Rejuvenate Your Cells by Growing New MITOCHONDRIA

By Kirk Stokel

Mitochondrial dysfunction is a primary cause of age-related decline.[1-7] In a revealing study, a team of researchers showed that muscle tissue of a **90**-year-old man contained **95%** damaged **mitochondria** compared to almost no damage in that of a **5**-year-old.[8]

When one looks at the boundless energy of a child compared to an elderly person, the devastating impact of *mitochondrial degradation* become instantly apparent.

A myriad of recent scientific reports link defective and deficient ***mitochondria*** to virtually all degenerative diseases including Alzheimer's, type 2 diabetes, heart failure, and cancer.[9-13]

Up until now, the best we could do was protect and improve the ***function*** of existing mitochondria using nutrients like **L-carnitine**, **lipoic acid**, and **coenzyme Q10**.

In an unprecedented breakthrough, a compound has been discovered that promotes the growth of ***new*** mitochondria structures within aging cells![14]

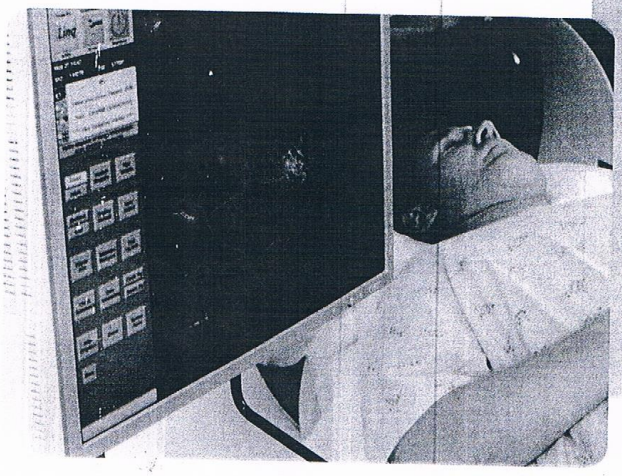
In this article, you will discover how this novel compound can help ***reverse*** cellular aging by activating **genes** that stimulate ***mitochondrial biogenesis***, which means **the generation of new mitochondria.** >>

especially effective in neutralizing the ubiquitous ***superoxide*** and ***hydroxyl*** radicals.[36] According to the most recent research, "PQQ is **30 to 5,000 times** more efficient in sustaining redox cycling . . . than other common [antioxidant compounds], e.g. ascorbic acid."[37]

Protection Against Brain Aging

PQQ has been shown to optimize function of the entire central nervous system. It *reverses* cognitive impairment caused by chronic oxidative stress in pre-clinical models, improving performance on memory tests.[40] It has also been shown to safeguard a gene involved in the development of Parkinson's disease (called DJ-1) from *self-oxidation*—an early step in the onset of Parkinson's.[41]

Reactive *nitrogen* species (RNS), like reactive *oxygen* species, impose severe stresses on damaged neurons.[42] They arise spontaneously following stroke and spinal cord injuries and have been shown to account for a substantial proportion of subsequent long-term neurological damage. PQQ directly *suppresses* RNS in experimentally induced strokes.[43] It also provides additional protection by blocking gene expression of *inducible* nitric oxide synthase, a major source of RNS, following spinal cord injury.[44]



How PQQ Generates New Mitochondria

Mitochondrial biogenesis can be defined as the growth and division of pre-existing mitochondria. This phenomenon is not only accompanied by increased mitochondria numbers, but also their size and mass.

Mitochondrial biogenesis requires the *coordinated* synthesis and import of 1,000-1,500 proteins where they facilitate the production of healthy new mitochondria.

Mitochondrial biogenesis occurs through the combined effects of **genes** activated by **PQQ** via the following three mechanisms:

- **PQQ** increases expression of peroxisome proliferator-activated receptor gamma coactivator 1-alpha or **PGC-1α**. PGC-1α is a "master regulator" gene that mobilizes your cells' response to various external triggers. It directly activates genes that boost mitochondrial and cellular respiration, growth, and reproduction. Its capacity to modulate cellular metabolism at the genetic level favorably affects **blood pressure**, **cholesterol** and **triglyceride** breakdown, and the onset of **obesity**.[29]

- **PQQ** activates a signaling protein known as cAMP-response element-binding protein or **CREB**. The CREB gene plays a pivotal role in embryonic development and growth. It also beneficially interacts with *histones*, molecular compounds shown to protect and repair cellular DNA. CREB *also* stimulates the growth of new mitochondria.[30]

- **PQQ** regulates a recently discovered gene called **DJ-1**. As with PGC-1α and CREB, DJ-1 is intrinsically involved in cell function and survival. It has been shown to **prevent cell death** by combating intensive antioxidant stress and is of particular importance to brain health and function. DJ-1 damage and mutation have been conclusively linked to the onset of **Parkinson's disease** and other neurological disorders.[31-34]