LETTER TO THE EDITOR

Beneficial effects of exogenous CDP-choline (citicoline) in EAE

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Sir,
I read with great interest the article by Skripuletz et al. (2015), who showed beneficial effects of exogenously applied CDP-choline (citicoline) in murine myelin oligodendrocyte glycoprotein-induced experimental autoimmune encephalomyelitis (EAE) and in cuprizone-induced mouse model of demyelination. I would like to congratulate the authors for their report, which already has been recognized as the best basic science paper published in the multiple sclerosis research area in 2014 (Hutchinson, 2015).

In the discussion of their findings, Skripuletz et al. (2015) stated that, although several previous studies indicated that CDP-choline may have the capacity to protect cells such as neurons, this substance was not tested in demyelinating diseases such as multiple sclerosis. Although this statement is correct, it is worth mentioning that two independent groups of investigators have previously tested citicoline in EAE; their positive findings have been communicated at scientific meetings, and the abstracts published in journal supplements.

The first abstract (Clayton et al., 1999) describes experiments in which EAE was induced in female Lewis rats and female PLSJLF1/J mice by injection of heterologous spinal cord and myelin basic protein (both in complete Freund's adjuvant), respectively. Treatments included citicoline (500 mg/kg, i.p.), dexamethasone (DEX, 37.5 mg/kg s.c.) or saline starting on Day 9 (rats) or Day 12 (mice) post-inoculation. Animals were scored daily for muscle weakness on a 4-point scale. In both EAE models, citicoline and dexamethasone were effective in significantly diminishing the severity of the muscle weakness as compared to saline controls. Additionally, while DEX treatment was associated with a progressive weight loss, citicoline-treated animals maintained normal weight gain.

The second abstract (Grieb et al., 2004) relates to a poster presented at the 56th meeting of the American Academy of Neurology in San Francisco. The aim of our study was to test whether citicoline influences the intensity of CNS inflammation in EAE produced by inoculation of Lewis rats with guinea pig brain homogenate. Treatment with citicoline sodium salt (500 mg/kg, i.p.) or saline was started 7 days after inoculation and lasted 7 days. Intensity of inflammation was evaluated by counting inflammatory infiltrations in haematoxylin and eosin-stained transverse sections of the brain at the level of optic nerves, and cervical and lumbar spinal cord. Treatment with citicoline significantly reduced the average number of inflammatory infiltrates per section in all locations; the largest effect was noted in brain and cervical cord white matter.

I recall the aforementioned abstracts not for claiming priority in discovering beneficial effects of citicoline in EAE (we were not the first), but rather as a ‘reverse confirmation’ of the data presented by Skripuletz et al. (2015). I agree with these authors that citicoline, currently classified as a food supplement both in the European Union and USA, is a promising substance for patients with multiple sclerosis. However, we shall take into account that ingestion or injection of this substance does not provide an increase in intracellular CDP-choline in the brain, and that the mechanism of neuroprotective and neuroregenerative effects of citicoline remains currently unknown (Grieb, 2014).

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