

An Introduction to Coenzyme Q10

Every living cell (human, animal or plant) contains Q10: its other name is ubiquinone to reflect its ubiquitous distribution. It is essential for energy production, acting as a cofactor of the electron transport chain. It is also an antioxidant in mitochondrial and lipid membranes. Q10 has been shown to control genes relating to inflammation and fat metabolism.

Q10 is safe to take. There are no known toxic effects, and Q10 cannot be overdosed.

Most research has focussed on the role of coenzyme Q10 in improving heart function, since heart cells are the hardest working cells in the body and Q10 can boost cellular energy. 420 patients with heart failure received Q10 or placebo alongside their conventional heart failure medicine for 2 years in a [Danish study](#). Cardiac-related deaths were much less likely in those receiving Q10.

According to scientists, Q10 might also cure AIDS, Alzheimer's, schizophrenia, [multiple sclerosis](#) and lupus, slow ageing and accelerate weight loss.

Parkinson's disease (PD)

PD is characterised by a deficiency of dopaminergic neurons in the substantia nigra. Most current drug therapies act to increase dopamine levels, by adding it, mimicking it, boosting it or 'regrowing' it (see my article on [current drug therapies](#)).

It is now generally accepted that the loss of dopaminergic neuron function is due to mitochondrial dysfunction (Chaturvedi and Beal, 2013). Specifically, mutations in the Parkin and PINK1 proteins of PD patients prevent identification and tagging of dysfunctional mitochondria for lysosomal degradation. Their subsequent accumulation results in oxidative stress: coenzyme Q10 is a powerful **antioxidant** so could remove this burden, preserving neurons. Coenzyme Q10 may also exert neuroprotection by affecting **gene expression** or by boosting **cellular energy** levels.

Coenzyme Q10 levels are known to decrease in the brain with age, with the substantia nigra having the lowest Q10 levels. There is, therefore, a robust scientific rationale for testing this agent as a potential neuroprotective therapy.

Chaturvedi RK, Beal MF (2013) Mitochondria targeted therapeutic approaches in Parkinson's and Huntington's diseases. *Mol Cell NeuroSci* **55**: 101-14

Studies supporting Coenzyme Q10 in the treatment of PD

- Animal studies
- 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) is a known neurotoxin, reducing striatal dopamine concentrations (Beal et al, 1998). One year old mice treated with MPTP received a standard diet or a diet supplemented with Coenzyme Q10 for 5 weeks. Dopamine

concentrations were less severely depleted in the mice receiving Coenzyme Q10 (Beal et al, 1998)

- In a mouse model of Huntington’s disease (thought to also be due to mitochondrial dysfunction) (Chaturvedi and Beal, 2013), administration of Coenzyme Q10 delayed the development of motor deficit and cerebral atrophy (Ferrante et al, 2002)
- Human studies
 - The QE2 Study (Schults et al, 2002). A randomized, double-blind, placebo-controlled, multicentre phase II study of Coenzyme Q10 in 80 early treated PD patients. This study tested the administration of 300, 600 and 1200 mg Coenzyme Q10 daily for up to 16 months. Subjects also received Vitamin E. Patients receiving Coenzyme Q10 showed up to 44% less functional decline than those receiving placebo (Schults et al, 2002)

Beal MF, Matthews RT, Tieleman A, Schults CW (1998) Coenzyme Q10 attenuates the 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP)induced loss of striatal dopamine and dopaminergic axons in aged mice. *Brain Res* **783**: 109-14

Ferrante RJ, Andreassen OA, Dedeoglu A, Ferrante KL, Jenkins BG, Hersch SM, Beal MF (2002) Therapeutic effects of coenzyme Q10 and remacemide in transgenic mouse models of huntington’s disease. *J Neurosci* **22**: 1592-9

Schults CW, Oakes D, Kieburtz K et al (2002) Effects of Coenzyme Q10 in Early Parkinson disease. *Arch Neurol* **59**: 1541-50

Studies in which no benefit of Coenzyme Q10 was observed for PD

- An open label phase-I pilot trial in 15 PD patients showed no effect of Coenzyme Q10 on motor function (Schults et al, 1998)
- A phase III multicentre, randomized, placebo-controlled, double-blind trial (QE3) was [halted prematurely due to futility](#). 600 patients were receiving Coenzyme Q10 at doses of 1200mg and 2400mg daily in combination with Vitamin E (The Parkinson Study Group QE3 Investigators, 2014). No difference was observed between the active treatment and placebo group. “This unfortunately is strong data that CoQ10 is not useful as a neuroprotective strategy for PD” (Chaturvedi and Beal, 2013)

Schults CW, Beal MF, Fontaine D, Nakano K, Haas RH (1998) Absorption, tolerability, and effects on mitochondrial activity of oral coenzyme Q10 in parkinsonian patients. *Neurology* **50**: 793-5

The Parkinson Study Group QE3 Investigators (2014) A Randomized Clinical Trial of High-Dosage Coenzyme Q10 in Early Parkinson Disease: No Evidence of Benefit. *JAMA Neurol* **71(5)**: 543-552

Future

The failure of coenzyme Q10 in PD may be explained by a number of factors. Firstly, are mitochondria the correct therapeutic target? The ability of Coenzyme Q10 to cross into the brain is also relevant.

Other more brain-penetrant forms of Coenzyme Q10 (eg, MitoQ) have, however, also failed in an early clinical trial in PD (Snow et al, 2010).

Dysfunction of gastrointestinal motility is a common symptom of Parkinson's and could affect drug absorption, confounding trial results (Cowan, 2016).

Future studies might involve testing different dosages of Coenzyme Q10, and different formulations (taking into account gastrointestinal absorption and ability to reach the brain).

More promise might be seen with Coenzyme Q10 if it is administered in the very early stages of Parkinson's. This will need clinicians to be vigilant to non motor signs and symptoms, since these often manifest years before the typical motor signs (Cowan, 2016)

Cowan H (2016) The lesser known symptoms of Parkinsons:

<http://www.readersdigest.co.uk/health/health-centre/lesser-known-symptoms-parkinsons>

Snow BJ, Rolfe FL, Lockhart MM et al (2010) A Double-Blind, Placebo-Controlled Study to Assess the Mitochondria-Targeted Antioxidant MitoQ as a Disease-Modifying Therapy in Parkinson's Disease. *Movement Disorders* **25(11)**: 1670-4