Mitochondrion 19 (2014) 238–244

Contents lists available at ScienceDirect

Mitochondrion

journal homepage: www.elsevier.com/locate/mito

Plant mitochondria under pathogen attack: A sigh of relief or a last breath?

Francisco Colombatti, Daniel H. Gonzalez, Elina Welchen *

Instituto de Agrobiotecnología del Litoral (CONICET-UNL), Cátedra de Biología Celular y Molecular, Facultad de Bioquímica y Ciencias Biológicas, Universidad Nacional del Litoral, CC 242 Paraje El Pozo, 3000 Santa Fe, Argentina

ARTICLE INFO

Available online 3 April 2014

Keywords:
Biotic stress
Pathogen
Plant mitochondria
Alternative oxidase
Defense response
Cytochrome *c*

ABSTRACT

Plants constitute excellent sources for pathogen nutrition and survival. To fight against pathogen attack, higher plants have developed a sophisticated immune system responsible for pathogen recognition and activation of downstream defense responses. After pathogen perception, mitochondria play an important role in the defense strategy of the plant cell, integrating and amplifying diverse signals such as salicylic acid, nitric oxide, reactive oxygen species (ROS) or pathogen elicitors. Signals perceived by mitochondria usually impact on their normal function, destabilizing the organelle, generating changes in respiration, membrane potential and ROS production. At this stage, mitochondria produce several signals influencing the redox state of the cell and promoting changes in the expression of nuclear genes by mitochondrial retrograde regulation. At more advanced stages, they promote programmed cell death in order to avoid pathogen propagation to the whole plant. Recent evidence indicates that plants and pathogens have evolved mechanisms to modulate the immune response by acting on mitochondrial functions. In this review, we summarize knowledge about the involvement of mitochondria in different aspects of the response of plants to pathogen attack.

1. Introduction

Plants represent an excellent source of water and nutrients for microbes that can easily enter plant tissues through wounds or natural entrances like hydathodes or stomata, remain in the apoplast and move through the xylem vascular system (Abramovitch et al., 2006). Plant–pathogen interaction is a complex process and the events triggered after the first recognition depend on many factors such as the type of microbe or pathogen (i.e. bacteria, viruses, fungi or insects), and the characteristics and health of the plant.

When a pathogen interacts with a plant cell, it induces a sophisticated immune response in the plant. The extent of the response depends on the pathogen characteristics and the mechanism used by the foreign organism to elude the plant defense system. Briefly, the plant immune system has two different branches: the basal defense or PTI/MTI (PAMP-triggered immunity or MAMP-triggered immunity) and the induced defense or ETI (effector-triggered immunity) (Dangl and Jones, 2001; Jones and Dangl, 2006). The first or basal defense barrier is raised when the extracellular part of plant cell membrane receptors, known as PRRs (Pattern Recognition Receptors), recognizes microbial or pathogen associated molecular patterns (MAMPs or PAMPs). PRRs are typical RLKs (receptor like kinases) and they are able to generate rapid transcriptional changes inside the plant cell (Fig. 1, #1–4). Many pathogens have evolved different strategies to elude the barrier imposed by PTI, by releasing effector molecules that can interfere with signal transduction between PRRs and the nucleus, leading to plant ETS (effector-triggered susceptibility). However, if pathogen-effectors are specifically recognized by R (Resistance) proteins of the plant cell, the second defense barrier known as ETI is raised. During ETI, pathogen effectors are recognized by intracellular receptors or NB-LRR (nucleotide binding-leucine rich repeat domain) proteins (Abramovitch et al., 2006; Jones and Dangl, 2006). ETI finally leads to HR (hypersensitive response) in the infection site. This response is a kind of plant programmed cell death (PCD) established in order to isolate pathogenic cells and confine

Abbreviations: AA, antimycin A; AOX, alternative oxidase; BA, Bongkrekic Acid; COX, cytochrome *c* oxidase; CsA, cyclosporine A; CypD, cyclophilin D; ETC, electron transfer chain; ETI, effector-triggered immunity; ETS, effector-triggered susceptibility; HR, hypersensitive response; IMM, mitochondrial inner membrane; MAMP, microbe-associated molecular pattern; MAPK, MAP kinase; mGDC, mitochondrial glycine decarboxylase; MMP, mitochondrial membrane potential; MnSOD, manganese superoxide dismutase; mROS, mitochondrial ROS; MTI, MAMP-triggered immunity; NB-LRR, nucleotide binding-leucine rich repeat domains; NO, nitric oxide; NOS1, nitric oxide synthase 1; O_2^-, superoxide; OMM, outer mitochondrial membrane; PAM16, pre-sequence translocase-associated protein import motor 16; PAMP, pathogen-associated molecular pattern; PCD, programed cell death; PPIX, protoporphyrin IX; PR, pathogenesis related; PRRs, Pattern Recognition Receptors; PTI, PAMP-triggered immunity; PTP, permeability transition pore; R proteins, resistance proteins; RBOH, respiratory burst oxidase homolog; RLK, receptor like kinase; RNS, reactive nitrogen species; ROS, reactive oxygen species; SA, salicylic acid; SNC1, suppressor of NPR-1 constitutive 1; T3E, type III effector; T3SS, type III secretion systems; UCP, uncoupling protein.

* Corresponding author at: Cátedra de Biología Celular y Molecular, Fac. de Bioq. y Cs. Biol. (UNL), Ciudad Universitaria, Paraje El Pozo, 3000 Santa Fe, Argentina. Tel./fax: +54 342 4575219.

E-mail address: ewelchen@fbcb.unl.edu.ar (E. Welchen).

http://dx.doi.org/10.1016/j.mito.2014.03.006
1567-7249/

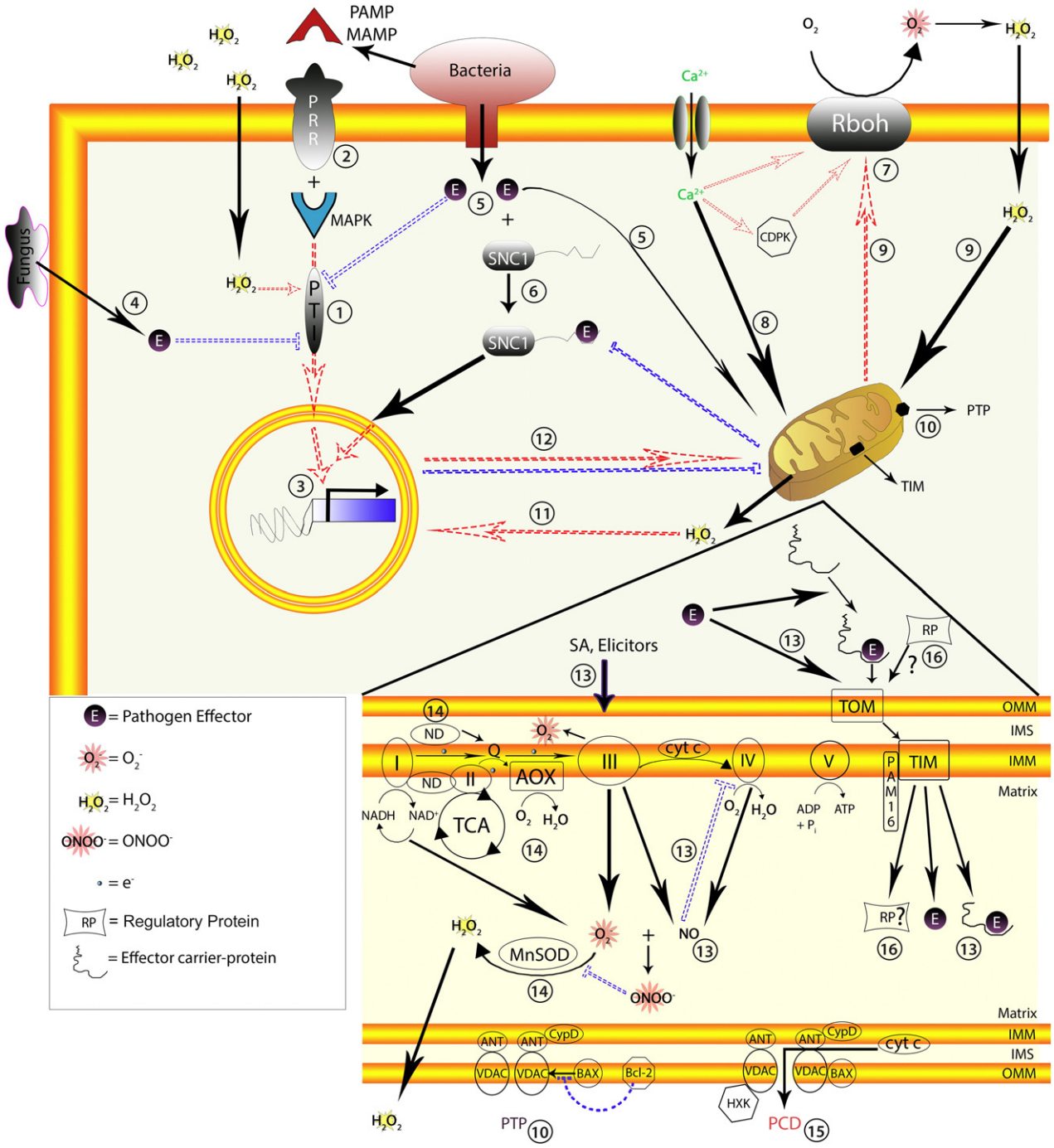

Fig. 1. The many roles of mitochondria in the response of plants to pathogens. Basal defense or PTI (1) is activated when extracellular plant cell membrane receptors (PRR, 2) recognize specific PAMPs or MAMPs and, through MAPK cascades, are able to generate rapid transcriptional changes in the nucleus of the plant cell (3). Many pathogens have evolved different strategies to elude the barrier imposed by PTI, thus releasing effector molecules (4, 5) involved in suppressing basal defense. Effector recognition by R proteins triggers the second defense barrier known as ETI. SNC1 is an example of a TIR NB-LRR R protein that would be responsible for recognition of unknown pathogen effectors (6). In addition, upon pathogen perception an oxidative burst takes place in the cell, promoted by RBOH-family proteins present in the plasma membrane (7). Mitochondria are deeply involved in the signaling networks related to pathogen attack. They modulate changes in cytoplasmic Ca^{2+} and ROS related to activation of RBOH (8, 9). These events initiate a depolarization of the mitochondrial membrane which initiates the formation of the PTP (10). As a consequence of changes in respiratory activity and ROS production, mitochondria produce several signals that influence the expression of nuclear genes in a process known as mitochondrial retrograde regulation (11). Changes perceived in the nucleus, in turn, modify the expression of several mitochondrial proteins (12). Several compounds related with plant–pathogen interactions, like SA, pathogen elicitors, effectors and NO, impact on mitochondrial function (13). The activities of alternative NAD(P)H dehydrogenases (ND), AOX and MnSOD modulate the balance between different ROS, like O_2^- and H_2O_2 (14). Under biotic stress conditions, mROS influence the behavior of the whole cell leading to diverse levels of responses that can culminate in the establishment of PCD (15). Recently, a protein connected with the TIM23 translocase, PAM16, was identified as a negative regulator of the immune response, suggesting the existence of regulatory proteins (RP) that are imported to mitochondria and impact on the development of ETI (16).

them at the site of infection, blocking their spread through the plant (Jones and Dangl, 2006; Lam et al., 2001) (Fig. 1, #5–7). Both PTI and ETI activate similar signaling pathways including MAP kinase (MAPK) cascades, calcium ion efflux, changes in transcriptional programs and hormone signaling, lignin and callose deposition at the cell wall and increase ROS production (Block and Alfano, 2011; Chisholm et al., 2006) (Fig. 1, #7–9, #11).

Upon pathogen recognition, an "oxidative burst" characterized by a rapid and transient apoplastic ROS (reactive oxygen species) production takes place. The sources for this apoplastic oxidative burst are plant