A supplement for myelin regeneration

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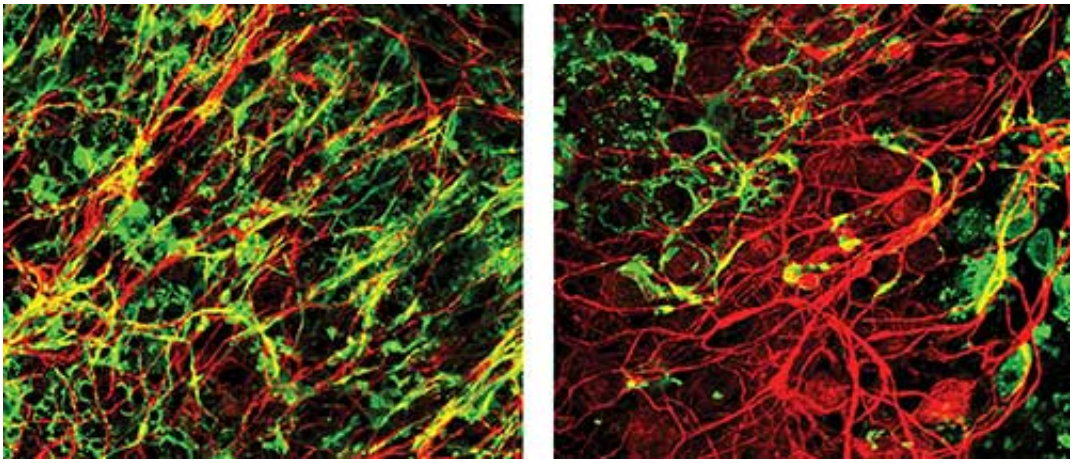

In a control brain slice (left), most axons (red) have regained a myelin sheath (green) eight days after demyelination. But regeneration is impaired when VDR is inhibited (right). Credit: De la Fuente et al., 2015

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 nuclear receptors 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.

RXRγ 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 vitamin D levels have been linked to the onset of multiple sclerosis, and the researchers' findings suggest that the vitamin might also affect disease progression by controlling myelin sheath regeneration, a critical step to alleviate the disease's symptoms that fails as patients age. VDR-activating drugs might therefore be able to enhance remyelination in multiple sclerosis patients and in patients 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

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