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Mini Review

Plant Mitochondrial Oxidative Stress and Cellular Signaling

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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.

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