mitochondrion [58] or differences in the number of mitochondria per unit area [59]). In recent years, several studies have shown that development at low temperatures is also associated with an increase in AOX (Box 1) [46,60] and PUMP levels [56,57]. Cold-grown plants also often exhibit greater leaf and whole-plant nitrogen concentrations [31] in addition to higher rates of  $R$ .  $R$  is often correlated with nitrogen concentrations in plant tissues [61–64]. Seasonal variation in  $R$  in leaves and roots might be, in part, related to changes in nitrogen and/or carbohydrates [21,65]. Thus, it seems likely that Type II acclimation is largely associated with temperature-mediated changes in respiratory capacity that can be maximally realized through growth of new tissues. It also seems likely that there will be a link between the extent to which processes supported by respiration (e.g. growth and maintenance) acclimate and the higher degree of acclimation in newly formed tissues [2], with demand for respiratory energy recovering once new tissues form at the new growth temperature. Yet more studies are needed to clarify the extent to which Type I and Type II acclimation occur. For example, the seasonal acclimation response of  $R$  in leaves among evergreen and deciduous plant species in temperate climates might reveal an important contrast. Furthermore, the extent to which root  $R$  acclimates to long-term changes in temperatures in concert with aboveground  $R$  is not known and controversial at least for some species [64,65].

### Conclusions and future prospects

This review has focused on the extent to which the short- and long-term temperature sensitivity of  $R$  varies among plants and the underlying mechanisms responsible for that variability. We have attempted to provide a mechanistic explanation for why  $Q_{10}$  values vary (Fig. 3). The near-linear temperature dependence of the  $Q_{10}$  across plant taxa and biomes probably reflect predictable shifts in the control exerted by maximum enzyme activity at low temperature and substrate limitations at high temperature. In general,  $Q_{10}$  values are lower in tissues where respiratory flux is limited by substrate availability; as a

result, we suggest that overall  $Q_{10}$  values will be lower in plants where photosynthesis and subsequent synthesis of sugars is limited (e.g. by drought and/or shade). Conversely, increases in the availability of respiratory substrate that result from sustained higher rates of photosynthesis (e.g. under elevated  $CO_2$ ) might result in an increase in the  $Q_{10}$  of  $R$ .

We have identified two types of acclimation, one of which is underpinned by adjustments in the  $Q_{10}$  (Type I) and the other by changes in the enzymatic capacity of the respiratory system (Type II). Although more limited in its effect on the magnitude of acclimation than its Type II counterpart is, Type I acclimation enables rapid changes in respiratory flux at high temperatures to occur following changes in thermal environment. Type II acclimation is likely to be maximal upon the development of new leaves and roots following a change in temperature. This leads to the prediction that plant species that produce long-lived leaves and roots and that are relatively slow at generating new tissues should, therefore, exhibit a relatively limited ability to acclimate to long-term changes in temperature. This might prove crucial if global temperatures increase rapidly in the coming decades, as predicted.

However, much remains unknown. Although available data suggests that acclimation will be greater in well-watered habitats than in areas experiencing drought [23], little is known about the interactive effects of irradiance, nutrient supply and water availability on acclimation of  $R$ . The mechanisms responsible for changes in respiratory capacity need to be established, as do the linkages between respiratory acclimation and how other processes (e.g. growth, maintenance and active transport) respond to temperature change. Establishing the signal(s) that lead to growth-temperature mediated changes in respiratory capacity is an intriguing area for future research; for example, acclimation might occur in response to systemic signals from remote parts of the plant, rather than through the tissue experiencing the temperature directly. Systemic signaling pathways that enable plants to respond to abiotic factors sensed in remote parts of the plant (e.g. light and atmospheric  $CO_2$  concentration) have recently been identified [66]. The role of temperature acclimation in mediating the balance between respiratory and photosynthetic carbon fluxes requires further experimental work [67]. At organismal scales, much is still not known about the extent to which  $Q_{10}$  values and acclimation vary systematically within and among plant species. Our review suggests that variations in  $Q_{10}$  values might be predictable (e.g. temperature dependence is consistent and any condition that results in substrate depletion might result in a decline in the  $Q_{10}$ ). The challenge ahead will be to identify commonly measured plant functional traits that will enable  $Q_{10}$  values and degrees of acclimation to be predicted. If they can, this will enable modellers to incorporate acclimation scenarios into models more easily.

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