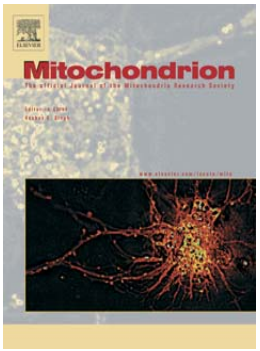

Evidence for genistein as a mitochondriotropic molecule

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PII: S1567-7249(16)30047-2
DOI: doi: 10.1016/j.mito.2016.05.005
Reference: MITOCH 1088

To appear in: *Mitochondrion*

Received date: 2 February 2016
Revised date: 13 May 2016
Accepted date: 16 May 2016

Please cite this article as: de Oliveira, Marcos Roberto, Evidence for genistein as a mitochondriotropic molecule, *Mitochondrion* (2016), doi: 10.1016/j.mito.2016.05.005

Evidence for genistein as a mitochondriotropic molecule

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Abstract

Genistein (4',5,7-trihydroxyisoflavone; $C_{15}H_{10}O_5$), an isoflavone, has been investigated as an anti-cancer agent due to its ability to trigger cell death (both intrinsic and extrinsic apoptotic pathways) in different cancer cells *in vitro* and *in vivo*. Furthermore, genistein has been viewed as a mitochondriotropic molecule due to the direct effects this isoflavone induces in mitochondria, such as modulation of enzymatic activity of components of the oxidative phosphorylation system. Apoptosis triggering may also be mediated by genistein through activation of the mitochondria-dependent pathway by a mechanism associated with mitochondrial dysfunction (*i.e.*, disruption of the mitochondrial membrane potential - MMP, release of cytochrome c, activation of the apoptosome, among others). Efforts have been made in order to elucidate how genistein coordinate these biochemical phenomena. Nonetheless, some areas of the mitochondria-associated research (mitochondrial biogenesis, redox biology of mitochondria, and mitochondria-associated bioenergetic parameters) need to be explored regarding the role of genistein as a mitochondria-targeted agent. This is a pharmacologically relevant issue due to the possibility of using genistein as a mitochondria-targeted drug in cases of cancer, neurodegeneration, cardiovascular, and endocrine disease, for example. The present review aims to describe, compare, and discuss relevant data about the effects of genistein upon mitochondria.

Keywords: genistein; mitochondria; antioxidant; mitochondrial biogenesis; apoptosis