The Enigmatic Metabolite Transport in Plant Mitochondria Lacking Proton Motive Force - news from Durum Wheat Mitochondria

Donato Pastore*
Department of Agricultural, Food and Environmental Sciences – University of Foggia, Italy

*Corresponding author: DonatoPastore, Dep. of Agricultural, Food and Environmental Sciences – University of Foggia, Italy, Tel.: +39(0)881589427/32; Fax: +39(0)881587108; E-mail: d.pastore@unifg.it

Received date: October 15, 2014; Accepted date: October 16, 2014; Published date: October 23, 2014

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Editorial

Mitochondria play a central role in all aerobic eukaryotic cells, being the site of respiration and synthesis of ATP via Oxidative Phosphorylation (OXPHOS). According to the chemo-osmotic theory, this occurs bycoupling substrate oxidation by the respiratory chain with proton ejection in the intermembrane space [1,2], thus generatingan electrical membrane potential (ΔΨ), which is the main component in plant mitochondria [3], and the proton gradient (ΔpH), both components contributing to the overall Proton Motive Force (pmf) utilized for ATP synthesis [4]. Interestingly, pmf represents the driving force also for many protein-mediated transport activities across the Inner Mitochondrial Membrane (imm). In fact, the flux of hydrophilic solutes across the imm, that represents a diffusion barrier generally impermeable to charged and polar molecules, is facilitated and regulated by a large number of specific hydrophilic transport proteins, including carriers and ion-conducting channels. Metabolites can thus easily cross the membrane, maintaining complex metabolic demands. In fact, mitochondria provide, through the tricarboxylic acid cycle, reducing equivalents for other compartments and carbon skeletons for biosynthesis of nucleotides, amino acids, fatty acids and vitamin cofactors, nitrogen assimilation, photosynthesis, photoregulation, and carbon and nitrogen storage compounds during seed germination [3,5-7].

In a previous editorial of this Journal, it was reported that plant mitochondria may apparently disobey classical chemiosmosis [8], in fact, Durum Wheat Mitochondria (DWM) isolated in vitro were found to be able to regularly oxidize succinate and accomplish ATP synthesis even though they had lostpmf. This occurs in a KCl medium able to inhibit of ROS production, according to a feedback mechanism found so far observed only under in vitro conditions, these findings have a relevant physiological importance since mitochondria depolarization is not unusual. Determination of the in vitrodynamics of individual mitochondrial membrane potentials in roots has demonstrated that plant mitochondria undergo sporadic and rapid cycles of partial dissipation and restoration of ΔΨ [12]. Moreover, plant mitochondria possess some dissipative systems able to strongly lower pmf, such as the above mentioned PmitoKATP as well as the Plant Uncoupling Protein (PUCP) able to lower pmf. In this regard, DWM which possess very active PmitoKATP and PUCP may help to shed some light on the enigmatic mechanism of metabolite transport in plant mitochondria. This, either lacking or having low pmf under in vivo conditions and to highlight the physiological conditions in which this occurs.

PmitoKATP, PUCP and Control of ΔΨ and Reactive Oxygen Species (ROS) Production

The PmitoKATP catalyzes the electrogenic uniport of K+ across the inner mitochondrial membrane towards the matrix. The cooperation between PmitoKATP and the K+/H+ anti-porter, which is also very active in DWM, allows the operation of a K+ cycle that may induce proton re-entry in the mitochondrial matrix [9,13]. This, as stated above, may dissipate ΔΨ in isolated mitochondria, thus potentially uncoupling mitochondria. Similarly, the PUCP catalyzes, in the presence of Free Fatty Acids (FFAs), re-entry of protons into the matrix; this may completely collapse respiring mitochondria, thus uncoupling DWM [14].

Interestingly, the functioning of both systems is enhanced by ROS, that is known to increase as a consequence of plant exposure to abiotic stress [15]. In practice, a ROS-induced activation of PmitoKATP and PUCP occurs under stress, able to dissipate ΔΨ and ΔpH up to a complete collapse; this strong pmf decrease induces in turn an inhibition of ROS production, according to a feed-back mechanism [13,15]. According to another pathway of regulation, under abiotic stress an increase of mitochondrial FFA content occurs in DWM, due to the activation of a mitochondrial Phospholipase A2 (PLA2) [16]; FFA increase activates both dissipative systems, moreover, the acylCoA deriving from FFAs stimulate K+ transport via PmitoKATP [17]; once again, activation of PmitoKATP and PUCP may lower ROS production [11]. So, DWM may dissipate pmf in order to avoid ROS over production and cellular oxidative stress. On the other hand, to maintain mitochondrial function, it is important to preserve metabolite transports though specific energy-dependent carriers are impaired under low or absent pmf. This may be obtained as hypothesized below.

Plant Inner Membrane Anion Channel (PIMAC), Carriers and Anion Transport in Energized and De-Energized DWM

A PIMAC exists in DWM able to transport Pi, dicarboxylates, oxo-dicarboxylates and tri-carboxylates; it is inhibited by ATP, FFAs (but...
not acyl-CoAs) and by high ΔΨ, but it is insensitive to ROS [18]. So, when ΔΨ and ΔpH generated by the respiratory chain are high and ATP is synthesized at a high rate, PIMAC is expected to be inactive. Under these conditions, metabolically relevant anions are transported by the specific energy-dependent carriers: cytosolic ADP exchanges with matrix ATP on the ADP/ATP Carrier (AAC); Pi uptake occurs in cytosol with H+ using the Pi Carrier (PiC); in turn, matrix Pi may exchange for Di-Carboxylate (Dic) on the dicarboxylate carrier (DIC), and Dic may exchange for either oxodicarboxylate or tri-caboxylate on the Di-Carboxylate-Tri-Carboxylate Carrier (DTC) [19].

A different situation is expected under stress when ΔΨ and ΔpH are lowered by activation of PmitoKATP and PUCP: classical carriers may be impaired by loss of pmf as well as by ROS [20], moreover ATP is synthesized at lower rate, so PIMAC activity may be unblocked. As a consequence, passive anion transport across the inner mitochondrial membrane may shift from the energy-dependent anion carriers toward the electrophoretic flux through the PIMAC. Results from in vitro studies on DWM are in line with this picture [10,11,19], with the notable exception of AAC function. Both ADP and ATP are not transported by PIMAC, moreover, in de-energized DWM the ADP/ATP exchange is inhibited by oligomycin, a powerful AAC inhibitor, thus suggesting AAC operation in the absence of measurable ΔΨ, the driving force for AAC. This enigmatic finding is still unsolved, lacking to date any possible explanation.

In conclusion, first information is now available about the interrelationship between transport systems and low pmf in plant mitochondria. In particular, under stress conditions PmitoKATP and PUCP may actively dissipate pmf to act against oxidative stress, but, unexpectedly, de-energization of mitochondria may not necessarily impair metabolite transport, as demonstrated in in vitro experiments. Probably, PmitoKATP may either induce partial dissipation in vivo, as a result of a balance between positive and negative modulators or promote cycles of partial dissipation and restoration of ΔΨ. Under dissipation cycles, cooperation between PIMAC and classical carriers might ensure mitochondrial metabolism in vivo, moreover the AAC might show unexpected activity also in the absence of ΔΨ.

Certainly, further studies are required to fully understand these novel behaviors, in fact for the maintenance of both the basic energy function and the complex metabolic network connecting plant mitochondria with other cell compartments, a rapid and controlled active movement of solutes in and out of mitochondrion is required whatever the available pmf.

References


