Research article

ROS signaling as common element in low oxygen and heat stresses

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1. Introduction

Many years of studies on plants that have been subjected to different biotic and abiotic stress conditions have suggested Reactive Oxygen Species (ROS) as ubiquitous markers of both oxidative stress and of signaling events for the induction of adaptive stress responses [64]. This dual function is tightly regulated by a balance between ROS production and scavenging, which is coordinated by a large gene network that is activated under stress conditions [63]. This equilibrium allows plants to maintain cell homeostasis, and also to accumulate ROS in subcellular compartments where they act as signals [64]. Various enzymes directly involved in the generation of ROS, such as the plant homolog of the mammalian respiratory burst NADPH oxidase (RBOH), have been identified and demonstrated to generate and amplify ROS production for signaling [62,97]. Organelles with a highly oxidizing metabolic activity or whose metabolism relies on electron transport (i.e. chloroplasts, mitochondria and microbodies) have also been found to be sources of ROS in plant cells and contribute to the oxidative burst during abiotic stresses [63,96]. ROS metabolisms in these organelles are also a source of retrograde signals to the nucleus that play important roles in stress acclimation [96].

Of the abiotic stresses, low O2 has prompted a controversial hypothesis regarding the activation of the oxidative metabolism, since it is seemingly illogical that an increase in ROS production is due to a decrease in the essential substrate O2. However, the presence of a ROS component in the transcriptome in response to low oxygen has been observed and an overlap with heat stress has been proved. It has been also demonstrated that ROS are produced during both anoxia and heat, but the site of their production remain contentious. Membrane NADPH oxidase and mitochondrial electron transport flow have been indicated as possible ROS generation systems. Both anoxia and heat have been shown to induce the transcription of Heat Shock Factors (HSFs) and Heat Shock Proteins (HSPs), among which HSFA2 and some of its targets. HSFA2 over-expressing plant has been shown to be more tolerant to anoxia, while the knockout hsfa2 lose the capability of wild type plants to cross-acclimate to anoxia through mild heat pre-treatment. The production of ROS seems to be an integral part of the anoxia and heat response, where HSFs likely play a central role in activating the HSP pathway. This mechanism is suggested to result in enhanced plant tolerance to both anoxia and heat.

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2. Low oxygen stress and adaptive responses in plants

Aerobic organisms require molecular O2 in order to efficiently produce ATP. Oxygen is the final acceptor of $e^{-}$ in the mitochondrial...
respiratory chain, which is the last step in respiration that leads to energy synthesis through ATP production [5]. Oxygen depletion has a rapid and severe consequence on cells, which are altered in terms of their metabolism, energy production and consumption as well as gene expression. However, aerobic organisms have evolved adaptive responses that can compensate for an energy crisis and in some cases confer tolerance [4]. In plants, transient flooding events, waterlogging and microbial activity in the soil frequently and rapidly lead to O₂ deficiency [29]. With an O₂ deficit, ATP formation is severely reduced and a tight regulation of energy production occurs through the shift of respiration from aerobic to anaerobic. This change relies primarily on glycolysis and fermentation to generate ATP and regenerate NAD⁺ to sustain glycolysis. However, an energy crisis follows since fermentation of one molecule of hexose yields only 2 mol ATP per mol hexose compared to up to 38 mol ATP produced by aerobic respiration. Moreover, both a short initial lactate and a long-lasting ethanol fermentation occur after glycolysis [73]. The accumulation of lactate can impair plant survival under water, since its dissociation seems to contribute to the acidification of the cytosol [109]. Under hypoxia, cells are still capable of producing ATP via oxidative phosphorylation, although consuming less O₂ and reorganizing metabolic fluxes for a more focused use of energy [53,83]. Together with a metabolic adjustment, plants adapt to O₂ limiting conditions through morphological modification. In rice, variety-specific adaptations include either rapid internode-leaf elongation to escape submergence and restore the contact with the air [35], or induction of a quiescent state to reduce the growth to a minimum under unfavorable conditions [25]. The formation of aerenchyma to facilitate gas exchange [19,72], the production of adventitious roots on stem areas [110] and the hyponastic growth of leaves [20] are only some of the morphological traits that contribute to the plant’s ability to survive low O₂. In addition, molecular reprogramming has been examined through several genome-wide analyses of many plant species exposed to different levels and extents of O₂ reduction [12,41,49,51,56,59,67,108]. Some of the transcripts modulated under O₂ depletion correspond to ROS detoxification enzymes, ROS-regulated TF and Heat Shock Proteins (HSPs), suggesting that ROS contribute to low O₂ signaling.

3. Secondary ROS signal in plants under low oxygen

Plant sensing of O₂ deprivation is a key aspect in adaptation. Recently, much progress has been made in understanding the mechanism behind low O₂ sensing, and a direct sensing mechanism of O₂ availability in Arabidopsis has been described [30,54]. The conserved N-terminal amino acid sequence Met-Cys present in some Ethylene Responsive Factors (ERF) of group VII (e.g. Arabidopsis HRE1, HRE2, RAP2.2 and RAP2.12) is the target of an ubiquitin-dependent protein degradation under normoxic conditions, through the oxidation of a Cys residue after Met cleavage [30,54]. Stabilization of the Met-Cys N-terminal motif under low O₂ with the consequent TF migration to the nucleus [54] leads to enhanced plant survival, through the control of the expression of core hypoxic genes related to the anaerobic metabolism such as ADH1, PDC1, and SUS4 [30]. It is still unclear whether the N-terminal Met-Cys stability depends directly on O₂ or cellular changes associated with its availability, such as cytosolic pH or ROS balance [30].

Interestingly, transcriptome adjustment under low O₂ also includes other genes that are likely not directly regulated by the N-end rule mediated oxygen sensing. A survey of microarray data produced under low O₂ has shown that a conserved group of HSPs was expressed together with the group of core genes related mostly to anaerobic metabolism [8,59,67]. Unlike the core of anaerobic genes, these HSPs are not constitutively expressed in the N-end rule Arabidopsis mutants such as atei1/2 and prt6 that lack some of the recognition steps required for proteasomal degradation [30,31]. atei1/2 lacks the arginyl-tRNA-protein transferase (ATE1 and ATE2) required to arginylate the oxidized Cys [120], prt6 lacks the E3 ligases called N-recognins (PRT6) that recognize the primary destabilizing residues for proteasome degradation [27]. Rather than directly depending on direct low-oxygen sensing, the induction of these HSPs under low O₂ seems instead to be dependent on an H₂O₂ burst that is observed early after the onset of O₂ deprivation [8]. Thus, it is tempting to speculate that the up-regulation of ROS-related proteins such as HSPs, but also TFs such as ZAT10 and ZAT12, which are known to be modulated under low O₂ [8,56,57], constitute a specific response under the control of a secondary signaling mechanism that may rely on redox modifications (Fig. 1).

Recent results have demonstrated that the oxidative burst is a common theme in heat and anoxia stress [8]. Of the TFs transcriptionally regulated by both conditions, HSFA2 is the most responsive to H₂O₂ [60], and is one of the TFs that are commonly up-regulated in different experiments related to ROS production in different plant cell compartments [26]. In eukaryotes, HSFs have been suggested to be the direct sensors of the ROS that modulate their activity [60]. Hydrogen peroxide has been found to promote a shift from the inactive monomers to the active homotrimer form of mammalian and Drosophila HSF1, likely via the redox regulation of the disulfide bond formation in the Cys residues located within the DNA-binding domain of the TF [3,122]. Superoxide anion is, on the other hand, responsible for the conformational change in Saccharomyces cerevisiae HSFs trimeric form to the activated state [50]. The S. cerevisiae response regulator Skn7 has been proposed to require H₂O₂ to interact with HSF1 and activate HSPs transcription [76]. Two hypothetical models of HSFs action in plants have been proposed by Miller and Mittler [60]. In the first, H₂O₂ regulates the constitution of the HSFs homotrimer form which acts as a transcriptional activator, in the second, the homotrimeric form of a particular HSF interacts with a second HSF via H₂O₂ mediation to induce gene expression [60]. Indeed, plants’ HSFs have been found to cooperate to regulate downstream events [61,88,87], however whether H₂O₂ directly functions as activator in plants is still unclear [58].

The presence of an oxidative burst under anoxia has been reported by Baxter–Burrel et al. [9], who showed that the activation of Rop2, a RHO-like small G protein of plants (Rop), under low O₂ contributes to H₂O₂ accumulation. This was shown to occur via a NADPH oxidase mechanism, which is also required for ADH expression [9]. Dominant negative Rop2 (DN-rop2) seedlings under O₂ deprivation showed neither transcript accumulation nor activity of ADH but increased sensitivity to stress [9]. The activity of Rop2 is likely to be controlled by a negative feedback regulation which requires a H₂O₂-regulated Rop GT-Pase activating protein (GAP), RopGAP4, that preferentially stimulate the GT-Pase activity of Rop and thus inactivates Rop2 [9]. The disruption of the RopGAP4 in ropgap4-1 seedlings increases ADH expression but decreases tolerance to O₂ deprivation. Thus, tolerance to low O₂ requires both Rop2 activation and RopGAP4-dependent negative feedback regulation which modulates H₂O₂ production and ADH activity and expression [4,9]. Whether this mechanism might in part overlap with the N-end rule pathway in regulating ADH expression is still unclear (Fig. 1). The physiological and biochemical nature of the signal that activates this pathway remains speculative.

In animals, the heterodimer TF Hypoxia Inducible Factor 1 (HIF-1) has been identified as a direct sensor and regulator of transcriptional responses under hypoxia [38,39]. Mammalian HIF-1 is
made up of the hypoxia-induced HIF-1α subunit and the constitutively expressed HIF-1β one. Under aeration, HIF-1 is not active, since HIF-1α is hydroxylated at two prolyl residues by the prolyl-hydroxylase (PHD) enzymes which require O2 as a co-substrate. The aerobically hydroxylated HIF-1α is degraded via the ubiquitin-mediated proteasome degradation through the recognition of hydroxylation tags by the von Hippel-Lindau (VHL) tumor suppressor [37,69]. Reduced O2 availability reduces the rate of HIF-1α degradation with the subsequent activation of the transcriptional complex responsible for the activation of the transcription of a large number of hypoxia-related genes [91]. The exogenous or endogenous induction of ROS under normoxia is known to stabilize HIF-1α [15,32]. In addition, treating mammalian cells with antioxidants abolishes the HIF-1 response [14,32], thus suggesting a role for ROS in controlling HIF-1 action. In plants, no orthologs of the animal HIF have been found so far [55].

4. Enigmatic site of ROS production under oxygen depletion

4.1. RBOH—NADPH oxidase function under low oxygen in plants

Reactive oxygen species are by-products of normal cellular metabolism with high oxidizing activities such as photosynthesis, respiration and photorespiration. They are also directly produced by enzymes, which include oxalate and amine oxidase, peroxidases and NADPH oxidases (RBOHs). RBOHs likely play a key role under several stress conditions [97], regulating ROS signaling in response to heat, drought, cold, high-light, salinity and wounding, but also biotic stresses and developmental processes [97]. RBOHs produce O2 by oxidizing NADPH and transferring electrons to O2 at the plasma membrane [61,85,100]. The oxidative burst which produces ROS via this system occurs in the apoplast [103]. Under low O2, plants treated with diphenylene iodonium chloride (DPI), which is an inhibitor of flavin-containing NADPH oxidases, show a reduction in ADH activity, thus indicating that ADH action requires a DPI-sensitive NADPH oxidase [9]. DPI treatment reduces the low O2 stress tolerance of wild type Arabidopsis plants, but also increases the survival of ropgap4-1 seedlings, thus showing that low O2 tolerance needs either the activation or deactivation of DPI-sensitive NADPH oxidase [9]. RBOHD is known to mediate many processes such as (a)biotic stress responses, stomatal movements, lignification [23,47,61,101,102]. It is transcriptionally regulated under some abiotic stresses [97], and its up-regulation is observed under low O2, rbohD and rbohB mutants have been shown to be sensitive to heat stress [48]. ROS generated by RBOHD could be a common response between heat and anoxia.

A relationship between RBOH-dependent ROS production and Ca2+ homeostasis has been suggested in Arabidopsis [97]. It is likely that Ca2+ binds to the EF-hands of the N-terminal region of RBOH.

Fig. 1. Model of the signal transduction events under low oxygen. ERF transcription factors are constitutively expressed under normoxia and are the target of proteasomal degradation via the N-end rule pathway. Under hypoxia, the N-end is stabilized and TFs migrate to the nucleus to activate anaerobic gene expression [30,54]. Under anoxia, a mitochondrial imbalance leads to the production of ROS that regulate MAPK3/6 [17]. A ROS rheostat mediated by NADPH oxidase is also activated [9]. TFs related to ROS signaling are induced, such as HSFA2, ZAT12, ZAT10, leading to the promotion of ROS-related genes such as HSPs [7,8]. These two processes contribute towards plant tolerance to hypoxia and anoxia.
and, together with RBOH phosphorylation, promotes ROS production, with the subsequent activation of Ca^{2+} channels in a positive feedback amplification [68,97,100]. Transfer to anoxia has been shown to induce Ca^{2+} modulation in Arabidopsis, maize, and rice. In maize, the induction of ADH under anoxia is likely to require an increase in cytosolic Ca^{2+} [94,95]. In Arabidopsis and rice, the oscillation of cytosolic Ca^{2+} was observed following transfer to anoxia, with Ca^{2+} channel blockers partially inhibiting the ADH anaerobic induction [90]. Since a cytosolic increase in Ca^{2+} is a prerequisite for the activation of H_{2}O_{2} production by Ca^{2+} dependent NADPH oxidase, it is tempting to speculate a connection between Arabidopsis Rop-promoted H_{2}O_{2} production [9] and cytosolic Ca^{2+} increases [90] in regulating ADH expression and activities (Fig. 1).

### 4.2. The role of mitochondria in ROS production under low oxygen: evidence from animals and plants

Mitochondria represent the major sink of cellular O_{2}. It is thus reasonable to suppose that this organelle is equipped with an O_{2} availability sensing mechanism [116]. The nature of the hypoxic signal produced by mitochondria is still under debate, since it may involve the cell energy state, the redox homeostasis, and also the direct production of ROS [132].

In mammalian cells belonging to vascular tissues, the role of mitochondria in ROS production under low O_{2} has been hypothesized [116,118]. Although related to very specific structures (e.g. pulmonary and systemic arteries cells in response to alveolar hypoxia) these hypothesis could furnish suggestions for other less specialized systems. One hypothesis is that severe hypoxia limits the mitochondrial electron chain at complex IV, where O_{2} is reduced to H_{2}O. Oxygen deprivation at complex IV might affect the redox state of the upstream electron carriers and initiate redox-signaling downstream events [118]. Under normoxia, mitochondria complexes produce O_{2}, not only in the mitochondrial matrix but complex III also in the intermembrane space [32,66,104]. In this latter subcellular compartment, an over-production of ROS as consequence of homeostasis disruption may be more likely to reach the cytosol and act as a signal [32,33]. Superoxide can spontaneously, or via SOD, dismutate to yield H_{2}O_{2}, which as a non-charged molecule, can likely diffuse through the mitochondrial membranes [33,104]. Recently, the fluorescent redox sensor roGFP was used to provide a measure of the protein thiol redox state in the mitochondrial matrix, the intermembrane space, and the cytosol in mammalian cells [117]. During hypoxia, roGFP oxidation in the cytosol and the intermembrane space showed an oxidative shift, indicating ROS production in that cell compartment [117]. This hypoxia-induced ROS production was reduced by over-expressing cytosolic catalase, thus demonstrating H_{2}O_{2} involvement in the process [117].

The use of specific inhibitors has further led to the suggestion that the critical step for ROS generation in mammalian cells under low O_{2} resides in mitochondria at Complex III [14]. Indeed, while Complex I inhibitors, such as rotenone, and Q_{o} site of the Complex III inhibitors, like myxothiazol (MYX), were able to suppress the rise of HIF-1α under low O_{2}, the Q_{o} site inhibitor antimycin A (AA) had no effect on the subunit rise [14,15,32].

Very recently, evidence of mitochondria-associated ROS production involvement in response to O_{2} deprivation in Arabidopsis seedlings has been demonstrated [17]. Upon O_{2} deprivation and also reoxygenation, the rapid and transient activation of mitogen-activated protein kinases (MPK) 3, 4 and 6 has been found to occur and to be activated by mitochondrial ROS. Treatment of normoxic plants with mitochondrial ETC inhibitors acting at complex III (AA) and at complex IV (potassium cyanide, KCN) also showed MPK6 and 3 activation, together with ROS production in mitochondria [17]. Both AA and KCN promote ubisemiquinone accumulation, increasing ROS formation. Moreover, both AA and KCN pre-treatment decreased MPK6 and 3 activation when seedlings were transferred to O_{2} deprivation or reoxygenation, sustaining the link between mitochondrial ROS and MPK activation [17]. MPK cascades are known to be regulated by several stresses that have an oxidative component [2,40,114,121]. The over-expression of MPK6 leads to enhanced survival to O_{2} deprivation, but not significant modulation of classic transcripts related to the anaerobic metabolism, thus suggesting a benefit for plant survival that is probably related to another pathway [17].

Indeed, a link between functional mitochondria and HSP production has been shown [81]. Arabidopsis cell treatment with mitochondrial inhibitors and uncouplers during mild heat shock down-regulates HSP production, which is necessary for thermotolerance [81].

In pulmonary artery smooth muscle cells, Rathore et al. [77] suggest that hypoxia-driven mitochondrial ROS signaling triggers NADPH oxidase activity. This would imply a mechanism by which both mitochondria and cytosol contribute to the increase in ROS production and signaling during low O_{2}, at least in animals.

### 5. ROS is a common theme in low oxygen and heat stress

#### 5.1. ROS-mediated activation of heat shock response (HSR)

Heat is one of the most serious abiotic stresses for plants due to the extent of irreversible damage it can cause at a morpho-anatomical, physiological and biochemical level [113]. Heat stress can strongly interfere and affect normal cell homeostasis by disrupting the metabolic coordination existing within cells and inside a cell, where each pathway is likely to have a temperature optimum in order to work properly [98]. Usually, plants can withstand temperatures of 5–10 °C above the optimum, specific for each species, by activating the HSR [88] without being stressed [52]. However, when temperatures suddenly rise to high levels, cells can die in a few minutes, as a consequence of a collapse in cellular organization [113].

When plants are subjected to heat stress, they activate a plethora of physiological, molecular and biochemical responses in order to react and survive. General modifications include changes in water status, the accumulation of osmolytes such as proline, hormonal changes, the production of secondary metabolites and alterations in photosynthetic activity, assimilate partitioning, and cell membrane permeability [113]. All these events take a part in a well ordered temporal program to prevent cell death during heat stress (Fig. 2) [113]. At a molecular level, heat stress can lead to genome-wide re-programming events, with de novo synthesis of a heat-related set of proteins, specific to HSR. The synthesis of HSPs represents the typical mechanism of a heat-stress response, which has been observed in various kingdoms, from bacteria to animals [111]. HSPs prevent aggregation, denaturation, misfolding and the degradation of proteins under stress conditions [34]. However, Heat Shock related Genes (HSG) up-regulation has been found to be a general response to biotic [1] and abiotic stress conditions [84,86,99,115], including low O_{2} stress [78,67].

An intimate relationship between HSR and oxidative stress is likely to exist [3], since one of the events occurring in response to high temperatures is ROS production. A burst of H_{2}O_{2} has been found to occur in tobacco Bright-Yellow 2 cells undergoing PCD at high temperatures, likely due to an impaired mitochondrial oxidative phosphorylation and imbalance in H_{2}O_{2} scavenging [106]. Hydrogen peroxide has been suggested to be required for heat stress and the HSF-dependent expression of genes in Arabidopsis cell cultures, where it likely contributes to the induction of
that during the early phase of heat stress at both 44 °C and that this TF is accumulated in the presence of H2O2 [22].

transcript is up-regulated in AtHSF21 knockdown plants [21,74] and that this TF is accumulated in the presence of H2O2 [22]. Another study revealed that transgenic Arabidopsis plants over-expressing HSF3 show increased APX activity during the post-hypothermia phase and APX2 transcript accumulation during increasing temperature [70,78].

All this evidence from biochemical and genetic studies suggests a link between ROS and HSGs and can help to build the hierarchy of genes involved in ROS-mediated stress responses. Miller and Mittler suggested that HSFA4a might rely on a function of TF as a direct ROS sensor, since it likely acts upstream of ZAT12 and APX1 and shows a rapid response to H2O2 [21,60]. However, further studies are necessary to strengthen this hypothesis and to extend it to a more general ROS-related mechanism of sensing, directly mediated by HSFs.

5.2. Nuclear-mitochondrial cross-talk under heat stress

Retrograde signaling from organelles communicates a change in cell homeostasis to the nucleus, allowing the nucleus to subsequently modulate anterograde control for plant acclimation [119]. Several possible mechanisms of signaling between the mitochondria and nucleus are thought to occur under stress conditions, involving changes in [Ca^{2+}], ROS production, O2 tension and the hyperpolarization of the mitochondrial inner membrane. Although some progress has been made in understanding the communication between the two cellular compartments, this process is still largely unknown [4,10,75,79,81]. Recent evidence supports the idea of mitochondrial retrograde regulation as being intimately linked to ROS generation and accumulation [78,79]. During heat stress, Ca^{2+} uptake by mitochondria and ROS generation have been suggested [79,81]. The ROS source is hypothesized as residing predominantly in the inner membrane of mitochondria where hyperpolarization occurs [81]. Agents that are capable of preventing an increase in inner mitochondrial membrane electrochemical potential also inhibited HSG expression and thermotolerance [81].

In fact, a connection between mitochondrial-mediated signaling and HSG induction has been observed in several plant species. In maize mutants impaired in mitochondrial activity, the expression of some HSGs is up-regulated [46]. Arabidopsis plants transformed with a mitochondrial HSP from Zea mays show the induction of nuclear HSGs [80]. In this heterologous system, the ZmHSF22 transgene was expressed constitutively and it could enter Arabidopsis mitochondria to constitute the mature protein. Under heat stress, Arabidopsis transgenic plants expressing ZmHSF22 display altered expression of several nuclear genes encoding mitochondrial HSPs (AtHSP23.6), and also HSPs localized in chloroplast (AtHSP25.3 and AtHSP70.6) and cytosol (AtHSP17.4 and AtHSP70.1). In addition, Arabidopsis ZmHSF22 plants showed improved thermotolerance [80]. These results suggest that a heat-induced mitochondrial
retrograde signal via ROS production could regulate HSGs expression, and thus also tolerance.

5.3. Is heat-anoxia cross tolerance a ROS-mediated mechanism?

Plant responses to different (a)biotic stress conditions include a transcriptional modulation that is regulated by the presence of cis motifs on the promoter region of stress-responsive genes. Of these motifs, HSEs are represented in several stress-related genes [21,60,65,82]. Indeed, Arabidopsis seedlings show the expression of HSFs and HSP family members under different stress conditions, such as heat, cold, osmotic stress, salt, drought, genotoxicity, ultraviolet light, oxidative stress, wounding, pathogen infection [99], and also O2 deprivation [7,8,67].

This molecular communion might support cross-tolerance, which allows plants to adapt/acclimate to a range of different stresses after exposure to a specific stress [71]. As sessile organisms, plants are usually needed to counteract a combination of (a)biotic stress conditions after exposure to a specific stress. Of these, some transcripts (e.g. AT12 and AOX1a) belong to the oxidative-responsive gene group [26,42,107]. This might explain the cross-tolerance between heat and anoxia and the higher tolerance of HSF2A2 over-expressing plants to anoxia [7,8]. Mild heat pre-treatment, in addition to hypoxic treatment, increases tolerance to anoxia in Arabidopsis [7]. Moreover, hsflA2 mutants do not cross-acclimate to anoxia following mild heat pre-treatment [8].

This heat-mediated cross tolerance event in Arabidopsis is likely to be independent of a low O2 metabolism and the shift towards the adaptive fermentative pathway, since anoxic acclimation through a mild heat stress has also been found to be present in adh mutants impaired in the alcoholic fermentation pathway [7,24]. In fact, OX2-HSFA2 show a constitutive higher expression of target HSGs (e.g. HSP25.3-P, HSP18.2-CL, APX2) but not of anaerobic genes (e.g. ADH1 and SUS4) [8]. In animal systems, a moderate heat shock is involved in increased resistance to hypoxia, suggesting the involvement of HSPs in cross-tolerance heat versus low O2 [92]. In addition, the constitutive expression of some HSPs in turtle hearts has been associated with an increase in anoxia tolerance [16].

6. Concluding remarks

Although the role of ROS as secondary messengers in plant responses to O2 deprivation is controversial, a common modulation of transcripts related to the redox-metabolism has been observed. A striking overlap between the molecular signaling produced under low O2 and heat stress has also been found, concomitantly with cross tolerance when anoxia takes place after mild heat stress treatment.

Both anoxia and heat have been shown to induce the transcription of HSGs. The heat shock TF HSFA2 likely plays a central role in activating the HSP pathway, which then results in enhanced plant tolerance to both O2 deprivation and heat. Results from mammalian and yeast cells suggest that HSFs are sensors of H2O2, a model that still awaits confirmation for plants. Indeed, an oxidative burst has been observed after both O2 deprivation and heat, thus supporting the link between H2O2 production and HSFs activation.

However, many questions remain. The production site of ROS under these stresses is still being debated, although evidence from both the plant and animal kingdoms supports both plasma membrane NADPH oxidase and mitochondria ETC as principal sources. In the latter case, a fascinating area of research regards the idea of mitochondrial retrograde signaling in the control of plant acclimation to stress. The mechanisms that finally lead to heat-anoxia cross-tolerance and acclimation remain elusive and further research is needed to elucidate this link.

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