

Plant Mitochondrial Oxidative Stress and Cellular Signaling

Hanan A Hashem*

Department of Botany, Faculty of Science, Ain Shams University, Cairo, Egypt

*Corresponding Author: Hanan A Hashem, Department of Botany, Faculty of Science, Ain Shams University, Abbassia, Cairo, Egypt.

Received: January 10, 2018; Published: February 24, 2018

Abstract

Environmental, biotic and abiotic stresses applied to plants are well known to induce oxidative stress in plant cells. These stresses alter plant metabolism, growth and development and, at their extremes, can lead to death. Recently, a number of studies have begun to examine the changes that occur within plant mitochondria following the induction of oxidative stress. The accumulation of reactive oxygen species and reactive nitrogen species, changes in protein abundance and their interactions in mitochondria following exposure to external stress, and the role of these changes in signaling beyond mitochondria, combine to define the importance of mitochondria as environmental sensors. In this mini-review we will spot light on the major metabolic changes occurred in plant mitochondria as induced by oxidative stress.

Keywords: Mitochondrial Oxidative Stress; Cellular Signaling; Reactive Oxygen Species

Introduction

Our general understanding of mitochondria is as organelles in mammalian and plant cells that produce energy in the form of adenosine triphosphate (ATP) through an electron transport chain (ETC) containing four large protein respiratory complexes. These four complexes are: NADH-dehydrogenase (complex I), succinate dehydrogenase (complex II), ubiquinone-cytochrome c oxidoreductase (complex III) and cytochrome oxidase (complex IV). Mitochondria produce carbon dioxide through the citric acid cycle (Krebs cycle, tricarboxylic acid cycle, TCA) in addition to many intermediates with central role in the synthesis of biologically important compounds such as fatty acids and amino acids [1].

In plants, as in all other eukaryotes with mitochondria, the mitochondrial DNA (mtDNA) plays a vital role in mitochondrial biogenesis and respiratory function. It does so by encoding a small number of essential polypeptides of the respiratory chain. As a result of being sessile unable to avoid deleterious environmental stresses, plants have developed defense mechanisms to tolerate stress, some of which involve the mitochondrion. Plant mitochondria also play an important role in gene functions coordination with other organelles, including plastids [2].

Accumulation of ROS and RNS in mitochondria

Molecules typically referred to as reactive oxygen species (ROS) in plant cells include ozone, singlet oxygen, superoxide, H_2O_2 , and the hydroxyl radical. There is no reliable information on any significant generation of ozone and singlet oxygen by plant mitochondria, and the short half-life of the hydroxyl radical makes it incompatible with specific roles in signal transduction through selective modification of target molecules [3,4]. This suggest that superoxide and H_2O_2 are the reactive oxygen species generated in plant mitochondria (mtROS) with regulatory significance.

Mitochondria contain two terminal oxidases that reduce oxygen to water and the entire electron transport chain ETC is known to be a significant source of ROS under normal conditions. Under steady state conditions, this ROS production is dealt with by antioxidant enzymes and small molecules to limit cellular damage. However, under some conditions, these defenses are overwhelmed and ROS accumulate. Superoxide is produced in mitochondria by peripheral single electron transfers from reduced components in the ETC to oxygen [5]. Components in complex I, II and III have all been identified as major production sites but display fundamentally different rates of superoxide release [6]. The rate of superoxide production by mitochondria depends on the concentration of oxygen and on the redox balance of ETC components. Therefore, ROS production by mitochondria is low during hypoxic conditions [7] and can be altered by environmental factors and chemicals that alter the rate of these peripheral electron transfer reactions [8].

Several studies suggested that mitochondria are capable of producing nitric oxide (NO) via the reduction of nitric acid. NO is considered a key signaling molecule play a vital function in various physiological processes of plant under both normal and stress conditions. NO also react with O_2 to give reactive nitrogen species (RNS). This finding was proved in *Fusarium* fungus [9], green algae *Chlorella sorokiniana* [10], tobacco cell suspension [11] and pea, barley, *Arabidopsis* roots [12]. Under oxygen free condition, complexes III and IV are the sites for NO production in the presence of NADH via a leakage of electrons to nitrite [13,14]. Plants use mitochondrial-derived ROS and RNS as signaling molecules, particularly during stress [15]. ROS react with NO to produce peroxynitrite and other RNS which are proved to be signal molecules during the nitrosative stress. These findings proved that mitochondria are not only a powerhouse of the cell but also have a crucial role in cell signaling.

Proteomic changes in mitochondria during oxidative stress

A number of studies have revealed global changes in protein abundance of mitochondrial proteins following conditions that induce oxidative stress in a wide range of plant species [16-18]. Recently it has also been shown that the large respiratory subunits of the ETC also coordinate protein changes to alter respiration in response to oxidative stress conditions [19]. Changes in the thiol redox state of mitochondrial proteins are significant response to oxidative stress [20]. Potential protein thiol alterations include formation of mixed disulfides or internal disulfides from vicinal dithiol, S-nitrosation, and the formation of higher oxidation states [21]. Protein thiols have differential reactivity and range of lifetimes for redox states that enable them to act as signal sensors or transducers that potentially affect mitochondrial function [22].

Generally, mitochondrion protected from excess ROS by accumulating a number of antioxidant enzymes that detoxify ROS and many have been observed to vary in abundance during oxidative stress including: Mn-superoxide dismutase; metalloprotein [23], ascorbate peroxidase [24] and glutathione peroxidase [23]. In addition, other proteins have been observed to increase in abundance including mitochondrial class I and mitochondrial class II small heat shock proteins (sHsps) [25]. Small heat shock proteins are molecular chaperones that play major roles in preventing protein denaturation and aggregation, as well as facilitating the correct refolding of denatured proteins [26].

Conclusion

Plant mitochondria are proved to play a critical role in plant response machinery to oxidative stress. Accumulation of ROS and RNS as signaling molecules as well as proteomic alteration are detected. Further studies are still needed to explore the mechanisms underlying mitochondrial transcriptomic and proteomic changes as well as molecular signaling associated with oxidative stress.

Bibliography

- Gerald K. "Cell and Molecular Biology: Concepts and Experiments". Publisher: John Wiley & Sons; 5th Edition (2007).
- Machenzie S and McIntosh L. "Higher plant Mitochondria". *The Plant Cell* 11.4 (1999): 571-585.
- Rejeb IB., *et al.* "Plant responses to simultaneous biotic and abiotic stress: Molecular mechanisms". *Plants (Basel)* 3.4 (2014): 458-475.
- Huang S., *et al.* "The roles of mitochondrial reactive oxygen species in cellular signaling and stress response in plants". *Plant Physiology* 171.3 (2016): 1551-1559.
- Moller IM. "Plant mitochondria and oxidative stress: Electron transport, NADPH turnover, and metabolism of reactive oxygen species". *Annual Review of Plant Physiology and Plant Molecular Biology* 52 (2001): 561-591.
- Murphy MP. "How mitochondria produce reactive oxygen species". *Biochemical Journal* 417.1 (2009): 1-13.
- Noctor G., *et al.* "Mitochondrial redox biology and homeostasis in plants". *Trends in Plant Sciences* 12.3 (2007): 125-134.
- Moller IM., *et al.* "Oxidative modifications to cellular components in plants". *Annual Review of Plant Biology* 58 (2007): 459-481.
- Tielens AG., *et al.* "Mitochondria as we don't know them". *Trends in Biochemical Sciences* 27.11 (2002): 564-572.
- Tischner R., *et al.* "Mitochondrial electron transport as a source for nitric oxide in the unicellular green alga *Chlorella sorokiniana*". *FEBS Letters* 576.1-2 (2004): 151-155.
- Planchet E., *et al.* "Nitric oxide emission from tobacco leaves and cell suspensions: rate limiting factors and evidence for the involvement of mitochondrial electron transport". *Plant Journal* 41.5 (2005): 732-743.
- Gupta KJ., *et al.* "In higher plants, only root mitochondria, but not leaf mitochondria reduce nitrite to NO, in vitro and in situ". *Journal of Experimental Botany* 56.420 (2005): 2601-2609.
- Gupta KJ and Kaiser WM. "Production and scavenging of nitric oxide by barley root mitochondria". *Plant and Cell Physiology* 51.4 (2010): 576-584.
- Gupta KJ and Igamberdiev AU. "Reactive nitrogen species in Mitochondria and their implications in plant energy status and hypoxic stress tolerance". *Frontiers in Plant Science* 7 (2016): 369.
- Gupta KJ and Igamberdiev AU. "The anoxic plant mitochondrion as a nitrite: NO reductase". *Mitochondrion* 11.4 (2011): 537-543.
- Huang BR., *et al.* "Root carbon and protein metabolism associated with heat tolerance". *Journal of Experimental Botany* 63.9 (2012): 3455-3465.

17. Komatsu S., *et al.* "Comprehensive analysis of mitochondria in roots and hypocotyls of soybean under flooding stress using proteomics and metabolomics techniques". *Journal of Proteome Research* 10.9 (2011): 3993-4004.
18. Hossain Z., *et al.* "Plant cell organelle proteomics in response to abiotic stress". *Journal of Proteome Research* 11.1 (2012): 37-48.
19. Tan YF, *et al.* "Components of mitochondrial oxidative phosphorylation vary in abundance following exposure to cold and chemical stresses". *Journal of Proteome Research* 11.7 (2012): 3860-3879.
20. Jha N., *et al.* "Glutathione Depletion in PC12 Results in Selective Inhibition of Mitochondrial Complex I Activity". *Journal of Biological Chemistry* 275.34 (2000): 26096-26101.
21. Demple B. "A bridge to control". *Science* 279.5357 (1998): 1655-1656.
22. Lin., *et al.* "Specific Modification of Mitochondrial Protein Thiols in Response to Oxidative Stress". *The Journal of Biological Chemistry* 277.19 (2002): 17048-17056.
23. Jiang Y., *et al.* "Comparative proteomic analysis of NaCl stress-responsive proteins in Arabidopsis roots". *Journal of Experimental Botany* 58.13 (2007): 3591-3607.
24. Dooki AD., *et al.* "Proteomic responses of rice young panicles to salinity". *Proteomics* 6.24 (2006): 6498-6507.
25. Siddique M., *et al.* "The plant sHSP superfamily: Five new members in Arabidopsis thaliana with unexpected properties". *Cell Stress Chaperones* 13.2 (2008): 183-197.
26. Zeng L., *et al.* "The potential role of small heat shock proteins in mitochondria". *Cell Signal* 25.11 (2013): 2312-2319.

Volume 2 Issue 3 March 2018

© All rights are reserved by Hanan A Hashem.