# Researchers explore role of vitamin D in remyelination

**Tuesday, December 29, 2015**

Start

Research reported in the *Journal of Cell Biology* on December 7, 2015 reveals a role for vitamin D in remyelination of the central nervous system.

"Remyelination involves the generation of new myelin sheath–forming oligodendrocytes after primary demyelination in the central nervous system," explain Robin J. M. Franklin of the University of Cambridge and colleagues. "In common with other regenerative processes, remyelination efficiency declines with aging, with the result that in chronic demyelinating diseases such as multiple sclerosis (MS), remyelination becomes ineffective."

In previous research, Dr Franklin and associates identified the nuclear receptor retinoid X receptor gamma (RXRgamma) as a regulator of oligodendrocyte progenitor cells. Because nuclear receptors usually function in pairs, the team sought to find RXRgamma's binding partners. They found that RXRgamma binds to several nuclear receptors in oligodendrocytes and their progenitor cells, including the vitamin D receptor. Inhibition of the vitamin D receptor impaired the progenitor cells' ability to differentiate, thereby reducing their ability to remyelinate nerve axons, however, vitamin D, which binds to and activates the vitamin D receptor, improved oligodendrocyte progenitor cell differentiation.

Examination of post-mortem brain lesions from individuals with multiple sclerosis revealed that the vitamin D receptor was highly expressed, indicating significant relevance of the current finding to human disease. Epidemiologic studies have established an association between MS risk and vitamin D deficiency; however, the vitamin's immunomodulatory role has been the focus of research that has sought to determine its mechanism of action.

"Our data suggest that hypovitaminosis D in MS patients may be a contributor to remyelination failure," the authors conclude. "Further investigation into the molecular mechanisms of VDR in remyelination will open up new opportunities for the development of regenerative medicines for demyelinating diseases."

## What's Hot

### Higher vitamin D levels predict reduced rate of progression in early MS



Having a higher serum 25-hydroxyvitamin D level upon an initial episode suggestive of **multiple sclerosis** (MS) could reduce the chance of a second episode or other signs of disease progression according to an article published in the March 2014 issue of the American Medical Association journal *JAMA Neurology*.

The study evaluated data from 465 participants in the Betaferon/Betaseron in Newly Emerging multiple sclerosis for Initial Treatment (BENEFIT) trial, which evaluated early versus delayed interferon beta-1b treatment in patients with an initial episode of neurological dysfunction suggestive of MS. (Definitive diagnosis of MS requires the occurrence of a second clinical event.) The subjects were followed for up to five years, during which magnetic resonance imaging (MRI) was conducted quarterly for 12 months, and at 18, 24, 36, 48 and 60 months. For the current study, Alberto Ascherio, MD, DrPH, and his associates examined the association between the risk of disease progression and serum 25-hydroxyvitamin D levels measured at least once among four time points over the two years following enrollment in the BENEFIT trial.

Having a higher serum level of vitamin D was associated with a reduced rate of conversion to MS over the follow-up period. "By the end of the follow-up at 60 months, those patients with serum 25(OH)D concentrations greater than or equal to 50 nmol/L [20 ng/mL] had a 4-times lower change in T2 lesion volume, a 2-fold lower rate of brain atrophy, and lower disability than those below 50 nmol/ [20 ng/mL]," the authors report.

"Our results suggest that identification and correction of vitamin D insufficiency has an important role in the early treatment of MS," they conclude.

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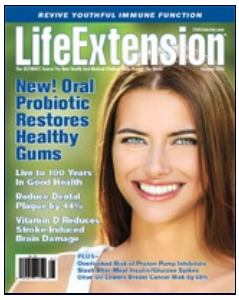


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## Health Concern

### Multiple sclerosis

Neuronal communication is similar to the transmission of an electrical current through a series of wires. Droves of neurons work together to deliver messages to every corner of the body by transmitting signals along their long, cylindrical mid-sections called *axons* (see figure at left) and passing it on to the next neuron. This is repeated until the message reaches its destination. Like electrical wires, neuronal axons require insulation to ensure that they are able to transmit a signal accurately, and at high speeds. Specialized cells called oligodendrocytes provide this insulation to neurons by wrapping the axons in an insulating material called myelin. Without this myelin sheath, neuronal communication becomes nearly impossible, and neurons become susceptible to damage.

Remyelination is the process by which demyelinated axons are naturally re-wrapped with myelin, restoring nerve conduction and functionality (Smith 1979). This phenomenon is the result of oligodendrocytes repairing the damage to the myelin sheath that occurs during an episode of increased disease activity. However, as the disease progresses over years (usually decades) the oligodendrocytes begin to lose their ability to repair the damage, and symptoms become progressively worse and episodes more frequent due to remyelination failure.

It is now known that MS occurs more frequently in individuals with lower blood levels of vitamin D. A study published in the

prestigious *Journal of the American Medical Association* found that, compared to those with the highest vitamin D blood levels, those with the lowest blood levels were 62% more likely to develop MS.

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