Bioenergetics: Open Access
ISSN: 2167-7662

Pastore, Bioenergetics 2014, 3:3
DOI: 10.4172/2167-7662.1000e121

Editorial Open Access

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

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 hydrophobic transport proteins, including carriers and ion-conducting channelsthat meet the complex metabolic demands. In fact, mitochondria provide, through the tri-carboxylic acid cycle, reducing equivalents for other compartments and carbon skeletons for biosynthesis of nucleotides, amino acids, fatty acids and vitamin cofactors, nitrogen assimilation, photorespiration, photosynthesis in C4 and CAM plants, utilization of 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 activate the ATP-sensitive mitochondrial potassium channel (PmitoKATP); under this particular condition, the massive K+ uptake by DWM may strongly decreaseΔΨ without compensation due to ΔpH increase, thus collapsing pmf [9,10]. A possible mechanism explaining this unexpected ATP synthesis has been recently reported [11], but another point remains still unsolved regarding the transport of metabolites: to synthesize ATP via OXPHOS it is necessary to carry out uptake of succinate, Inorganic Phosphate (Pi) and ADP as well as to release the newly synthesized ATPoutside DWM. Since the force driving these transports should derive from ΔΨ and ΔpH, the question arises about how these movements of metabolites may be possiblein the absence of pmf in this *in vitro* model system.

Although theabove reported enigmatic behavior of DWM has been so far observed only under *in vitro* conditions, these findings may have a relevant physiological importance since mitochondria depolarization is not unusual. Determination of the *in vivo*dynamics of individual mitochondrial membrane potentials in roots has demonstrated that plant mitochondria undergo sporadic and rapid cycles of partial dissipation and restoration ofΔΨ [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 either lacking or having lowpmf under *in vivo* conditions and to highlight the physiological conditions in which this occurs.

PmitoK$_{ATP}$, PUCP and Control of ΔΨ and Reactive Oxygen Species (ROS) Production

The PmitoK$_{ATP}$ catalyzes the electrophoretic uniport of K+ across the inner mitochondrial membrane towards the matrix. The cooperation between PmitoK$_{ATP}$ 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 dissipateΔΨ 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 it is knownto increase as a consequence of plant exposure to abiotic stress [15]. In practice, a ROS-induced activation of PmitoK$_{ATP}$ 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 acylCoAs deriving from FFAs stimulate K+ transport via PmitoK$_{ATP}$ [17]; once again, activation of PmitoK$_{ATP}$ and PUCP may lowerROS 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, di-carboxylates, 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 energydependent carriers: cytosolic ADP exchanges with matrix ATP on the ADP/ATP Carrier (AAC); Pi uptake occurs in symport with H+ using the Pi Carrier (PiC); in turn, matrix Pi may exchange for Di-Carboxylate (Dic) on the di-carboxylate carrier (DIC), and Dicmay exchange for either oxo-dicarboxylate or tri-caboxylate on the Di-Carboxylate-Tri-Carboxylate Carrier (DTC) [19].

A different situation is expected under stress whenΔΨ andΔpH are lowered by activation of PmitoK$_{ATP}$ and PUCP: classical carriers may be impaired by loss of pmfas well as by ROS [20], moreoverATP is synthesized at lower rate, so PIMACactivity may be unlocked. 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 DWMthe ADP/ATP exchange is inhibited by oligomycin, a powerful AAC inhibitor, thus suggesting AAC operation in the absence of measurableΔΨ, the driving force for AAC. This enigmatic finding is still unsolved, lacking to date any possibleexplanation.

In conclusion, first information is now available about the interrelationship between transport systems and low pmf in plant mitochondria. In particular, under stress conditions PmitoK$_{ATP}$ 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, PmitoK$_{ATP}$ 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

1. MITCHELL P (1961) Coupling of phosphorylation to electron and hydrogen transfer by a chemi-osmotic type of mechanism. Nature 191: 144-148.
2. Mitchell P (1966) Chemiosmotic coupling in oxidative and photosynthetic phosphorylation. Biol Rev CambPhilosSoc 41: 445-502.
3. Douce R (1985) Structure, Function, and Biogenesis. In Mitochondria in Higher Plants. Academic Press: Orlando, FL, USA.
4. Nicholls DG, Ferguson SJ (1992) The chemiosmotic proton circuit. In Bioenergetics 2, pp 82-87. Academic press limited, London.
5. Picault N, Hodges M, Palmieri L, Palmieri F (2004) The growing family of mitochondrial carriers in Arabidopsis. Trends Plant Sci 9: 138-146.
6. Linka N, Weber AP (2010) Intracellular metabolite transporters in plants. Mol Plant 3: 21-53.
7. Palmieri F, Pierri CL, De Grassi A, Nunes-Nesi A, Fernie AR (2011) Evolution, structure and function of mitochondrial carriers: a review with new insights. Plant J 66: 161-181.
8. Pastore D (2012) Fifty Years of Chemiosmotic Theory – Many Lights and Some Shade. Bioenergetics 1:3, http://dx.doi.org/10.4172/2167-7662.1000e107.
9. Pastore D, Stoppelli MC, Di Fonzo N, Passarella S (1999) The existence of the K(+) channel in plant mitochondria. J BiolChem 274: 26683-26690.
10. Trono D, Soccio M, Laus MN, Pastore D (2011) Potassium channel-oxidative phosphorylation relationship in durum wheat mitochondria from control and hyperosmotic-stressed seedlings. Plant Cell Environ 34: 2093-2108.
11. Pastore D, Soccio M, Laus MN, Trono D (2013) The uniqueness of the plant mitochondrial potassium channel. BMB Rep 46: 391-397.
12. Schwarzländer M, Logan DC, Johnston IG, Jones NS, Meyer AJ, et al. (2012) Pulsing of membrane potential in individual mitochondria: a stress-induced mechanism to regulate respiratory bioenergetics in Arabidopsis. Plant Cell 24: 1188-1201.
13. Pastore D, Trono D, Laus MN, Di Fonzo N, Flagella Z (2007) Possible plant mitochondria involvement in cell adaptation to drought stress. A case study: durum wheat mitochondria. J Exp Bot 58: 195-210.
14. Pastore D, Fratianni A, Di Pede S, Passarella S (2000) Effects of fatty acids, nucleotides and reactive oxygen species on durum wheat mitochondria. FEBS Lett 470: 88-92.
15. Trono D, Flagella Z, Laus MN, Di Fonzo N, Pastore D (2004) The uncouplingprotein and the potassium channel are activated by hyperosmotic stress in mitochondria from durum wheat seedlings. Plant Cell Environ 27: 437-448.
16. Trono D, Soccio M, Laus MN, Pastore D (2013) The existence of phospholipase A(2) activity in plant mitochondria and its activation by hyperosmotic stress in durum wheat (Triticum durum Desf.). Plant Sci 199-200: 91-102.
17. Laus MN, Soccio M, Trono D, Liberatore MT, Pastore D (2011) Activation of the plant mitochondrial potassium channel by free fatty acids and acyl-CoA esters: a possible defence mechanism in the response to hyperosmotic stress. J Exp Bot 62: 141-154.
18. Laus MN, Soccio M, Trono D, Cattivelli L, Pastore D (2008) Plant inner membrane anion channel (PIMAC) function in plant mitochondria. Plant Cell Physiol 49: 1039-1055.
19. Trono D, Laus MN, Soccio M, Pastore D (2014) Transport pathways--proton motive force interrelationship in durum wheat mitochondria. Int J MolSci 15: 8186-8215.
20. Pastore D, Laus MN, Di Fonzo N, Passarella S (2002) Reactive oxygen species inhibit the succinate oxidation-supported generation of membrane potential in wheat mitochondria. FEBS Lett 516: 15-19.