In this issue of our newsletter, we address the many significant health benefits of Coenzyme Q10 and some recent published documentation regarding the benefits of Co-Q10 and its neuroprotective effects for the prevention of Alzheimer’s (which could be due to its microglia inhibitory mechanism).

Related to this, there is considerable evidence showing that mitochondrial dysfunction and oxidative damage play a key role in the development of neurodegenerative disease, including both Alzheimer’s disease (AD) and Parkinson’s disease (PD). This relationship has been known for many years. Further detail on this topic is covered in the included article from the Life Enhancement Magazine.

First, let’s document a quick review of some of the many benefits of Co-Q10:

- Activates AMPK
- Use for increased oxygenation need in cardiac stress or insufficiency
- Immune dysfunction
- Chronic Fatigue Syndrome
- High blood pressure
- Viral infections
- Periodontal disease
- Multiple Sclerosis (MS)
- Cell membrane stabilization
- Inhibition of inflammation by modulation of NF-kappaB
- Reduces blood pressure in hypertensive patients as effectively as 1-2 drug combination treatment
- Congestive Heart Failure
- Chronic Renal Failure
- Anti-allergy benefits
- CoQ:10 deficiency is common in migraines and supplementation greatly reduces migraine attacks and Headaches
- After heart attack
- Heart failure (HF)
- High blood pressure
- High cholesterol
- Heart damage caused by chemotherapy
- Heart surgery
- Gum (Periodontal) disease
- Help produce more cellular energy
- Boost your heart health
- Act as an antioxidant for protection from free radicals
- Helps reduce the signs of normal aging
- Helps maintain blood pressure levels within the normal range
- Provides a boost to the immune system
- Supports the nervous system
Preliminary clinical studies also suggest that CoQ10 may:

- Improve immune function in people with HIV or AIDS
- Increase sperm motility, improving male fertility
- Be used as part of the treatment for Parkinson’s disease
- Improve exercise ability in people with angina
- Help prevent migraines
- Protect against hearing loss
- Improve learning
- Extend life span
- Prevent cachexia
- Assist muscle regeneration
- Protect against surgical stress
- Promote male fertility
- Protect against senile plaques
- Impede diabetic complications
- Lower triglycerides
- Benefits in cancer therapy
- Slowing early Macular Degeneration
- Guarding the brain after cardiac arrest
- Preventing the onset of migraine
- Slowing Neurodegenerative Disease progression

Of particular note are the significant benefits of Co-Q10 supplementation for cardiovascular conditions (especially those patients who consume statins) as well as mitochondrial dysfunction.

One of the key considerations with respect to Co-Q10 supplementation is its bioavailability, and this is an area where the Biotics microemulsfication technology can have a significant impact. Biotics utilizes a proprietary microemulsfication technology for all its fat soluble vitamin products (A, D, E, K, and Co-Q10)

The efficacy and bioavailability of the Biotics Co-Q-Zyme products is documented as follows on the Biotics website:

Based on a double blind clinical study, daily ingestion of 1 tablet (30 mg) of Biotics Research Corporation’s emulsified CoQ10 for 4 weeks was demonstrated to increase plasma CoQ10 levels by 210%, equivalent to 90-100 mg of dry CoQ10. Furthermore, dry CoQ10 powder increased serum levels in only 57% of subjects, while the Biotics Research Corporation emulsified CoQ10 produced an increase in serum CoQ10 levels in 80% of the subjects.1,2

Another topic of debate related to Co-Q10 is the controversy as to which form of CoQ10 is superior: Ubiquinol or Ubiquinone.

The following documentation again from the Biotics website addresses this topic:

Ubiquinone and ubiquinol, the reduced form of CoQ10, are known as redox pairs, meaning that in the cellular matrix they cycle back and forth. These two entities are rapidly inter-converted, regardless of the form ingested. Of importance to note is that the body naturally produces ubiquinone and not ubiquinol.

Human studies utilizing ubiquinol are lacking, thus claims on its superiority are presently invalid. Conversely, CoQ10 or ubiquinone has been utilized in hundreds of clinical studies, which have demonstrated its benefits for cardiovascular health, as well as for numerous other health issues.

CoQ10 is arguably of the key core nutrients that should be a part of everyone’s supplement regime, and like many of these key core nutrients, new benefits continue to be identified with ongoing research and publication of these results.

From the Biotics Website:

**CoQ-Zyme 100 Plus ~ Emulsified**

**Categories:** Allergies, Anti-Aging, Anti-Inflammatory, Antioxidants, Brain-Support, Cardiovascular-Support, Cellular-Metabolism, Energy, Folic-Acid, Immune-Support, Microemulsified, NPN, Neurological-Support, Vegetarian, Vitamins - Microemulsified

**Indications:**

ACTIVATES AMPK. AMPK is a master control mechanism for cellular energy homeostasis. It determines body fat composition and has a significant impact on mitochondrial biogenesis, the diabesity spectrum and life span extension in mice models. Necessary for one of the terminal steps in the electron transport chain in the process of energy (ATP) production).

Thus, as a necessary component of mitochondria for the production of energy, CoQ-10 has potential to improve physiologic function in a number of conditions associated with impaired mitochondrial function. Use for increased oxygenation need in cardiac stress or insufficiency, immune dysfunction, FM, CFS, headaches, high blood pressure, allergies, asthma, diabetes,
ulcers, viral infections, periodontal disease, and multiple sclerosis (MS). As an Antioxidant, Cell membrane stabilization, Inhibition of inflammation by modulation of NF-kappaB, reduces blood pressure in hypertensive patients as effectively as 1-2 drug combination treatment, Benefits pts with CHF or CRF, anti-allergy benefits. CoQ-10 deficiency is common in migraines and supplementation greatly reduces migraine attacks and Headaches 2007;47:73-80.

**Ingredients:**

- Thiamin (B1) (as cocarboxylase chloride) 1.5 mg
- Riboflavin (B2) (as riboflavin-5-phosphate) 1.7 mg
- Niacin (as niacin & niacinamide) 20 mg
- Vitamin B6 (as pyridoxal-5-phosphate) 2 mg
- Folate 400 mcg
- Vitamin B12 (as methylcobalamin) 6 mcg
- Biotin 300 mcg
- Pantothenic acid (as calcium pantothenate) 10 mg
- Coenzyme Q10 (Emulsified) 100 mg

**Suggestion:**

One tablet daily as directed by your health care professional.

**Safety:**

Biotics Research uses no soy byproducts, no artificial flavors or colorants, no propylene glycol, and no detergents or other artificial surfactants in our proprietary emulsification process. As a cellular component, CoQ10 has two primary functions in the body; first, to act in the transfer of electrons as a necessary part of ATP production, and second, to function as an essential antioxidant. CoQ10 participates in all energy processes in the body, and has been termed “the hub around which life processes revolve in the human body.”

It also plays a vital role in the cellular membrane, functioning in its stability, fluidity and permeability, in addition to stimulating cell growth and inhibiting cell death. In the body there is no means for storage for CoQ10, thus it must be made or replenished on a daily basis. Its synthesis in the cell mitochondria involves a complex 17-step process, which is dependent upon at least seven vitamin cofactors, including riboflavin (vitamin B2), niacin (vitamin B3), vitamin B6, vitamin B12, pantothenic acid (vitamin B5), folic acid, and vitamin C, along with several trace elements. In humans, the highest concentrations of CoQ10 are found in the heart, liver, muscle, kidney and brain. CoQ10 is an organic, nonprotein molecule which is ubiquitous in the cellular matrix.

The fact that it is ubiquitous initiated its primary designation, that of ubiquinone. Ubiquinone and ubiquinol, the reduced form of CoQ10, are known as redox pairs, meaning that in the cellular matrix they cycle back and forth. These two entities are rapidly inter-converted,
regardless of the form ingested. Of importance to note is that the body naturally produces ubiquinone and not ubiquinol. Human studies utilizing ubiquinol are lacking, thus claims on its superiority are presently invalid.

Conversely, CoQ10 or ubiquinone has been utilized in hundreds of clinical studies, which have demonstrated its benefits for cardiovascular health, as well as for numerous other health issues.

Biotics offers a spectrum of formulations which incorporate Co-Q10 which include standalone products as well as multi-ingredient formulations:

- **CoQ-Zyme 100 Plus ~ Emulsified**
- **CoQ-Zyme 30**
- **Bio-Cardiozyme Forte**
- **Bio-Detox Packs**
- **Bio-Immunozyme Forte**
- **Bio-Multi Plus**
- **BioProtect**
- **VasculoSirt**

Regards,

Rob Lamberton

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Here is the article from Life Enhancement Magazine

Along with Galantamine ...

**Coenzyme Q10 Helps Prevent Alzheimer’s**

The neuroprotective effect of CoQ10 could be due to its microglia inhibitory mechanism

By Will Block

There is considerable evidence showing that *mitochondrial dysfunction* and *oxidative damage* play a key role in the development of neurodegenerative disease, including both *Alzheimer’s disease* (AD) and *Parkinson’s disease* (PD). This relationship has been known for many years.

**CoQ10 Efficacy Along with Other Antioxidants**
In a review published eleven years ago (back in 2004), both mitochondrial dysfunction and oxidative damage were found to contribute to amyloid-beta (Aβ) damage in AD. Also discussed in this paper were the cumulative findings that PD is associated with abnormalities in the electron transport gene as well as oxidative damage, and that the efficacy of Coenzyme Q10 (CoQ10) along with other antioxidants might help treat both AD and PD.

CoQ10, an atypical lipophilic antioxidant, is an essential component of the mitochondrial electron-transport chain. It is involved in the manufacturing of adenosine triphosphate (ATP) and has been linked with improving cognitive functions. ATP is used in cells as a coenzyme and is often called the “molecular unit of currency” of intracellular energy transfer. ATP transports chemical energy within cells for metabolism.

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Both mitochondrial dysfunction and oxidative damage were found to contribute to amyloid-beta damage in AD.

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Mitochondrial Damage and AD

As far back as 1991 (how old were you then?), research showed that CoQ10 levels—studied in AD patients—were greatly reduced (approximately 50%) compared with controls. In 2000, a researcher announced that a role for mitochondrial dysfunction in neurodegenerative disease is gaining increasing support. Furthermore, it was concluded that if mitochondrial dysfunction plays a role in neurodegenerative diseases, then therapeutic strategies such as coenzyme Q10 and creatine may be useful in attempting to slow the disease process.

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Evidence indicated that AD is associated with oxidative damage that is caused in part by mitochondrial dysfunction.

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Vitamin E and CoQ10: Better Together for Learning Deficits

In another paper, the findings suggested that concurrent supplementation of alpha-tocopherol (vitamin E) with CoQ10 is more likely to be effective as a potential treatment for age-related learning deficits than supplementation with CoQ10 or alpha-tocopherol alone.

The same year, it was recognized that inhibition of the formation of amyloid-beta (Aβ) fibrils, as well as the destabilization of preformed Aβ fibrils in the central nervous system would be attractive therapeutic targets for the treatment of AD.

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CoQ10 Helps Prevent Oxidative Damage in the Brain
Dietary supplementation with CoQ10 at a dose of 10 g/kg diet to mice for one month—the human equivalent is 65 mg/day for a 176 lb human—significantly suppressed brain protein carbonyl levels, which are markers of oxidative damage.

In a 2008 article published in the Journal of Alzheimer’s Disease, more evidence indicated that AD is associated with oxidative damage that is caused in part by mitochondrial dysfunction (see Fig. 1). Is it possible to modify Alzheimer’s pathology with the nutrient CoQ10, the researchers asked? What they found is that CoQ10 protected neuroblastoma cells from Aβ protein precursor C-terminal fragment-induced neurotoxicity in a concentration-dependent manner, with concentrations of 6.25 microM and higher providing near complete protection.

MitoQ: A Targeting Form of CoQ10

In the 1990s, two New Zealand biochemists, desiring to get more CoQ10 more efficiently into mitochondria came up with an idea. They considered that a portion of the mitochondria’s electrochemical activity is employed to unceasingly pump protons out through their membranes. Thus the mitochondria maintain a negative electric potential compared to the rest of the cell. Furthermore, anything with a positive charge is drawn into the mitochondria, and if it’s not a proton, it won’t be pumped right back out again. Aha! So the two biochemists created a molecule that combined CoQ10 at one end with a chemical “tugboat” at the other, so as to create two propellers within the mitochondria.

The chemical tugboat, called TPP (tri-phenyl phosphonium), had been developed many years earlier in a Russian lab of Vladimir Skulachev. Connecting the tugboat to CoQ10 is a simple carbon chain that prevents interference between the chemical properties of the two ends.

Once the understanding had been achieved, synthesis followed, but not before years of testing, progressing through cell cultures, animal models, and eventually humans. This process, called targeting, eventually led to nearly all of newly created synthesized molecules taken up by mitochondria. This means that very low doses can result in very high effectiveness.

The manufacturers called this achievement MitoQ. MitoQ is considered an unapproved new drug by the FDA, and confiscations have been reported.

References

1. Burns RJ1, Smith RA, Murphy MP. Synthesis and characterization of


Dietary supplementation with CoQ10 at a dose of 10 g/kg diet to mice for one month—the human equivalent is 65 mg/day for a 176 lb human—significantly suppressed brain protein carbonyl levels, which are markers of oxidative damage.

It is widely known that one of the neuropathological features of AD is the deposition of senile plaques containing Aβ. In a study reported in 2008, researchers tested CoQ10, an endogenous antioxidant and a powerful free radical scavenger on Aβ in aged transgenic mice overexpressing Alzheimer presenilin 1, a mutation involved in the majority of early onset, autosomal dominant Alzheimer’s disease.7

**Mitochondrial dysfunction is widely implicated in the pathogenesis of AD.**

The transgenic mice fed CoQ10 for 60 days (at 1,200 mg/kg/day) partially reduced Aβ overproduction and intracellular Aβ deposit in the cortex of the transgenic mice compared with the age-matched untreated transgenic mice.

Supplementation with CoQ10 also decreased malondialdehyde levels and upregulated the activity of superoxide dismutase. These results indicate that CoQ10 would be beneficial for the therapy of AD.

**Increased autophagic degradation of mitochondria has also been observed in AD.**

**Mitochondrial Dysfunction Is Implicated in AD**

Mitochondrial dysfunction is widely implicated in the pathogenesis of AD. Evidence for mitochondrial dysfunction in AD pathogenesis comes from impaired activities of the three key Krebs cycle enzyme complexes: pyruvate dehydrogenase, isocitrate dehydrogenase, and α-ketoglutarate dehydrogenase.
These impairments have been observed in postmortem AD brain and fibroblasts from AD patients. Also, reduced respiratory chain complex I activity was found in platelets and lymphocytes from AD patients and in postmortem brain tissue.

More recently, increased autophagic degradation (autophagy is the natural, destructive mechanism that disassembles, through a regulated process, unnecessary or dysfunctional cellular components) of mitochondria has also been observed in AD. In a review, several bioenergetic agents are discussed that improve mitochondrial function including CoQ10, creatine, nicotinamide, riboflavin, and lipoic acid, which are being tested for their neuroprotective efficacy in neurodegenerative disorders.

Animal studies aside, 2009 marks the year of a human study measuring the ratio of the oxidized and reduced forms of CoQ10 related to the pathogenesis of AD. The findings in living AD patients suggest a possible role for oxidized CoQ10 in the pathogenesis of the early stage of AD development.

Improved Behavior in Transgenic AD Mice
Another mouse study found decreased pathology and improved behavior in transgenic AD mice treated with the naturally occurring antioxidant compound CoQ10. First off, CoQ10 decreased brain levels of protein carbonyls, a marker of oxidative stress. Then, CoQ10 resulted in decreased plaque area and number in the hippocampus and in overlying cortex immune-stained with an Aβ42-specific antibody.

Also, brain Aβ42 levels were decreased by CoQ10 supplementation, while levels of amyloid-β protein precursor β-carboxyterminal fragments were decreased. Significantly, CoQ10-treated mice showed improved cognitive performance during Morris water maze testing. The results showed decreased pathology and improved behavior in transgenic AD mice treated with CoQ10.

CoQ10 Neuroprotective Effect
Researchers from Panjab University in Chandigarh, India recently reported that the neuroprotective effect of CoQ10 could be due to its microglia inhibitory mechanism along with
its mitochondrial restoration and anti-oxidant properties. Microglia are a type of glial cell that are the resident macrophages of the brain and spinal cord, and thus act as the first and main form of active immune defense in the central nervous system. One way to control neuroinflammation is to inhibit microglial activation.

Intrahippocampal Aβ (1-42) was administered in rats (not easy to do in humans) to induce the equivalency of many aspects of AD in humans. The intent of the study was to investigate the neuroprotective potential of CoQ10 and its modulation by minocycline (a microglial inhibitor) against Aβ (1-42), which induced cognitive dysfunction in the lab animals. Minocycline is a broad-spectrum tetracycline antibiotic drug that penetrates the brain. Minocycline has many side effects (see below).

One way to control neuroinflammation is to inhibit microglial activation.

Following this, galantamine (2 mg/kg), CoQ10 (20 and 40 mg/kg), minocycline (50 and 100 mg/kg) and their combinations were administered for a period of 21 days. The scientists measured various neurobehavioral parameters followed by biochemical factors, including acetylcholinesterase (AChE) levels, proinflammatory markers (TNF-α), and mitochondrial respiratory enzyme complexes (I-IV). Histopathological examinations were also assessed. In clinical medicine, histopathology refers to the examination of a biopsy or surgical specimen by a pathologist.

Aβ (1-42) administration significantly impaired cognitive performance in the Morris water maze performance test, caused oxidative stress, raised AChE levels, caused neuroinflammation, mitochondrial dysfunction and histopathological alterations, as compared to sham treatment.

Treatment with CoQ10 (20 and 40 mg/kg) and minocycline (50 and 100 mg/kg) alone for 21 days significantly improved cognitive performance as evidenced by reduced transfer latency and increased time spent in the target quadrant—treading water over the submerged platform area, after it had been removed.

The neuroprotective effect of CoQ10 could be due to its microglia inhibitory mechanism along with its mitochondrial restoring and anti-oxidant properties.

The rats were also found to have reduced AChE activity (that’s one of the benefits of galantamine), oxidative damage (reduced lipoperoxides, nitrite level, and restored SOD, catalase and glutathion peroxidase levels), TNF-α level, while restoring mitochondrial respiratory enzyme complex (I, II, III, IV) activities and histopathological alterations as compared
to Aβ (1-42) treated animals.

Furthermore, the combination of minocycline (50 and 100 mg/kg) with CoQ10 (20 and 40 mg/kg) significantly modulates the protective effect of CoQ10 dose dependently as compared to their effect alone. The neuroprotective effect of CoQ10 could be due to its microglia inhibitory mechanism along with its mitochondrial restoring and anti-oxidant properties.

**The Findings of the Indian Study**

In the Indian study, the administration of Aβ (1-42) significantly impaired cognitive performance in the Morris water maze test as indicated by delayed transfer latency time to reach the platform (taking longer to find the platform), and getting to where the platform had been, suggesting that Aβ (1-42) induced impairment in spatial learning and memory.

Aβ (1-42) also caused significant increase in AChE activity indicating an impairment of the cholinergic system, a measure of cholinergic dysfunction. Also, the study revealed that, administration of Aβ (1-42) resulted in a significant increase in MDA; nitrite concentration, depleted reduced glutathione, SOD and catalase activities in Aβ (1-42)-treated animals. Thus, Aβ (1-42) instigated oxidative stress.

**Altered Activity of Mitochondrial Respiratory Enzymes**

The study also indicated that Aβ (1-42) accumulation significantly altered the activity of mitochondrial respiratory enzymes (NADH dehydrogenase, succinate dehydrogenase activity, and cytochrome C oxidase). Aβ (1-42) accumulation also impaired the neural cell availability of Aβ (1-42)-treated animals as compared to sham treated animals. This suggested mitochondrial dysfunction.

Moreover, the study found that administration of Aβ (1-42) significantly increased TNF-α level in Aβ (1-42)-treated animals as compared to sham-treated animals, indicating the role of neuroinflammation in the Aβ (1-42) model of cognitive dysfunction. Previous studies have also demonstrated the neuroprotective potential of both CoQ10 and minocycline in different neurological conditions. However, their mechanism of action is not yet clear. Supporting the above, the Indian study demonstrated the neuroprotective effect of CoQ10 and minocycline against Aβ (1-42)-induced cognitive dysfunction.

Therefore, beneficial treatment of CoQ10 along with minocycline in neurodegenerative disorders including AD with multiple pathogenic properties can be achieved through drugs
targeting multiple pathways or polytherapeutic interventions directed toward specific aspect of the neurodegenerative phenotype, the composite of an organism's observable characteristics or traits.

**Aβ (1-42) instigated oxidative stress.**

**CoQ10 Facilitates ATP Synthesis**

CoQ10 improves cognitive function, upregulates mitochondrial function, and facilitates ATP synthesis. Supplementation of CoQ10 increases brain endogenous CoQ10 content and acts as an antioxidant against free radical generation and oxidative modification of biomolecules. It also provides significant enhancement in patients with neurological disorders.

**Aβ (1-42) accumulation significantly altered the activity of mitochondrial respiratory enzymes.**

The major role of CoQ10 is to transfer electrons between redox components of the electron transfer chain, to create a proton gradient across the inner mitochondrial membrane, drive ATP formation, and act as a powerful antioxidant that is protective against oxidative stress (see Fig. 2).

Thus, CoQ10 *in vivo* protects against oxidation of lipids, proteins, and DNA. Previous evidence has suggested an intimate link between excessive generation of reactive oxygen and reactive nitrogen species and development of hippocampal neuronal death. In the present study, chronic treatment with CoQ10 (20 and 40 mg/kg) in Aβ (1-42)-treated animals significantly lessened impairment of spatial learning and memory task performance, AChE activities, mito-oxidative damage, restored mitochondrial respiratory enzyme complex activities and TNF-α level suggesting its anti-oxidant, mitochondrial-restoring and anti-inflammatory action as compared to Aβ (1-42)-treated animals.

Minocycline, a semi-synthetic second-generation tetracycline analog has both antimicrobial and anti-inflammatory activity. It also possesses efficacy against a broad range of neurological disorders with a good clinical safety record. Previous studies on animal models showed that minocycline prevented the release of cytochrome C (a facilitation protein operating in the mitochondria electron transport chain) in isolated mitochondria, suggesting mitochondria as a direct target of minocycline. Minocycline also inhibits the formation of Aβ aggregates and disassembles the preformed fibrils.

**Aβ (1-42) accumulation also impaired the neural cell availability.**

Furthermore, chronic treatment with minocycline improved spatial learning and memory task performance, reduced AChE activities, mito-oxidative damage, restored mitochondrial
respiratory enzyme complex activities and attenuated TNF-α level, suggesting its anti-oxidant, mitochondrial restoring and anti-inflammatory action as compared to Aβ (1-42)-treated animals.

Furthermore, chronic treatment with CoQ10 in combination with minocycline significantly enhanced their neuroprotective effect (lessening spatial learning and memory performance task), AChE activities, mito-oxidative damage, restored the mitochondrial respiratory enzyme activities and TNF-α level) suggesting their synergistic action. In the present study, Aβ (1-42) administration caused significant damage in the hippocampal and cortical region of the brain that plays important roles in learning and memory.

\[ \text{CoQ10 improves cognitive function, upregulates mitochondrial function, and facilitates ATP synthesis.} \]

For the histopathological evaluation, the hippocampal region was targeted and it was observed that brain areas of Aβ (1-42) treated animals had significantly higher density of pyknotic neuronal cells suggesting degeneration and disrupted functioning of the hippocampal neurons.

Besides, CoQ10 in combination with minocycline, significantly improved cell layer organization, reduced density of pyknotic (the irreversible condensation of chromatin in the nucleus of a cell undergoing necrosis or apoptosis) cells and pyramidal cell in the CA1 and CA3 regions of the hippocampus as compared to their effects alone in Aβ (1-42) treated rat brain hippocampus.

**Adverse Effects of Minocycline**

In support, these results highlight anti-oxidant, anti-inflammatory and mitochondrial restoring properties of CoQ10. However, when in combination with minocycline, the neuroprotective effect against Aβ (1-42)-induced cognitive dysfunction is increased. Nonetheless, minocycline is a drug that has many adverse effects, some of which may be serious, including confusion, diarrhea, dizziness or lightheadedness, severe headache, severe stomach pain, and many more. It is not recommended for AD or PD. However, its mechanism—microglia inhibition, which is also true for CoQ10—could lead to other discoveries such as herbal medicines and dietary supplements. These may include catechol-containing antioxidants.

Furthermore, since both galantamine and CoQ10 alone were shown to have positive effects and work by different mechanisms, the total effects are likely to be additive.

**References**


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