Acetyl-L-carnitine, ALCAR or ALC, is an acetylated form of L-carnitine. It is naturally produced by the body, although it is often taken as a dietary supplement. Acetylcarnitine is broken down in the blood by plasma esterases to carnitine which is used by the body to transport fatty acids into the mitochondria for breakdown.

Acetyl-l-carnitine (ALCAR), regulate mitochondrial metabolism and has been reported to improve cognitive functions in different neurodegenerative disorders through an unknown mechanism. For the first time, we investigated the effect of ALCAR on adult neurogenesis in the 6-hydroxydopamine (6-OHDA) induced rat model of PD-like phenotypes and also explored the possible underlying mechanism of action. A single unilateral administration of 6-OHDA into the medial forebrain bundle reduced neural progenitor cell (NPC) proliferation, long-term survival and neuronal differentiation in the hippocampus. Interestingly, chronic treatment with ALCAR (100 mg/kg/day, i.p) potentially enhanced proliferation, long term survival and neuronal differentiation of NPCs in rat model of PD-like phenotypes. ALCAR treatment stimulates cell survival related signals (AKT and BCL-2) by inhibiting cell death related cues (GSK-3β and BAX) which might be responsible for a neuroprotective effect of ALCAR in rat model of PD-like phenotypes. We conclude that ALCAR exerts neuroprotective effects against 6-OHDA-induced impairment in hippocampal neurogenesis by regulating cell survival and cell death-related signals [1)].

It is well known that acetyl-l-carnitine (ALC) has various neuroprotective effects against neurodegenerative diseases. In addition, it has been reported that ALC facilitates myelination of regenerated axons after peripheral nerve injuries. We previously reported that spontaneous regeneration of the lateral olfactory tract (LOT), the main fiber tract of the central olfactory system, consistently occurred in newborn rats and a majority of these regenerated fibers were unmyelinated in neonatally LOT-transected young adult rats. To investigate the effects of ALC treatment on myelination in LOT, neonatal rats were treated with ALC after LOT transection. Immunohistochemistry for myelin basic protein showed more positive areas in ALC-treated rats than in control rats. Moreover, the number of myelinated axons of regenerated fibers was assessed using electron microscopy and was found to be statistically higher in ALC-treated rats compared to control rats. The study revealed that ALC accelerates myelination of regenerated fibers in neonatally LOT-injured young adult rats [2)].

1)
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2)
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