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Abstracts List of Topics:

Neurosciences

501. Basic Sciences

Abstract Title:

Study of Mechanisms for Acetyl-L-Carnitine Neuroprotective Action

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Abstract Text:

Introduction: Acetyl-L-Carnitine (ALC) has been proposed to have beneficial effects in chronic neurodegenerative disorders caused by production of abnormal proteins, mitochondrial dysfunction and oxidative stress. Recently, our group demonstrated that pre-treatment with ALC confers effective neuroprotection against 3,4-methylenedioximethamphetamine (MDMA)-induced neurotoxicity. These pre-clinical studies reinforce the beneficial potential of ALC as a neuroprotectant in neurodegenerative disorders. However, little is known about the molecular mechanisms underlying ALC action.

Material and Methods: To study the molecular mechanisms involved in the neuroprotective features of ALC, we exposed PC12 cells, a cell line derived from rat adrenal pheochromocytoma, to methamphetamine (METH). The protective effect of ALC was assessed by treating cells with three concentrations of ALC (1.0; 0.1; and 0.01 mM), 30 min prior to the addition of METH (1 and 100 µM). 24h and 72h after METH treatment cell viability (MTT assay) and Dopamine (DA) and Epinephrine release (HPLC). Since some authors suggested that ALC contribute to enhanced efficiency of glucose utilization, ^{18}F-FDG uptake assay will allow to verify this issue, as well as to validate the results from MTT assay and HPLC. Medium was replaced by serum-free medium 2h before ^{18}F-FDG addition and incubated for 20 min with fresh medium, containing 37 kBq of ^{18}F-FDG/mL. Cells were washed, trypsinized and centrifuged. The pellet was lysed by adding NaOH 10mmol/L. Aliquots from cell lysates as well as supernatant were assayed for radioactivity by gamma-counting. Phase-contrast microscopic analysis was also used.

Discussion: Comparing to control, we observed a decrease in cell viability of 20% and 40%, 24h after the addition of 1 and 100 µM of METH, respectively. At 72h these figures were more pronounced (20% and 90%). Treating the cells with ALC before METH did not result in a protective effect at 24h, but at 72h cell viability was increased for intermediary concentrations of ALC when using 1µM of METH. DA production was not significantly affected at 24h. Inversely, at 72h there was an increase in DA production. However, we observed a reduction in DA concentration when the higher concentrations of METH and ALC were used concomitantly. Similar results were obtained for Epinephrine. Microscopic analysis revealed that ALC may also reduce the METH-induced altered morphology of PC12 cells.

Conclusion: As a whole, these preliminary results indicate that ALC might have a protective role that is dose and time-dependent.

Foi decidido que não será apresentada a versão integral deste documento.

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