

Original Article

Combined Effects of Coenzyme Q10 & L-Carnitine on Neuroinflammation in a Rat Model of Multiple Sclerosis.

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Abstract

Objective: This study aimed to determine the potential additive effects of Coenzyme Q10 and L-Carnitine on neuroinflammation in a rat model of Multiple Sclerosis (MS), based on histological and biochemical parameters.

Methodology: This Experimental study was conducted at Islamic International Medical College in collaboration with NIH from March 2023 to June 2023. Fifty male Sprague-Dawley rats were randomly divided into five groups of ten rats each. Group 1 served as a negative control. The remaining groups were disease models, administered 0.2% cuprizone w/w to induce MS. After 4 weeks, Group 3 received 100 mg/kg/day L-carnitine, Group 4 received 150 mg/kg/day CoQ10, and Group 5 received both, while cuprizone administration continued. The experiment lasted for 12 weeks, after which the rats were sacrificed, and their brains were dissected. Coronal sections were stained with H & E to evaluate inflammatory infiltrates. IL-6 and TNF- α levels in the neuronal tissue were measured using ELISA kits.

Results: The combined treatment group showed a significant reduction in inflammatory infiltrates compared to the CPZ group ($p \leq 0.001$). Better control of the reduction in IL-6 and TNF- α levels was observed in the combination group than in the single-agent group ($p \leq 0.05$).

Conclusion: The study concluded that CoQ10 and L-carnitine have a potential additive effect in controlling neuroinflammation, as evidenced by fewer inflammatory infiltrates and reduced levels of IL-6 and TNF- α .

Keywords: Coenzyme Q10, L-Carnitine, Inflammatory Infiltrate, Neuroinflammation.

Introduction

Multiple sclerosis (MS) is a well-known chronic neurodegenerative disease that targets the Central Nervous System (CNS)¹ and affects peripheral nerves.² It is considered a major health concern globally.³ MS is believed to be autoimmune, mediated by autoreactive lymphocytes that cross the blood-brain barrier and enter the CNS, where they activate astrocytes, microglia, and inflammatory cells.⁴ These cells secrete various cytokines, such as tumour necrosis factors, interleukins, and proteolytic enzymes, which cause neuroinflammation, demyelination, and axonal degeneration.⁵

The cuprizone (CPZ) model is an established animal model used to study MS. CPZ is a selective copper-chelating agent that causes oxidative stress at the mitochondrial level. The CPZ model resembles MS in humans, as it also activates glial cells, such as astrocytes and microglia, which are responsible for neuroinflammation by the release of TNF- α , IL-6, and other cytokines.⁶

Neuroinflammation in the central nervous system (CNS) is associated with immune responses, leading to a cascade of pathological processes, including glial cell activation and the release of various inflammatory cytokines.⁷ In an animal model of MS, CPZ administration causes oxidative injury, and a similar inflammatory response is observed through the differentiation of T-lymphocytes into Helper T-cells and Regulatory T-cells.⁸ CPZ also causes the proliferation and accumulation of microglia and macrophages in the white matter, which release pro-inflammatory cytokines, such as interleukins, especially IL-6, and tumour necrosis factor.⁹

Conflicts of Interest: None

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AI Disclosure: No generative AI tools were used for the data analysis. AI-assisted language refinement was performed using ChatGPT-4 and Grammarly, and the results were verified by the authors.

Data Availability Statement: The datasets generated and analysed during the current study are available from the corresponding author upon reasonable request, including raw ELISA data and histological image data.

Institutional Review Board

Approval

Riphah/IRC/23/3013

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L-Carnitine is an amino acid essential for the metabolism of energy in tissues and the balance of energy across cell membranes. Long-chain fatty acids are transported by carnitine from the cytoplasm into the mitochondria, where they are used during beta-oxidation. Additionally, it eliminates some metabolic by-products, including short- and medium-chain fatty acids, to stop their build-up in the mitochondria and protect the cell from oxidative damage.¹⁰

L-carnitine is considered neuroprotective and has been utilised in patients with Alzheimer's disease, cardiac dysfunction, and severe depressive disorders because it protects key bodily tissues from mitochondrial oxidative stress.^{11,12}

Coenzyme Q10 (CoQ10), also known as ubiquinone, is an endogenous lipid. The majority of it is produced intracellularly, although a small quantity can also be found in select foods, including fish, whole grains, and organ meat.¹³

The mitochondrial respiratory chain depends on CoQ10 for electron transfer. It aids aerobic cellular respiration, boosting ATP production and energy. CoQ10 also protects mitochondrial deoxyribonucleic acid and lipoproteins found in the plasma membrane from oxidative damage and inflammation caused by free radicals. Additionally, CoQ10 can regenerate and recycle additional anti-inflammatory agents, such as vitamins C and E.¹⁴ CoQ10 has been shown to have several positive effects on neuroinflammation in both basic and clinical investigations.¹⁵

This study determined the potential combined effect of CoQ10 and L-carnitine on controlling inflammatory infiltrates and inflammatory markers (IL-6 and TNF- α) in the Corpus Callosum of the rat brain.

Methodology

The study commenced after approval from the Institutional Review Committee, Riphah International University, Ref No. Riphah/IIMC/ IRC/23/3003. A total of 50 male Sprague-Dawley rats (4–6 weeks old, weighing 200–250 g) were purchased from the animal house of the National Institute of Health (NIH), Chak Shehzad, Islamabad. Rats were randomly assigned to five experimental groups (n=10) using a computer-generated randomisation sequence. Allocation concealment was not performed in this study. All animals were kept under standard laboratory conditions. The procurement of animals, experimental procedures, and ethical considerations were in accordance with the Animal Research: Reporting of In Vivo Experiments (ARRIVE) guidelines.¹⁶

CPZ was purchased from Beijing Solarbio Science & Technology Co., Ltd. Group 1 was the negative control and received a standard diet. All other groups were MS models; Group 2 was the disease control and received 0.2% CPZ w/w mixed in standard food for 12 weeks. Group 3 received 0.2% CPZ w/w for 12 weeks, and 100 mg/kg/day L-carnitine was added from the 5th week.¹⁷ Group 4 received 0.2% CPZ w/w for 12 weeks, and 150 mg/kg/day CoQ10 was started from the 5th week. Group 5 was the combined group, and it received 100 mg/kg/day L-carnitine and 150 mg/kg CoQ10 from the 5th week, while the administration of 0.2% CPZ powder lasted for 12 weeks.

After 12 weeks, the rats were euthanised, and their brains were dissected. The specimens were fixed in 10% formalin solution. For the corpus callosum, two coronal sections of 5 mm thickness were obtained from each rat brain. The first section was taken at the level just anterior to the optic chiasma, and the second was at the level of the infundibulum.¹⁸ Tissue sections underwent standard dehydration, clearing, infiltration, and embedding procedures, and 5 μ m thin slices were stained with H&E solution.⁴ Inflammation was qualitatively graded (Table 1).

Table 1: Grading criterion for Inflammatory Infiltrates¹⁹

Parameters	Grade 0	Grade 1	Grade 2	Grade 3
Inflammatory infiltrates	Absent (No cells)	Mild (<25cells/hpf)	Moderate (25-125cells/hpf)	Severe (>125cells/hpf)

A small section of the corpus callosum was collected and homogenised in cold phosphate-buffered saline (PBS) with a pH of 7.4, after centrifugation at 10,000rpm for 15 min. The supernatant was collected, and ELISA kits were used to estimate the levels of IL-6 and TNF- α .

Although the RRIDs for the materials used could not be retrieved or verified, specific catalogue numbers or manufacturer details are mentioned here to ensure experimental reproducibility.

CoQ10: *Lezyme by Herbiotics*, purchased from D-Watson

L-Carnitine: *L-Carnitine by Herbiotics*, purchased from D-Watson

Cuprizone (*bis(cyclohexanone)oxaldihydrazone*; Beijing Solarbio Science & Technology Co. Ltd; Cat. No.: C8060)

ELISA Kit for IL-6: *Sunlong Medical™*. One-EK015M

ELISA Kit for TNF- α : *Sunlong Medical™*

The investigators responsible for the histological assessment and ELISA analysis were blinded to the group allocation to minimise observer bias.

Data were entered and analysed using the Statistical Package for Social Sciences (SPSS version 26). The normality of the sample was ensured using the Shapiro–Wilk test. One-way analysis of variance (ANOVA) was applied to compare quantitative variables, which were expressed as mean $\pm$ SD. Post hoc Tukey's test was applied for comparisons among groups. Qualitative variables were expressed as frequencies and percentages. The Kruskal-Wallis test was used to analyse the differences between groups. A p-value of less than or equal to 0.05 was considered significant.

Results

Inflammatory cells were distinguished by densely packed round blue nuclei (Fig. 1f). The normal control group did not show any inflammatory infiltrates. In the disease control group, 90% of the slides showed grade 3 inflammation. With CoQ10, 70% of the rats showed grade 2 inflammation, whereas 30% showed grade 1 inflammation. After the administration of L-carnitine, 90% of the rats showed grade 2 inflammation, and the remaining 10% showed grade 1 inflammation. In the combined group, 60% of the rats showed grade 1, 20% showed grade 2 inflammatory infiltrate, and 20% did not show any inflammatory cells.

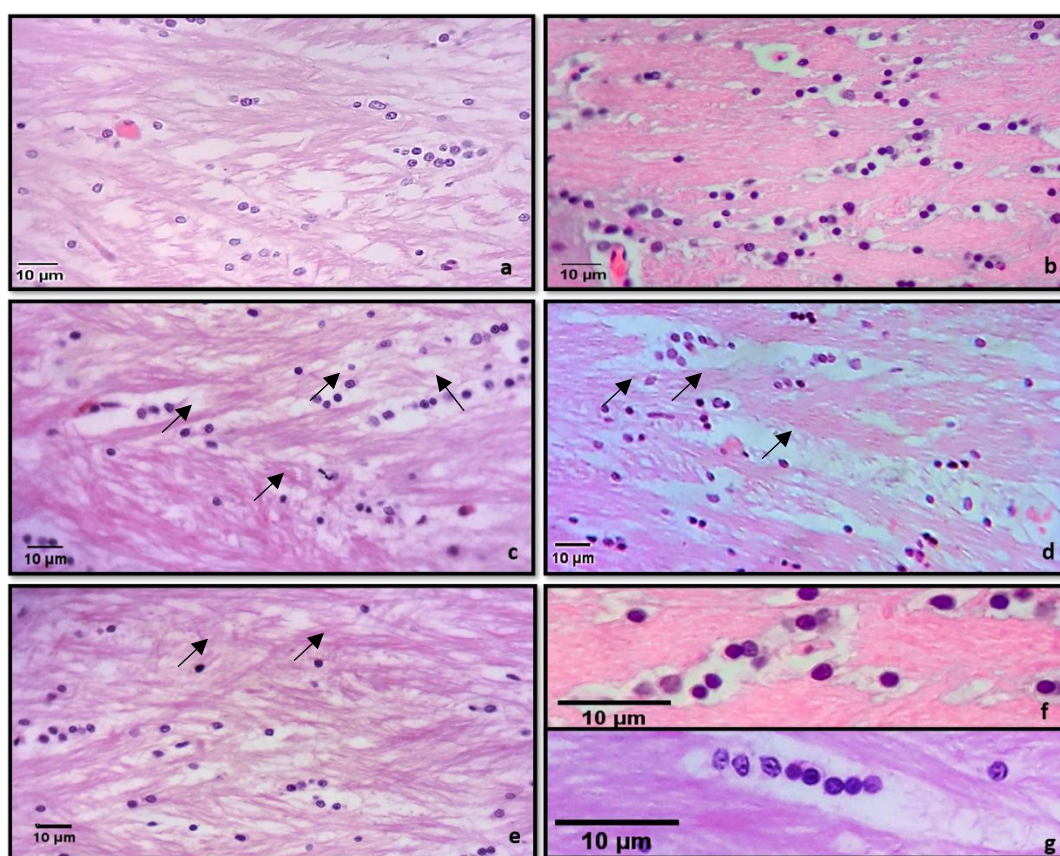


Figure 1: Photomicrographs of H & E-stained sections showing inflammatory infiltrate (400X)
a: No inflammatory cells can be seen in Group1; b: Severe infiltration of inflammatory cells can be seen in Group 2 (encircled); c: Some inflammatory cells are visible in Group 3 (arrows); d: Few inflammatory cells can be seen in Group 4(arrows); e: Occasional inflammatory cells are visible in Group 5 (arrows); f: Inflammatory cells; g: Oligodendrocytes

The Kruskal-Wallis test showed that inflammatory infiltrates differed significantly among the five groups ($p < 0.001$). The combined treatment significantly reduced infiltration compared with the CPZ + disease control ($p < 0.001$), with infiltrate levels comparable to those observed in the normal control group ($p = 0.610$). CoQ10 and L-carnitine alone did not significantly reduce the number of infiltrates relative to the CPZ group after Bonferroni correction ($p = 0.066$ and $p = 0.245$, respectively). Both monotherapy groups exhibited significantly greater inflammatory infiltration than the normal control group (CoQ10: $p = 0.007$ and L-carnitine: $p = 0.001$).

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No significant difference was observed when the combination group was compared with the CoQ10 ($p=1.000$) and L-carnitine ($p=0.480$) groups. (Table 2)

Table 2: Difference in Inflammatory Infiltrate analyzed by Kruskal-Wallis test

Group comparison	Bonferroni-adjusted p -value
Normal diet vs. CPZ	<0.001**
Normal diet vs. CoQ10	0.007**
Normal diet vs. L-carnitine	0.001**
Normal diet vs. CoQ10 + L-carnitine	0.610
CPZ vs. CoQ10	0.066
CPZ vs. L-carnitine	0.245
CPZ vs. CoQ10 + L-carnitine	<0.001**
CoQ10 vs. L-carnitine	1.000
CoQ10 vs. CoQ10 + L-carnitine	1.000
L-carnitine vs. CoQ10 + L-carnitine	0.480
Kruskal Wallis: $H(4) = 41.49, p < 0.001$.	
**$p < 0.001$ after Bonferroni correction.	

TNF- α levels were significantly higher in the MS model, while these levels decreased significantly with CoQ10 and L-carnitine, and their combination was even more effective in reducing TNF- α levels. The p -value was highly significant (<0.001), and the post-hoc Tukey test also showed highly significant results when individual agents were compared with the combined group and when treated groups were compared with the control groups (Table 3).

Discussion

Multiple sclerosis (MS) is an autoimmune inflammatory disease of the Nervous System. It is believed to cause oxidative damage at the mitochondrial level in oligodendrocytes, leading to an intense glial response. Histologically, a severe inflammatory infiltrate is observed, and the activated glial cells secrete a large amount of pro-inflammatory cytokines, such as TNF- α and IL-6. The CPZ model mimics MS by causing inflammatory changes and glial responses (Fig. 8). In this study, two antioxidants were administered to reverse the inflammatory changes caused by CPZ. We compared the effects of individual agents on neuroinflammation with those of the combination to better control symptoms.

CPZ induced severe neuroinflammation, as evidenced by inflammatory infiltrates in Group 2, mimicking MS. CoQ10 & L-Carnitine significantly reduced inflammatory cells, and their combination was comparable to the normal control group. These findings were similar to those of a study that showed that CoQ10 regulates neuroinflammation in subarachnoid haemorrhage.²⁰ Another study showed that CoQ10 ameliorates neuroinflammation in Alzheimer's disease.²¹ L-Carnitine also plays an anti-inflammatory role in a model of hyperphenylalaninemia.²² The same findings were observed in a study where L-carnitine lowered inflammatory infiltrates in ischaemia-reperfusion injury of the spinal cord in rats.²³

The mean TNF-alpha expression levels were very high in the CPZ Group compared to the normal group. L-carnitine, CoQ10, and their combination significantly reduced these levels. T Emran in 2021 showed the protective effect of L-carnitine on cardiac damage by lowering TNF- α levels.²⁴

IL-6 levels were increased by CPZ administration, whereas our therapeutic agents significantly reduced them. The combined treatment was even more effective in lowering IL-6 levels than individual agents. A recent study

Table 3: Mean values of TNF- α levels, with Standard Deviation

Groups	Mean $\pm$ SD TNF- α levels (pg/ml)
Group 1	40.7 $\pm$ 1.9
Group 2	535.0 $\pm$ 3.6
Group 3	168.8 $\pm$ 7.8
Group 4	225.4 $\pm$ 2.5
Group 5	134.4 $\pm$ 4.0
P value <0.001	

The levels of IL-6 were markedly increased in Group 2, and CoQ10 and L-carnitine decreased the levels of IL-6. Group 5 showed an additive effect by decreasing IL-6 levels more than that of individual agents. The p -value was highly significant (<0.001). The Post Hoc Tukey test showed highly significant results when individual agents were compared with the combined group and when the treated groups were compared with the control groups (Table 4).

Table 4: Mean values of IL-6 levels, along with Standard Deviation

Groups	Mean $\pm$ SD IL-6 levels (pg/ml)
Group 1	107.2 $\pm$ 10.9
Group 2	482 $\pm$ 31.29
Group 3	224.8 $\pm$ 25.11
Group 4	281.2 $\pm$ 30.25
Group 5	145.8 $\pm$ 35.7
P value <0.001	

combined CoQ10 and L-carnitine to show their additive effect in lowering IL-6 levels in acute brain injury.²⁵ Similar results were reported by Alhusaini in 2022 in propionic acid-induced oxidative damage, using L-Carnitine.²⁶ CoQ10, an essential lipid component of the mitochondrial respiratory chain, is directly involved in electron transport, facilitating aerobic cellular respiration and adenosine triphosphate (ATP) generation. Moreover, it acts as a highly potent lipid-soluble antioxidant, protecting mitochondria from radical oxidative damage while recycling other antioxidants, such as vitamins C and E. L-carnitine, being indispensable for cellular energy homeostasis, serves as a transport shuttle for long-chain fatty acids across the mitochondrial membrane for β -oxidation. When administered in combination, these agents target mitochondrial distress. This dual metabolic support effectively reduced inflammation and rescued CPZ-induced mitochondrial dysfunction (Figure. 2)

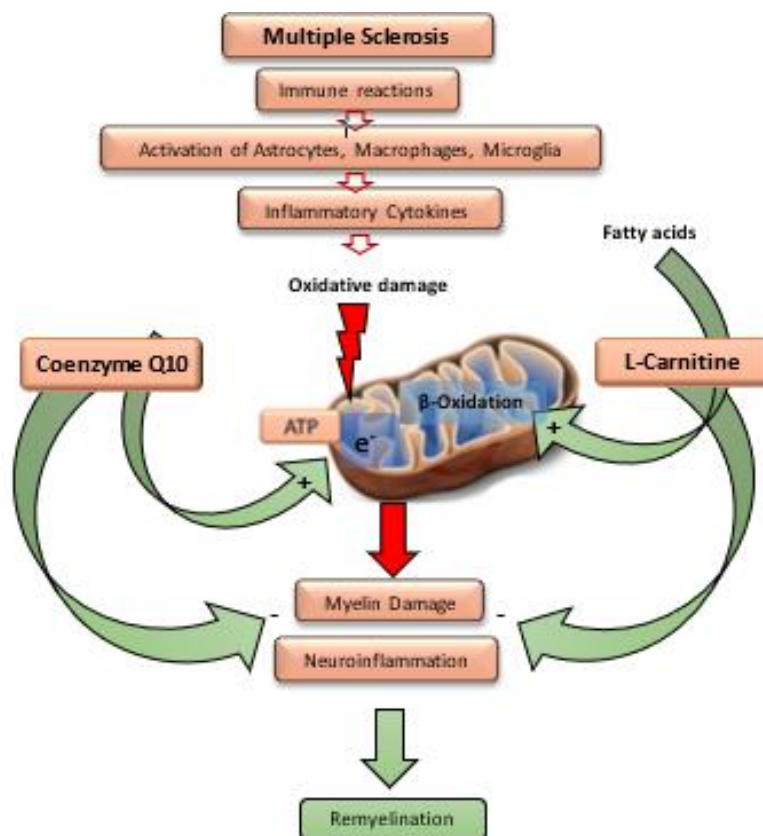


Figure 2: Mechanism of action of CoQ10 & L-Carnitine in decreasing neuroinflammation in Multiple Sclerosis.

Conclusions

Overall, this study showed that CoQ10 and L-carnitine have a potential additive effect on reducing inflammatory infiltrates and inflammatory cytokines (TNF- α and IL-6). Thus, the combined effect of these agents may be beneficial in minimising the frequency of symptoms in patients with MS.

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TQ SA SN MH SB TF - Critical Review

All authors approved the final version to be published & agreed to be accountable for all aspects of the work.

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