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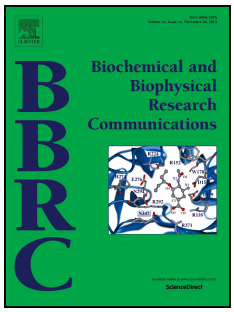

Glial cell-derived neurotrophic factor alleviates sepsis-induced neuromuscular dysfunction by decreasing the expression of γ- and α7-nicotinic acetylcholine receptors in an experimental rat model of neuromyopathy

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Glial cell-derived neurotrophic factor alleviates sepsis-induced neuromuscular dysfunction by decreasing the expression of γ- and α7-nicotinic acetylcholine receptors in an experimental rat model of neuromyopathy

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Highlights

- Sepsis-induced demyelination of sciatic nerve up-regulated the expression of γ- and α7-nicotinic acetylcholine receptors (nAChRs), which are associated with neuromuscular dysfunction.
- Exogenous injection of glial cell-derived neurotrophic factor (GDNF) alleviated neuromuscular dysfunction by decreasing γ- and α7-nAChR expression.

Abstract

Sepsis-induced neuromuscular dysfunction results from up-regulation of the expression of γ- and α7-nicotinic acetylcholine receptors (nAChR). Although glial cell derived neurotrophic factor (GDNF) has been implicated in repairing and supporting neurons, little is known about the effects of GDNF on demyelination of nerves in sepsis. In this study, we tested the hypothesis that GDNF could alleviate sepsis-induced neuromuscular dysfunction by decreasing the expression of γ- and α7-nAChR in an experimental rat model of neuromyopathy.