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Review

'Breathing Out' under Heat Stress—Respiratory Control of Crop Yield under High Temperature

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Abstract: Respiration and photosynthesis are indispensable plant metabolic processes that are affected by elevated temperatures leading to disruption of the carbon economy of the plants. Increasing global temperatures impose yield penalties in major staple crops that are attributed to increased respiratory carbon loss, through higher maintenance respiration resulting in a shortage of non-structural carbohydrates and an increase in metabolic processes like protein turnover and maintenance of ion concentration gradients. At a cellular level, warmer temperatures lead to mitochondrial swelling as well as downregulation of respiration by increasing the adenosine triphosphate:adenosine diphosphate (ATP:ADP) ratio, the abscisic acid-mediated reduction in ATP transfer to the cytosol, and the disturbance in a concentration gradient of tricarboxylic acid (TCA) cycle intermediates, as well as increasing lipid peroxidation in mitochondrial membranes and cytochrome c release to trigger programmed cell death. In this review, we discuss the mechanistic insight into the heat stress-induced mitochondrial dysfunction that controls dark respiration in plants. Furthermore, the role of hormones in regulating the network of processes that are involved in retrograde signaling is highlighted. We also propose different strategies to reduce carbon loss under high temperature, e.g., selecting genotypes with low respiration rates and using genome editing tools to target the carbon-consuming pathways by replacing, relocating, or rescheduling the metabolic activities.

Keywords: acclimation; alternative oxidase; heat stress; mitochondria; maintenance; respiration

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1. Introduction

The rising temperature is an intrinsic component of global climate change that controls the carbon fluxes in all the crops. High temperature affects the major plant physiological processes, such as photosynthesis and respiration; therefore, it becomes important to estimate the plant carbon dioxide (CO_2) balance that finally decides the crop productivity [1–3].Through these two pathways, the terrestrial ecosystems exchange about 120 Gt of carbon per year with the atmosphere [4]. A rough estimate states that half of the CO_2 assimilated annually through photosynthesis is released back to the atmosphere by plant respiration [5–7], and merely 15–25% of the fixed carbon finally translates into yield [8,9]. The projected elevation in temperature beyond 2.0 °C by the end of the decade [10] may increase the magnitude of carbon loss exponentially in the physiological temperature range of 0 to 38 °C [11], which will further exacerbate in a species-and environment-dependent manner at higher temperatures between 48 and 60 °C [12–15].

The carbon lost through the 'breathing out' processes in plants can occur via two mechanisms, namely photorespiration and dark/mitochondrial respiration. These processes release CO_2, but dark respiration occurs regardless of light in the plant cells [16,17]. Biochemically, dark respiration is an enzymatically regulated, multistep, amphibolic process that produces ATP by the oxidation of glucose formed during photosynthesis. Glucose is initially broken into pyruvate during glycolysis, which is oxidized to form acetyl-CoA, releasing a molecule of CO_2. The acetyl-CoA then enters the tricarboxylic acid (TCA) cycle, where it is oxidized to CO_2 and also produces reductants (nicotinamide adenine dinucleotide: NADH; dihydroflavine-adenine dinucleotide: $FADH_2$) that pass through the mitochondrial electron transport chain (ETC). The oxidation of the reductants produces a proton gradient across the inner membrane of the mitochondria that drives the synthesis of ATP. High temperatures impact dark respiration in plants with an exponential increase [18], which can become detrimental due to irreversible damage to the enzymatic machinery [15]. Climate change prediction models have speculated a 3–20% decline in the yield of major crops like wheat, rice, maize, and soybean with every 1 °C increase in the global mean temperatures [19,20], which makes it pertinent to relate this loss to the waste of carbon due to respiration. The contribution of dark respiration in limiting the productivity of crops under elevated temperatures has not been extensively reviewed, in comparison to photorespiration. Therefore, our present review discusses the heat-induced alterations in dark respiration in plants and proposes strategies to reduce the carbon loss under the inevitable reality of a changing climate.

2. Respiratory Carbon Loss-A Constraint to Crop Yield

Respiration, rather than photosynthesis, may be the primary contributor to yield losses in a high temperature climate [11]. Low respiration rates are generally correlated with high crop yields [21,22]. Walker et al. [23] reported that photorespiration decreased soybean and wheat yields by 36% and 20%, respectively, in the United States. In another study, a 10–12% and 17–35% decrease in the yields of wheat and rice, respectively, was reported due to high temperatures [24]. The yield loss in wheat and rice due to high night temperature (HNT) is mainly ascribed to higher dark respiration, which increases the consumption of photoassimilates, thereby resulting in the reduction of non-structural carbohydrates (NSCs) in stem tissues [25,26]. Glaubitz et al. [27] reported that increasing night temperature from 25 °C to 35 °C resulted in increased leaf respiratory carbon losses in grapevines, as reflected by the decrease in NSCs of 0.025 and 0.041 mg g^{-1}dry weight, respectively. Such losses are consistent with metabolite profiling studies in wheat and rice, which revealed an increase in TCA cycle intermediates in leaves exposed to HNT, supporting increased respiration in the photosynthesizing tissue [25,28]. Xu et al. [29] suggest that increased dark respiration restrains source availability under the combined stress of high day and night temperatures, leading to a considerably more severe yield penalty due to carbon loss.

3. Heat-Induced Changes in the Proportion of Maintenance Respiration

Dark respiration (R_d) is typically partitioned into two functional components, i.e., growth respiration (R_g) and maintenance respiration (R_m), which are impacted upon by environmental stresses [9,30,31]. Figure 1 illustrates the differences in these components under elevated temperatures. Growth respiration is a dominant component of respiration in younger tissues, while the latter contributes majorly to the older tissues [32]. Growth respiration is defined as the amount of photoassimilates respired to provide energy for the synthesis of additional biomass [33]. It also provides a carbon skeleton and reductants to facilitate nutrient uptake/assimilation followed by biosynthesis of cellular components to drive the growth of tissues. Thus, the relationship between the growth rates of a species and temperature is actually a measure of the rate of the growth respiration component [34]. A recent analysis of 101 evergreen species growing in different biomes (boreal to tropical) showed that respiration increased with an increase in growth temperatures in accordance with previous studies [35,36]. Leaf form accounted for the response ratio of R_g to warming,

as species with needle-like leaves had a significantly higher response (25 ± 9%) than broad-leaved ones [36].

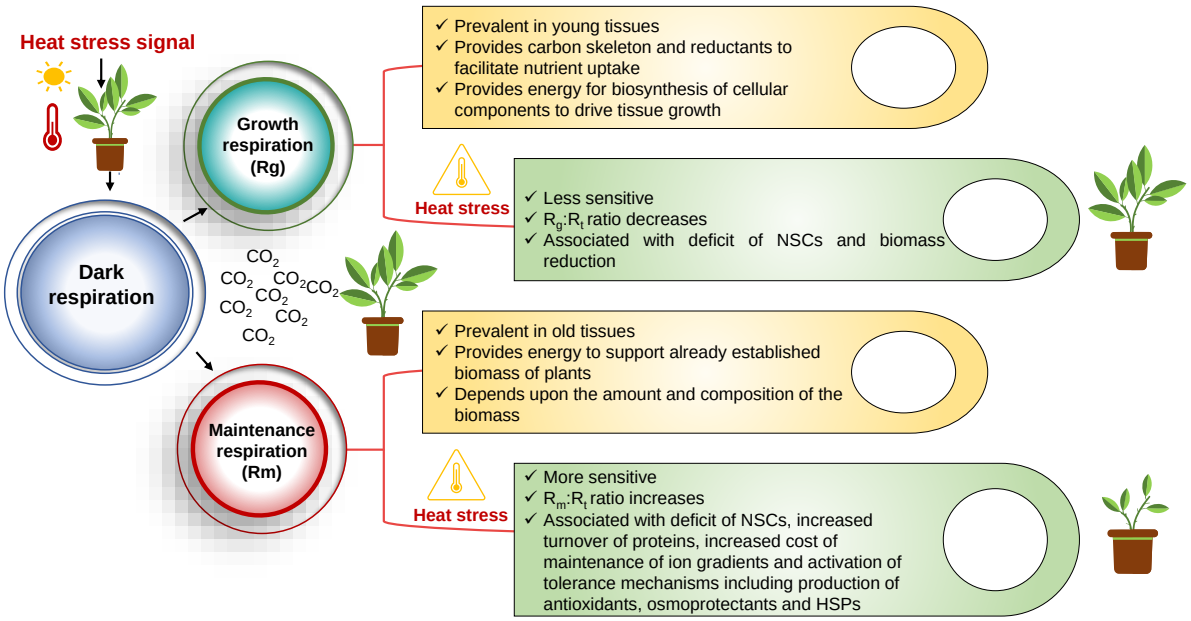

Figure 1. Growth respiration and maintenance respiration under elevated environmental temperature. HSPs: heat shock proteins; NSCs: non-structural carbohydrates; R_g: growth respiration; R_m: maintenance respiration; R_t: total respiration.

On the other hand, maintenance respiration comprises the respiratory processes that help in supporting the already established biomass of the plant [33]. It depends upon the amount and composition of the biomass, as both these factors undergo change depending on the environment and developmental stage of the plant. Although the role of both the components is integral to the life cycle of the plants, their estimation can only be done by employing physiological models [32,37–39]. The higher temperature responsiveness of R_m over R_g in mature tissues was concluded from various studies, e.g., Marigolds when exposed to a 10 °C increase in temperature resulted in a 43% to 55% increase in the proportion of maintenance respiration to total respiration (R_t) [40]. Additionally, a significant reduction in ATP content and total biomass was observed in rice plants subjected to 10 °C higher temperature at the reproductive stage than the ambient temperature (28 °C), thereby suggesting that energy produced by respiration under high temperature conditions was mainly attributed to maintenance respiration rather than growth respiration [32]. Mathematically, maintenance respiration is expressed as the product of maintenance respiration coefficient and plant size. The Q_{10} value (proportional increase in rate of respiration with a 10 °C rise in temperature) of the maintenance respiration coefficient varies between 1.35 and 3.0 depending upon the species, developmental stage, and environmental conditions as shown in the data compiled from various studies (Table 1). The sensitivity of Q_{10} to temperature indicates that the response of respiration to temperature cannot be represented by one value.

Table 1. Q_{10} values for maintenance respiration coefficient in various crops.

Crop	Experimental Temperature	Q_{10} Value	Reference
Marigold (*Tagetes patula*)	20 °C (Control) 30 °C (Elevated)	1.35–1.55	[40]
Barley (*Hordeum vulgare*)	15 °C (Control) 28 °C (Elevated)	3.00	[41]
Subterranean clover (*Trifolium subterraneum*)	10 °C (Control) 35 °C (Elevated)	1.85	[42]
Japanese knotweed (*Reynoutria japonica*)	15 °C (Control) 25 °C (Elevated)	1.90	[43]
Wheat (*Triticum aestivum*)	15 °C (Control) 20 °C (Elevated)	1.80	[44]
	10 °C (Control-Night temperature) 21 °C (Elevated-Night temperature)	1.97	[45]

4. Substrate Availability for Respiration under High Temperature

The considerable variation observed in the Q_{10}–temperature relationship is influenced by the supply of the respiratory substrate and the respiration capacity [4,46]. Environmental variables that affect the biosynthesis of the substrates [18,46] or increase the metabolism of energy consuming processes like turnover of proteins and maintenance of ion gradients [47], make Q_{10} values highly dynamic in response to temperature. Additional energy costs are incurred by mechanisms imparting heat tolerance in the crops, e.g., upregulation of the antioxidant defense system to counteract the upsurge in the level of reactive oxygen species (ROS), synthesis of osmoprotectants, and accretion of heat shock proteins (HSPs). The need for respiratory substrate in the plants is mainly met from the non-structural carbohydrates [25–27,48] and the protein turnover [11,32]. Studies on the effect of elevated night temperatures have shown that the high rate of nighttime respiration exerted pressure on the supply of NSCs, which subsequently reduced the biomass and yield of rice [25,26]. The concentration of sugars has been positively correlated with the rate of dark respiration in *Pinus* [49], *Quercus rubra* [50], and *Spinacia oleracea* [51]. The light control of carbohydrate synthesis affected the rate of dark respiration in *Geum urbanum* plants grown under 75% shade as it declined due to limited photosynthate supply, but Q_{10} declined only when the leaves experienced near darkness for long periods. It was concluded that intense shade for a prolonged period would cause a reduction in both respiration and Q_{10} due to adenylate restriction on respiration in addition to the substrate availability [4].

5. Regulation of Respiratory Flux at High Temperature

Adenylates (in particular the ratio of ATP to ADP and the concentration of ADP per se), are likely the most important in regulating respiratory flux at warm temperatures [52]. Adenylate control would indicate that the respiratory capacity at warmer temperatures exceeded the level required for cell processes to proceed [18], which in turn would lead to elevated ATP:ADP ratios or low ADP concentration, causing downregulation of respiration [53]. The increased leakiness of membranes at high temperatures could further contribute to substrate limitation because concentration gradients of TCA cycle intermediates are more difficult to maintain when mitochondrial membranes are excessively fluid [18].

6. Positive Correlation between Protein Turnover Cost and Respiratory Cost at High Temperature

Nitrogen (N) utilization processes, including nitrate reduction and ammonium assimilation, are thought to have high respiratory costs [54]. In fact, the estimates of construction

respiration are greatly influenced by the form of N source, e.g., nitrate or ammonium [55]. The protein turnover rate increases with temperature, suggesting that the protein turnover cost is a major component of the N-utilization cost and dominates during maintenance respiration. Hachiya et al. [56] studied the protein turnover cost in *Petunia x hybrida* petals grown at three different temperatures (20, 25,and 35 °C) during the development of the petals. Most petals are non-photosynthetic; therefore, ATP and reducing equivalents are supplied mainly from the respiratory pathway. The integrated protein turnover cost on dry weight basis was similar between 20 and 25 °C but increased by more than four times at 35 °C, suggesting that the high temperature enhanced the cost of protein turnover, thereby increasing the total cost of N-utilization along with respiration in the petals.

7. Diurnal Dynamics of Respiration

The diurnal or diel cycle of plant growth interacts with the respiratory metabolism, which can be directly linked with the availability of respiratory metabolites regulating the process at different times of the day [57]. The photosynthate synthesized during the day supports carbon supply for the entire plant during the day, which is reduced to critical levels by the end of the night [58]. The strong coupling between carbon fixation through photosynthesis and loss due to respiration [59] indicates the diurnal fluctuation in rates of dark respiration as a result of changes in the concentration of various metabolites supporting the respiratory process [60]. In this case, the supply of sugars is stabilized over the day–night cycle, and the diel variation in respiration may be explained by changes in the availability of amino acids, proteins, organic acids, and/or lipids. These metabolites may drive respiration by supplying intermediates to the TCA cycle, reductants for ATP synthesis via oxidative phosphorylation, and carbon skeletons required for biosynthesis or nitrogen assimilation into amino acids [61].

Metabolomic studies have shown that warmer day (30 °C) and night (28 °C) temperatures lead to the accumulation of amino acids derived from shikimate pathways, such as phenylalanine, tyrosine, tryptophan, aspartic acid, lysine, proline, and γ-amino butyrate (GABA), in thermo-sensitive rice cultivars (DR2 and M202) but not in intermediate (IR64 and IRRI123) and temperature-tolerant cultivars (IR72 and Taipei 309) [28]. Similarly, in wheat, high night temperatures showed a prevalence of fumarate and alanine without any significant change in the level of glutamine, glutamate, and GABA [25]. The accumulation of TCA intermediates like malate and fumarate during the day, and citrate, aconitate, and succinate during the night [62,63], reiterates the circadian control of the TCA pathway, which is a hub for the process of respiration and can be markedly influenced by an increase in temperature [64]. Rashid et al. [64] assessed the influence of growth temperature and the diel cycle on the concentrations of metabolites involved in the respiratory network of rice. They raised the plants under 25 °C:20 °C, 30 °C:25 °C, and 40 °C:35 °C day:night cycles and measured the dark respiration and changes in metabolites at five time points spanning a single 24 h period and observed that shikimate pathway-derived aromatic amino acids were the only metabolites to interact in response to both the growth temperature and the day:night cycle. Cook et al. [65] reported increased concentrations of α-ketoglutarate, fumarate, malate, and citrate in *Arabidopsis* leaves when cooled from 20 °C to 4 °C. All these studies suggest that there are distinct respiratory metabolite adjustments to temperature and the diel cycle.Further, detailed experiments on the interaction of the diel cycle and temperature will generate a better understanding of the metabolites controlling dark respiration in plants. Therefore, the instantaneous measurement of respiration rates at a single point during the day can overlook the differential response prevalent during an extended period.

8. Thermal Acclimation of the Respiration Response in Plants under Heat Stress

Short periods of high temperatures show an exponential increase in dark respiration [66,67], whereas prolonged exposure can result in thermal acclimation of the respiration response to lessen the impact of continued carbon loss due to increasing tem-

peratures [31,68]. Under thermal acclimation, the tissues that develop under the new temperature show a better homeostatic response to respiration than the ones formed before the acclimation temperature [31,66,69]. There are two types of thermal acclimation responses [18] that occur across plant types and biomes (Figure 2):(i) Type I acclimation, where warm acclimated leaves show lower short-term sensitivity to temperature, and the regulation by the existing respiratory enzymes causes a reduction in Q_{10} [46].(ii) Type II acclimation, which involves a change in the respiratory capacity due to change in the concentration of the respiratory enzymes or mitochondrial proteins, resulting in lower respiration across the temperature range and no change in Q_{10}. Type I acclimation is less efficient and occurs in leaves that mature prior to the temperature change. In contrast, Type IIis common in leaves that are formed later under higher temperatures with a high degree of homeostasis. The advantage of Type II acclimation is that it allows the plant to make both the physiological and developmental adjustments in the size and density of mitochondria [70], whereas Type I, which solely influences the physiological plasticity to temperature. Based on this fact, it was found that boreal evergreen tree species, which grow under changing temperatures, are more efficient in acclimation during their lifetime than deciduous species that seasonally shed their leaves [71]. A recent meta-analysis by Crous et al. [36] highlighted the differential respiration response across various biogeographical regions and leaf forms and found that the leaves of gymnosperms showed a 30–40% reduction in respiration rates at a common temperature of 25 °C compared to broadleaved evergreens at >10 °C warming.

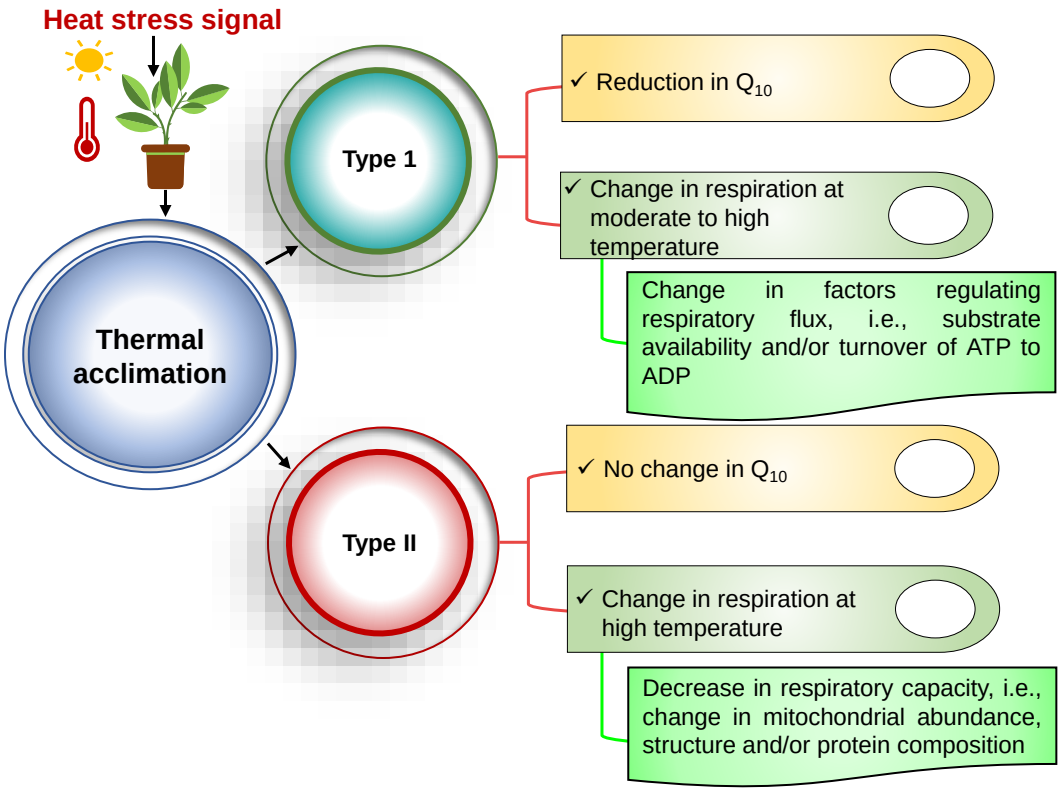

Figure 2. Types of thermal acclimation in plants in response to heat stress.

The dynamicity in the size, number, and signaling responses of mitochondria can cause a collective outcome during the acclimation response to meet the demand for metabolic energy, carbon skeleton, and reductants [72], and it is controlled by a network of genes [73]. The inability to acclimate is the consequence of mitochondrial disorganization under high temperatures that increase the leakiness of mitochondrial membrane and lipid peroxida-

tion [74] along with the disruption of the TCA cycle, mitochondrial NADH pool, and ATP synthesis [75].

9. Mitochondrial Physiology under High Temperature

Temperature stress exerts a thermodynamic influence on the subcellular structures and intracellular macromolecules in the plant cells [76]. Since mitochondria maintain the energy requirements of the cells, they become the primary targets for structural and functional changes under stress [77], as illustrated in Figure 3. The phospholipid, cardiolipin (CL), is an important constituent of the inner mitochondrial membrane and contributes approximately 10% toward the total lipid content of the mitochondria [78]. The loss-of-function mutants of cardiolipin synthase (*cls*), involved in the synthesis of CL, confirmed its role in morphogenesis of mitochondria during heat stress in *Arabidopsis* via stabilizing the protein complex of mitochondrial fission factor DYNAMIN-RELATED PROTEIN 3 [79]. Additionally, CL is rich in polyunsaturated fatty acids and more vulnerable to lipid peroxidation [80] by the excess ROS produced during high temperature stress. The damaged CL increases the pore formation capacity of the membrane, resulting in the dephosphorylation of the mobile electron carrier cytochrome c and its release from the inner membrane towards the cytosol [81,82]. This reduces the cytochrome c activity and ATP synthesis and triggers the programmed cell death (PCD) response under stress [83,84]. A significant association has also been explained between the release of cytochrome c and Ca^{2+} dynamics during heat stress [85]. Complex I, II, and III are known to be the important sites for the production of ROS in the mitochondrial respiratory chain [86,87]. Increased ROS production is positively correlated to hyperpolarization of the mitochondrial membrane in cultured wheat cells under heat treatment. The depolarization of the membrane using the protonophore CCCP (carbonyl cyanide *m*-chlorophenylhydrazone) inhibited ROS production and oxidative phosphorylation [88]. High temperature-induced ROS production increases the cytosolic concentration of calcium that eventually finds its entry into the mitochondria and other organelles [89]. Amongst the various channels and transporters, the Ca^{2+}, voltage-dependent anion channels (VDAC) in the outer mitochondrial membrane, and the mitochondrial calcium uniporter complex (MCUC) in the inner mitochondrial membrane are involved in Ca^{2+} influx into the mitochondria [90]. The influx of Ca^{2+} through VDAC is free, while MCUC are pore-forming proteins that regulate the entry of Ca^{2+} into the mitochondria. Though transient changes in Ca^{2+} levels are detected in the mitochondria under a stressed environment, knowledge of the Ca^{2+} sensors still remains obscure.

During the stress response, ROS generation in the mitochondria communicates the signal to the nucleus through the mitochondrial retrograde signaling pathway. Retrograde signaling operates in the organelles like mitochondria and chloroplast when the organelles signal to the nucleus about its dysfunction in order to activate certain genes to carry the adaptive response [91]. The nuclear-encoded upregulation of alternative oxidase (*AOX1*) is the most prominent gene involved in the mitochondrial retrograde signaling pathway. It is a cyanide insensitive terminal oxidase in ETC that, along with alternative NADH dehydrogenases, does not generate a proton motive force that is required to produce ATP [92]. The impairment of the cytochrome pathway during stress makes the re-routing of electrons through the alternative respiratory pathway necessary as it reduces the accumulation of ROS [93,94].

In addition to the secondary messengers and metabolites discussed above, mitochondrial biogenesis and function are also controlled by plant hormones like abscisic acid (ABA), auxin (AUX), cytokinin (CK), jasmonic acid (JA), and salicylic (SA). Other hormones like brassinosteroid (BS), ethylene (ET), and gibberellic acid (GA) play a minor role in the signaling network [95].

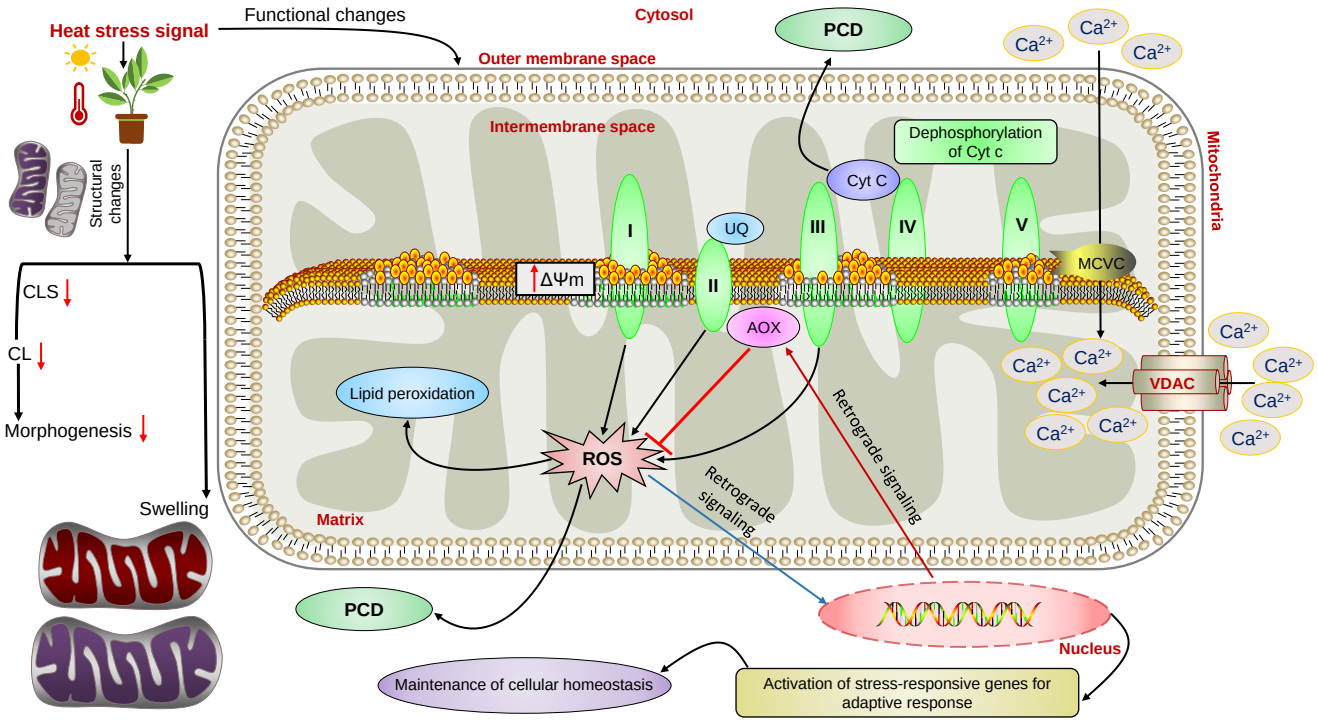

Figure 3. Structural and functional changes in mitochondria under heat stress. Cardiolipin (CL) involved in mitochondrial morphogenesis is reduced due to the low activity of cardiolipin synthase (CLS) under heat stress. The damaged CL results in depolarization of Cyt c ultimately leading to its release from the inner mitochondrial membrane into the cytosol and triggering PCD. ROS are generated at complexes I, II, and III. Under heat stress, overproduction of ROS in the inner mitochondrial membrane causes lipid peroxidation of phospholipids. ROS overproduction increases the cytosolic Ca^{2+} and influx into mitochondria *via* voltage-dependent anion channels (VDAC) in the outer mitochondrial membrane and the mitochondrial calcium uniporter complex (MCUC) in the inner mitochondrial membrane. ROS can communicate signals to the nucleus through retrograde signaling to activate genes for an adaptive response to maintain cellular homeostasis. *AOX1* is also upregulated *via* retrograde signaling, which ultimately inhibits ROS production and helps in maintaining cellular homeostasis.

10. Hormonal Regulation of Respiratory Metabolism under High Temperature

Heat stress alters the hormonal biosynthesis, stability, compartmentalization, and homeostasis within the plants [96]. The accumulation of hormones like ABA, ET, SA, CK, and JA may directly interact with mitochondrial functions in plants [97–100]. High concentration of SA interacts with mitochondrial ETC complexes I and III, whereas lower concentrations are observed as uncoupling agents [101,102]. SA oxidizes the ubiquinone (UQ) pool by altering the kinetics of dehydrogenases [103,104] and blocking the electron transport between succinate and UQ. Further, SA can also directly bind to the subunit of α-ketoglutarate dehydrogenase E2 (α-KGDH), an important enzyme of the TCA cycle, and act upstream to affect ETC during pathogen resistance to tobacco mosaic virus [105]. However, its role in abiotic stress tolerance has not been elucidated so far. Cytokinins like 6-benzylaminopurine, 6-(Δ^2-isopentenylamino) purine, and 6-furfuryl aminopurine target the mitochondrial respiration by restricting the electron transport from NADH to the cytochrome system in the stems of pea and hypocotyls of mung bean [106]. Cytokinin-like effects exhibited by N-(2-chloropyridyl)-N′-phenylurea inhibited the oxidation of malate, succinate, and NADH by the intact mitochondria of pea [107]. The respiratory control by AUXs was supported by previous studies, where decreased AUX levels and transport inhibited the functioning of mitochondrial respiratory chain complexes [108]. The

mechanistic link between AUX signaling and perturbation in mitochondria was inferred by employing the potent inducers of UDP–glucosyl transfer as encoded by the gene *UGT74E2,* which evoked a common response in mitochondrial dysfunction and inhibition of auxin-related transcription in the meristematic tissues during stress response [109].

ABA can hinder ATP/ADP exchange by the mitochondrial adenine nucleotide translocators (ANTs) to cause a reduction in ATP transfer to the cytosol or renewal of ADP to the mitochondria [95]. Consequently, the reduced availability of ADP results in the activation of ROS production in the mitochondria, with detrimental consequences, as discussed earlier [110]. The transcription factor abscisic acid insensitive 4 (ABI 4) has been reported to be a repressor of the mitochondrial *AOX1* gene of *A. thaliana*. However, *AOX* expression was stimulated by the application of ABA; therefore, the repressor effect of ABI4 on AOX1a is likely to be a part of the complex regulatory circuit [92]. Moreover, altered expression of genes like ABA hypersensitive germination 11 (*AHG 11)* involved in the editing of NADH dehydrogenase subunit 4 (*NAD4*) [111], slow growth 2 (*SLO2)* [112] involved in editing three complex I genes, ABA overly sensitive 6 (*ABO6*) [113] involved in the splicing of complex I genes and lovastatin insensitive 1 (*LOI 1)* involved in the RNA editing of cytochrome c maturation [114], were associated with altered ABA responses.

The information regarding the regulation of mitochondrial function by ET during stress is lacking. Nevertheless, the increase in AOX activity has been simultaneously related to ET biosynthesis during ripening in climacteric fruits, and its blockage leads to the inhibition of respiratory increase [115]. The implication of crosstalk between ET and AOX during stress response may help in deciphering a connection that may probably exist with ROS inhibition during retrograde signaling. Brassinolide also induces increased AOX activity in tobacco by directly affecting the promoter of the *AOX* gene [116]. Evidence linking the network of pathways that are impacted by the high temperature-induced mitochondrial dysfunction needs to be strengthened to understand the checkpoints that finally determine the respiratory control of productivity.

11. Strategies to Reduce Carbon Loss

Based on the literature, we propose the following strategies that can help in reducing the loss at the cellular and plant level.

11.1. Selection of Genotypes with Low Rates of Respiration under High Night Temperature

Studies relating to the increase in carbon loss due to high night temperatures [11,117–119] emphasize the need to screen genotypes that maintain normal respiration rates under different environmental regimes. Rice plants grown under high minimum temperatures have generated data to show that cultivars like Nagina 22 [119], Abhishek, SahbhagiDhan, and Bakal [120], with insignificant changes in post-flowering respiration, showed a marginal reduction in grain yield [117]. The biodiversity existing for this trait in the wild or related crop species can be used for introgression in high yielding cultivars of various crops with the target of generating a positive carbon balance under future climate change scenarios.

11.2. Genome Editing to Target the Metabolic Processes Consuming Carbon

The genetic improvement of crops by using genome editing approaches like knockout, replacement base editing, and regulation of expression of desirable/undesirable genes can effectively target the metabolic processes that lead to futile carbon loss in crops. The few pathways that can be replaced, relocated, or rescheduled through this approach have been discussed (Figure 4).

11.2.1. Substitution of the Lignin Biosynthesis Pathway

The lignin biosynthesis pathway involving phenylalanine ammonia lyase (PAL) can be overridden by substitution with tyrosine ammonia lyase (TAL) as it provides a gain due to the formation of two NADPH per *p*-coumarate [121] and can potentially decrease growth respiration [32].

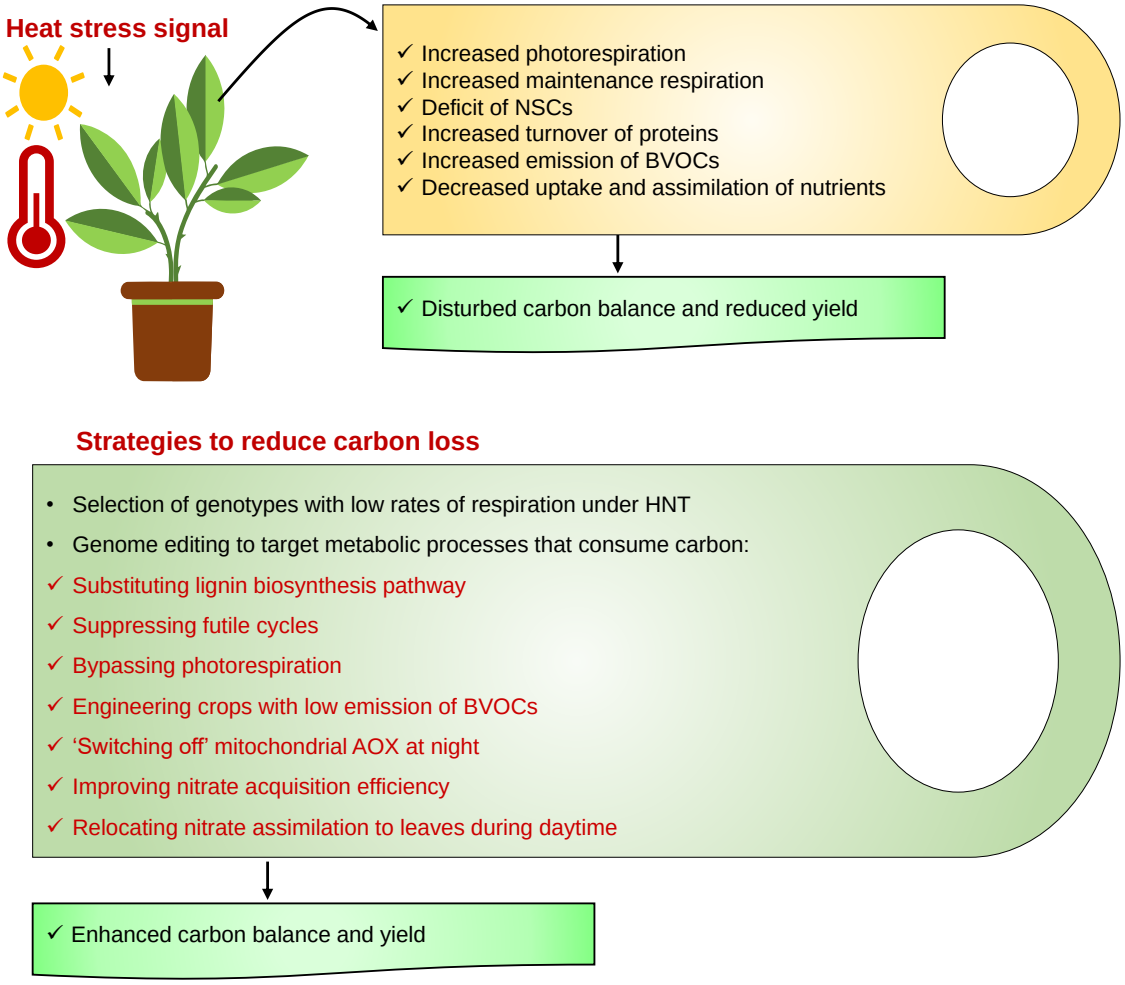

Figure 4. Respiratory carbon loss in plants and strategies to enhance yield under high temperature.

11.2.2. Suppression of Futile Cycles

A substantial proportion of the ATP generated during respiration is consumed by certain pathways that can be called 'futile' cycles. For example, the simultaneous synthesis and degradation of starch in leaves during the day [122], the simultaneous synthesis and degradation of sucrose [123], and cycling between fructose 6-phosphate and fructose 1,6-bisphosphate [124] are futile cycles. The suppression of these futile cycles will decrease the respiratory costs without exerting collateral damage on the metabolic machinery [32].

11.2.3. Designing Carbon Conserving Photorespiration

Photorespiratory bypass to eliminate the loss of CO_2 can be designed by incorporating synthetic routes through metabolic engineering. The reduction of glycolate to glycolaldehyde is a promising approach as it can assimilate 2-phosphoglycolate into the Calvin cycle without the loss of carbon. Screening the germplasm for highly stable and substrate-specific enzymes, such as acetyl-CoA synthetase and propionyl-CoA reductase, would help in favoring the reduction process over oxidation and generating a carbon-conserving pathway [125].

11.2.4. Engineering for Low Emission of Biogenic Volatile Organic Compounds

Plants release a considerable fraction of the assimilated carbon as biogenic volatile organic compounds (BVOCs). Temperature within the range of 20–40 °C has a strong influence on the activity of enzymes involved in the biosynthesis of BVOCs like isoperenes, monoterpenes, acetaldehyde, and (E)-2-hexenal. Though BVOCs impart thermal tolerance

at high temperature [126,127], it happens at the cost of 10% of fixed carbon loss. Engineering cultivars with reduced emissions of BVOCs can act as a promising strategy to save carbon under high temperatures.

11.2.5. "Switching Off" Mitochondrial AOX at Night

The AOX pathway continues to remain operative at night, accounting for 10–50% of the total respiratory rate, resulting in a reduced ATP yield per unit of carbon oxidized [128]. Among the different isoforms of AOX identified so far, one is constitutive, whereas the rest are stress inducible [129]. Engineering the constitutive AOX with a light-specific promoter can lower the alternative pathway rate at night and raise it again during the day without compromising the carbon loss [32].

11.2.6. Improving Nitrate Acquisition and Relocating Nitrate Assimilation

Plants take up nitrogen mainly in the form of nitrates from the soil in an energy-intensive process [130], which is further reduced in the roots and shoots [131]. The cost of NO_3^- acquisition can be minimized by identification and elimination of NO_3^- leaks that take place via the nitrate excretion transporter (NAXT1) or alternatively, increasing the flux density of an optimized NO_3^- transporter on root hair cells [32]. Further, the cost of nitrate reduction is 1.72 kg glucose C respired per kg nitrate N reduced to ammonia [132]. If the entire or most of the nitrate assimilation during the daytime takes place in the leaves, then the excess of NADPH and ATP produced during light reaction can be exploited under high light. This would reduce the additional cost of sucrose respiration in the roots, which is required for the generation of the carbon skeleton [32].

12. Conclusions and Future Outlooks

The significant upsurge in respiration rate under climate warming rather than an antagonistic change in photosynthetic rate disrupts the carbon economy of the plant, resulting in a yield penalty. The mechanism responsible for this yield penalty is increased utilization of non-structural carbohydrates to carry out maintenance respiration to support increased turnover of proteins, maintenance of ion gradients, and activation of energetically expensive heat tolerance mechanisms, thereby creating an overall deficit of carbohydrates partitioned towards growth respiration, eventually reducing the total dry matter production. At a cellular level, warmer temperatures lead to mitochondrial swelling as well as downregulation of respiration by increasing the ATP:ADP ratio, the ABA-mediated reduction in ATP transfer to the cytosol, and the disturbance in a concentration gradient of TCA cycle intermediates, as well as increasing lipid peroxidation in mitochondrial membranes and enough cytochrome c release to trigger programmed cell death. In plants, distinct respiratory metabolic adjustments are available in response to high temperatures and the diel cycle. Plants show thermal acclimation of the respiration response to lessen the impact of carbon loss due to increasing temperatures. Genome editing approaches to reduce unnecessary carbon loss and to increase the energy utilization efficiency of processes are ways to escalate positive carbon balance. This can be addressed by replacing, relocating, or rescheduling the metabolic pathways like substituting the lignin biosynthesis pathway, suppressing futile cycles that decrease the respiratory costs, bypassing photorespiration via metabolic engineering, engineering cultivars with reduced emission of BVOCs and a low alternative pathway rate at night, minimizing the cost of NO_3^- acquisition, and relocating NO_3^- assimilation from roots and shoots to leaves during the daytime. Thus, cutting respiratory losses and increasing photosynthesis are the most effective solutions to beat the heat in the presently warming world for and sustain crop productivity in the long run.

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