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Research Article

Effects of Quercetin on Disease Severity and Functional Recovery in an Experimental Autoimmune Encephalomyelitis Rat Model

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Abstract

Quercetin is a naturally occurring plant flavonoid known for its diverse biological activities, including anti-inflammatory, antioxidant, and anticancer effects. It has also been shown to promote the differentiation of neuronal precursor cells. This study investigated whether quercetin administration could slow the progression of Experimental Autoimmune Encephalomyelitis (EAE), an animal model of Multiple Sclerosis (MS), and facilitate neuronal repair in damaged tissues.

Keywords: Quercetin, Multiple sclerosis, Recovery, Regenerative, Disease, Flavonoid

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Background

Quercetin is a naturally occurring plant flavonoid widely distributed in fruits, vegetables, tea, and medicinal plants. It is recognized for its broad spectrum of biological activities, including potent anti-inflammatory, antioxidant, immunomodulatory, and anticancer properties. Numerous studies have demonstrated that quercetin scavenges reactive oxygen species (ROS), inhibits lipid peroxidation, and enhances the activity of endogenous antioxidant enzymes, thereby protecting cells from oxidative damage. In addition, quercetin modulates immune responses by suppressing the production of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-1β (IL-1β), and interleukin-6 (IL-6), which are implicated in the pathogenesis of several autoimmune and neurodegenerative disorders. Furthermore, quercetin has been reported to promote neuronal survival, stimulate neurite outgrowth, and enhance the differentiation of neuronal precursor cells, suggesting a potential role in neuro regeneration and repair **[Table 1].**

Parameter	Injectable quercetin	Oral quercetin
Bioavailability	High	Moderate
Tissue Distribution	Greater	Lower
Clinical Recovery	Superior	Moderate
Weight Maintenance	Better	Comparable to saline

Parameter	Injectable quercetin	Oral quercetin
Overall Therapeutic Effect	Highest	Moderate

Table 1: Comparison of injectable and oral quercetin.

Multiple Sclerosis (MS) is a chronic autoimmune demyelinating disease of the central nervous system characterized by inflammation, axonal degeneration, and progressive neurological dysfunction. Experimental Autoimmune Encephalomyelitis (EAE) is a widely used animal model that closely mimics the immunopathological and clinical features of MS, making it a valuable tool for investigating disease mechanisms and evaluating potential therapeutic agents. Given the antioxidant, anti-inflammatory, and neuroprotective properties of quercetin, it has emerged as a promising candidate for the treatment of autoimmune neurological disorders. Therefore, the present study aimed to investigate whether quercetin administration could attenuate disease progression, reduce clinical severity, and promote recovery in EAE-induced rats. Additionally, the study sought to evaluate the potential regenerative effects of quercetin on damaged neural tissues and compare the efficacy of oral and injectable routes of administration in improving disease outcomes.

Materials and methods

EAE was induced in female Lewis rats using a homogenate prepared from guinea pig spinal cord tissue mixed in a 1:1 ratio with Complete Freund's Adjuvant (CFA). Thirty rats were allocated into three experimental groups: **[Table 2].**

- ES group: EAE-induced rats treated with normal saline.
- EQI group: EAE-induced rats treated with injectable quercetin.
- EQO group: EAE-induced rats treated with oral quercetin.

Group	Number of rats	Treatment	Route	Duration
ES	10	Normal saline	Intraperitoneal	21 days
EQI	10	Quercetin	Injectable	21 days
EQO	10	Quercetin	Oral	21 days

Table 2: Experimental groups and treatment protocol.

Treatment commenced one day after the onset of disease symptoms and continued for 21 days. Body weight and clinical manifestations were monitored daily throughout the experimental period. Data were analyzed using one-way ANOVA followed by Tukey's post hoc test, with statistical significance established at $p < 0.05$.

Results

Quercetin treatment significantly improved disease outcomes and accelerated recovery in EAE-induced rats. Animals receiving quercetin exhibited reduced disease severity and better clinical recovery compared with saline-treated controls. Furthermore, the injectable form of quercetin demonstrated superior efficacy compared with oral administration. Weight changes in the orally treated group were comparable to those observed in the saline-treated group, whereas the injectable quercetin group showed a distinct and more favorable pattern of weight maintenance and recovery.

Discussion

Previous studies have demonstrated that quercetin suppresses inflammatory responses by reducing the proliferation of autoreactive immune cells and decreasing the production of pro-inflammatory mediators such as TNF-α, IL-1β, and matrix metalloproteinase-9. Quercetin has also been shown to inhibit microglial activation, reduce oxidative stress, and suppress signaling pathways involved in autoimmune inflammation, including JAK/STAT-mediated T-helper cell differentiation **[Table 3]**.

Biological effect	Mechanism	Expected outcome in EAE
Antioxidant	Scavenges ROS	Reduced oxidative stress
Anti-inflammatory	Decreases TNF-α, IL-1β, IL-6	Reduced CNS inflammation
Immunomodulatory	Suppresses autoreactive T cells	Lower disease severity
Neuroprotective	Prevents neuronal apoptosis	Improved neurological function
Regenerative	Promotes remyelination	Enhanced recovery

Table 3: Proposed biological actions of quercetin.

In addition, quercetin exerts neuroprotective effects through its antioxidant properties and iron-chelating capacity. These actions may reduce neuronal damage, edema formation, and apoptosis following central nervous system injury. Histological studies have indicated that EAE-induced pathology is particularly severe in the lumbar spinal cord, characterized by extensive demyelination and tissue damage. The observed therapeutic benefits of quercetin in this study are consistent with its reported anti-inflammatory and regenerative properties.

The greater effectiveness of injectable quercetin may be explained by its enhanced bioavailability. Earlier investigations have shown that oral quercetin undergoes extensive metabolic conversion, reducing its accumulation in target tissues. In contrast, intraperitoneal or injectable administration results in higher tissue concentrations and improved biological activity.

Conclusion

The findings of this study support the therapeutic potential of quercetin as a complementary treatment for Multiple Sclerosis. Quercetin administration reduced disease progression and promoted recovery in EAE-induced rats, likely through its antioxidant, anti-inflammatory, and regenerative mechanisms. Notably, injectable quercetin was significantly more effective than oral administration, highlighting the importance of delivery route in achieving optimal therapeutic outcomes. These results reinforce previous evidence suggesting that quercetin may contribute to neuroprotection and myelin repair in inflammatory neurological disorders.

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