Effect of vitamin D supplementation on axonal damage in relapsing-remitting multiple sclerosis

Egil Røsjø1*, Trygve Holmøy1,2, Henrik Zetterberg3,4,5,6, Kaj Blennow3,4, Jonas Christoffer Lindstrøm2,7, Linn Hofsøy Steffensen8,9, Margitta T. Kampman8

1 Department of Neurology, Akershus University Hospital, Lørenskog, Norway
2 Institute of Clinical Medicine, University of Oslo, Oslo, Norway
3 Department of Psychiatry and Neurochemistry, Institute of Neuroscience and Physiology, the Sahlgrenska Academy at the University of Gothenburg, Mölndal, Sweden
4 Clinical Neurochemistry Laboratory, Sahlgrenska University Hospital, Mölndal, Sweden
5 Department of Neurodegenerative Disease, UCL Institute of Neurology, Queen Square, London, UK
6 UK Dementia Research Institute at UCL, London, UK
7 Services and Research Centre, Akershus University Hospital, Lørenskog, Norway
8 Department of Neurology, University Hospital of North Norway, Tromsø, Norway
9 Department of Clinical Medicine, University of Tromsø, Tromsø, Norway

Short title: Vitamin D and axonal damage in RRMS

*Presenting author;

Egil Røsjø

Neurological Department, Akershus University Hospital, Lørenskog, Norway

Email: egil.rorvik.rosjo@ahus.no
Abstract

Background

The effect of vitamin D supplementation in relapsing-remitting multiple sclerosis (RRMS) is not established. Neurofilament light chain (NFL) is a sensitive biomarker for axonal damage and an inverse relationship has been found between NFL and 25-hydroxyvitamin D (25(OH)D) levels in RRMS.

Material and methods

To investigate this further, we have analyzed the association between serum levels of NFL and 25(OH)D in a two-year randomized placebo-controlled trial of high-dose oral vitamin D3 supplementation (20,000 IU/week) in 68 RRMS patients (NCT00785473).

Results

In contrast to earlier reports, we found a positive baseline correlation between the serum concentrations of 25(OH)D and NFL ($r=0.25$, $p=0.04$). However, despite the mean 25(OH)D level increased by approximately 70 nmol/L in the vitamin D group, no effect was noted on the change in NFL levels from baseline to week 48 ($p=0.93$) or week 96 ($p=0.56$) when compared to the placebo group. Still, in a subgroup analysis restricted to patients without disease modifying therapy at baseline, a strong trend was detected towards an effect of vitamin D with a decrease of 30.9% from baseline to week 48 and 32.6% to week 96 when alterations in NFL concentrations were compared between the vitamin D and the placebo group ($p=0.06$ for both times).

Conclusion

We conclude that with a possible exception for patients without disease modifying treatment, weekly oral supplementation with 20,000 IU vitamin D3 seems to have no clear effect on axonal damage in RRMS.