

## A supplement for myelin regeneration

7 December 2015



suggest that the vitamin might also affect disease progression by controlling <u>myelin sheath</u> regeneration, a critical step to alleviate the disease's symptoms that fails as patients age. VDRactivating drugs might therefore be able to enhance remyelination in multiple sclerosis patients and in <u>patients</u> suffering from other demyelinating diseases.

More information: De la Fuente, A.G., et al. 2015. *J Cell Biol.* dx.doi.org/10.1083/jcb.201505119

In a control brain slice (left), most axons (red) have regained a myelin sheath (green) eight days after demyelination. But regeneration is impaired when VDR is inhibited (right). Credit: De la Fuente et al., 2015

Provided by Rockefeller University Press

Multiple sclerosis patients continually lose the insulating myelin sheath that wraps around neurons and increases the speed of impulses in the central nervous system. Whenever neurons are demyelinated, OPCs migrate toward these cells and differentiate into mature, myelin-producing oligodendrocytes, but this process becomes less and less effective as people age.

A nuclear receptor protein called retinoid X receptor gamma (RXRgamma) is known to promote OPC differentiation and remyelination, but, because <u>nuclear receptors</u> generally function in pairs, a team of researchers led by Robin Franklin at the University of Cambridge, UK, set out to identify RXRgamma's binding partners and investigate their possible role in remyelination.

RXR? bound to several nuclear receptors, including VDR, in OPCs and mature oligodendrocytes. Inhibiting VDR impaired OPC differentiation and reduced the cells' ability to remyelinate axons ex vivo. In contrast, Vitamin D, which binds and activates VDR, boosted OPC differentiation.

Low <u>vitamin</u> D levels have been linked to the onset of multiple sclerosis, and the researchers' findings



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