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[BioMedical] Academia Sinica Biomedical Scientists Find Transporter of Vitamin C in Mitochondria That Aids Protection Against Oxidative Stress

[BioMedical] Academia Sinica Biomedical Scientists Find Transporter of Vitamin C in Mitochondria That Aids Protection Against Oxidative Stress (Chinese Version)

Academia Sinica Newsletter (2010/08/19) Researchers from the Institute of Biomedical Sciences, Academia Sinica, have identified GLUT10, a member of glucose transporter family, actually functions as a transporter of oxidized vitamin C to protect cells against oxidative stress. The finding helps increase understanding of the cause of the inherited connective tissue disorder arterial tortousity syndrome. The research was published online in the journal Human Molecular Genetics on July 24, 2010, and has been highlighted as a “must read” paper in the biological sciences by the influential Faculty of 1000 Biology online research service.

Oxidative stress is a cellular imbalance between the production of chemically reactive molecules containing oxygen (known as “reactive oxygen species”, ROS) and the cell’s ability to detoxify and repair the damage. Oxidative stress is involved in many human diseases and is thought to play a part in aging.

The findings of the current research provide an explanation for long-standing questions about how vitamin C enters mitochondria, a vital cell organelle. In most cells, mitochondria are thought to be the major source of ROS. Mitochondria are also known to incorporate and recycle vitamin C. Up until now, however, the molecular mechanisms underlying vitamin C uptake by mitochondria have been poorly understood.

Dr. Yi-Ching LEE a postdoctor fellow working with Dr. Yuan-Tsong CHEN found that GLUT10 is predominantly in the mitochondria of smooth muscle cells and insulin-stimulated fat cells. They also found that mitochondrial GLUT10 facilitates transport of oxidized vitamin C (dehydroascorbic acid, DHA) into mitochondria, and also increases its cellular uptake, which in turn protects cells against oxidative stress.

These findings provide a mechanism to explain the vitamin C in mitochondria and show how abnormalities in GLUT10 may lead to the arterial abnormalities found in arterial tortousity syndrome. These results also provide new insights into the relationship between GLUT10 and type 2 diabetes and reinforce the importance of vitamin C and ROS in degenerative diseases.

The article entitled “Mitochondrial GLUT10 facilitates dehydroascorbic acid import and protects cells against oxidative stress: mechanistic insight into arterial tortuosity syndrome” can be found at: http://hmg.oxfordjournals.org/cgi/content/full/ddq286v2?view=long&pmid=20639396. The complete list of authors is: Yi-Ching Lee, Hsun-Yi Huang, Chia-Jung Chang, Chao-Hung Cheng and Yuan-Tsong Chen

The “Faculty of 1000 Biology” evaluation can be found at: http://f1000biology.com/article/id/4361967/evaluation

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