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Co-Enzyme Q10 as Potent Antitoxic Agent in Different Biological Models

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Overview

Coenzyme Q10 (CoQ10), also known as ubiquinone, acts as electron and proton carrier in mitochondria and functions as an antioxidant in its reduced form (ubiquinol) [1]. It is a component of the electron transport chain and participates in aerobic cellular respiration, which generates energy in the form of ATP. Chemical structure of CoQ10 has been depicted in Figure 1. CoQ10 helps to convert food into energy and is found in almost every cell in the body. Oily fish, organ meats, and whole grains are good source of CoQ10. It is available as a supplement in the market in various forms including capsules, sprays and tablets. Some researchers consider that CoQ10 may help with heart-related ailments as it can enhance energy production in cells and prevent blood clotting. A number of studies exploring the effects of CoQ10 have been published [2-9]. In the present paper, literature related to beneficial role of CoQ10 has been reviewed so as to emphasize the potency of CoQ10 for its antitoxic nature (Figure 1).

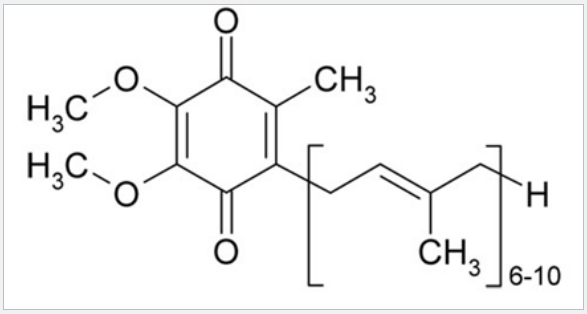

Figure 1: Chemical structure of Coenzyme Q10.

Antitoxic nature of CoQ10

CoQ10 has been reported to aid in different disease pathologies. Figure 2 depicts different health ailments for which CoQ10 supplementation is used. The comparative biochemical activities of CoQ10 and carnitine were reported to have protective synergistic effect in preventing the hyperbaric oxygen toxicity in mice [2]. Pretreatment for four days with CoQ10 was found to reduce the acute toxicity in mice treated with adriamycin (doxorubicin). In two sequential protocols, adriamycin allowed only 36 and 42% survival whereas pretreatment with CoQ10 allowed 80 and 86% survival ($p<0.05$) [3]. Similarly, administration of adriamycin was found to increase the interval of the electrocardiographic QRS traces in rats ($P<0.01$). A 7 day administration of coenzyme Q10 to such adriamycin-treated rats allowed restoration of a normal QRS complex [7]. Amitriptyline is a very frequently prescribed antidepressant drug having toxic systemic effects on cardiovascular system, autonomic and central nervous systems. In a study, the cytotoxic effects of amitriptyline treatment on cultured primary human fibroblasts and zebrafish embryos and antitoxic nature of CoQ10 was examined. Mitochondrial dysfunction in amitriptyline treatment was characterized by reduced expression levels of mitochondrial proteins and CoQ10, decreased NADH: cytochrome C reductase activity and a drop in mitochondrial membrane potential. CoQ10 supplementation was reported to attenuate ROS production, lipid peroxidation, mitochondrial dysfunction and cell death [4] (Figure 2).

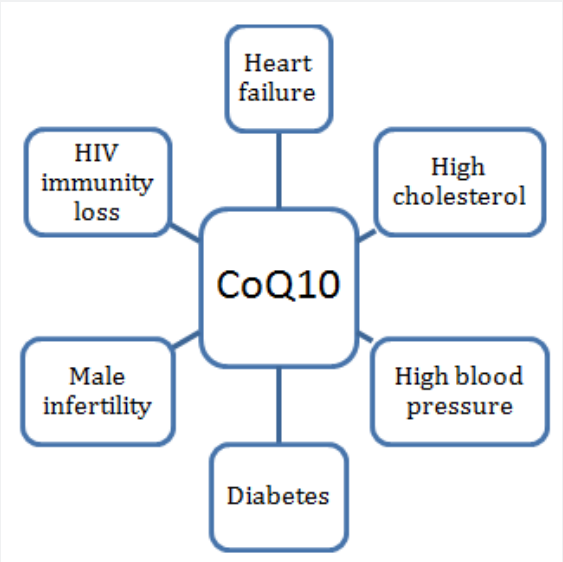

Figure 2: Health ailments for which CoQ10 supplementation is used.

Statins are potent cholesterol-lowering drugs that can have serious adverse effects on the muscles and liver [5,10]. Previous studies indicated that myotoxicity caused by statins may be linked to impairment of mitochondrial functions [10]. A study by Eghbal et al. [5] evaluated the efficacy of CoQ10 against cytotoxicity induced by atorvastatin, simvastatin and lovastatin in isolated rat hepatocytes. Various parameters were observed including cell death, reactive oxygen species formation, lipid peroxidation, mitochondrial membrane potential and cellular reduced and oxidized glutathione content. Pretreatment with CoQ10 was found to be effective in reducing the toxic effects of statins in rat hepatocytes suggesting the protective role of CoQ10 against hepatocytes. In another study, co-incubation of rat skeletal muscle samples with 1 mM L-carnitine, 100 μM mevalonate or 10 μM CoQ10 abolished simvastatin effects on both mitochondrial glutamate/malate-supported respiration and lactate release [10]. Similarly, doxorubicin (DOX) is reported to cause testicular toxicity. Simultaneous administration of CoQ10 with DOX was found to significantly restore testicular oxidative stress parameters and the distorted histopathological conditions. It also reduced the up-regulation of caspase 3 caused by DOX, and increased P-glycoprotein (PGP) expression (PGP is an efflux transporter which extrudes DOX from the testis) as a mechanism for gonadal protection [6]. Another study also evaluated the protective effects of coenzyme CoQ10 DOX-induced toxicity. Administration of CoQ10 resulted in a significant improvement of hepatic and renal functional parameters and an improvement in alpha-smooth muscle actin and proliferating cell nuclear antigen [11].

Hepatoprotective effect of coenzyme Q10 in rats with acetaminophen toxicity has also been reported [8]. The potential protective effect of CoQ10 against acute liver injury induced by a single dose of acetaminophen (700 mg/kg, p.o.) was investigated in rats. 10 mg/kg CoQ10 (i.p.) was administered at 1 and 12 h following acetaminophen administration. CoQ10 was found to significantly reduce levels of serum aminotransferases and lipid peroxidation. It also reduced the elevations of tumor necrosis factor-alpha and nitric oxide. CoQ10 also attenuated the decline of selenium and zinc ions in liver tissue resulting from acetaminophen administration [8]. CoQ10 increases brain mitochondrial concentration and exerts neuroprotective effects [12]. CoQ10 had no effect on cell death induced by either beta amyloid peptides (Abeta) or oxygen glucose deprivation (OGD) but an increased cell survival in the Abeta + OGD group was reported in SHSY5Y neuronal cells ($P<0.05$) [12]. Defects in mitochondrial energy metabolism due to respiratory chain disorders lead to a decrease in mitochondrial membrane potential which induces apoptosis. Menke et al. [13] reported a reduced toxicity of rotenone by CoQ10 in neuronal cultures by preserving the mitochondrial membrane potential. In another study, pretreatment for 4 days with 10 mg/kg CoQ10 reduced the acute toxicity of 0.5 mg/kg anthramycin in mice [14]. Only 40% of control mice survived after 27 days of anthramycin treatment as compared to those pretreated with CoQ10 (73% survival) revealing a protective effect of CoQ10. Pretreatment of Swiss Webster mice with CoQ10 was reported to markedly reduce the lethality of anthramycin as well as its ability to decrease ventricular weights [15].

Chlorpyrifos is an organophosphorus pesticide that induces oxidative stress through the production of free radicals and depletes intracellular antioxidant reserves. In a study, cytotoxicity of chlorpyrifos in human peripheral blood lymphocytes after 72-h exposure was determined. CoQ10 opposed toxicity of chlorpyrifos characterized by increased total antioxidant power and total thiol molecules; improvement of AChE activity; and reduced lipid peroxidation, myeloperoxidase, TNF-alpha, and apoptosis [9]. Microcystins are a group of cyclic heptapeptide toxins produced by cyanobacteria. In a study, the amelioratory effect of coenzyme Q10 on microcystin-LR induced toxicity was investigated in mice. When microcystin-LR treated mice (10 μg/kg bw/day, i.p.) were co-administered CoQ10 (10 mg/kg bw/day, i.m.) for 14 days, it was observed that CoQ10 ameliorated microcystin-LR induced toxicity via modulation of glycolytic-oxidative-nitrosative stress pathway [16]. Similarly, various other studies [17-19] report the protective role of CoQ10 against different types of induced toxicities.

Summary

Coenzyme Q10 is a fat soluble substance which resembles vitamin and functions as potent antioxidant. After reviewing the literature on ameliorative and protective nature of CoQ10, it was revealed that CoQ10 is potent to nullify the toxicities induced by various chemical compounds including adriamycin, microcystins, chlorpyrifos, anthramycin, acetaminophen and statins. CoQ10 has been explored from decades for its antitoxic nature and much has to be revealed in different fields of biological research. Further studies are recommended so as to explore the efficacy of CoQ10 in new and innovative areas of medical sciences.

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