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Oligodendroglial NMDA Receptors Regulate Glucose Import and Axonal Energy Metabolism

Saab AS, Tzvetavona ID, Trevisiol A, Baltan S, Dibaj P, Kusch K, Möbius W, Goetze B, Jahn HM, Huang W, Steffens H, Schomburg ED, Pérez-Samartín A, Pérez-Cerdá F, Bakhtiari D, Matute C, Löwel S, Griesinger C, Hirrlinger J, Kirchhoff F, Nave KA.

Oligodendrocytes make myelin and support axons metabolically with lactate. They express different glutamate receptors, including NMDA receptors of unknown function. A role for NMDA receptor signaling in oligodendrocyte differentiation and promoting myelination has been suggested. In this article, the authors aim to teste whether NMDA receptor signaling to oligodendrocytes is the missing link in coupling axonal activity and ATP consumption to oligodendroglial glucose utilization and lactate supply. They found that activation of oligodendroglial NMDA receptors stimulates GLUT1 export and release of lactate. Mouse mutants lacking NMDA receptors from oligodendrocytes are delayed in myelination. Targeted inactivation of oligodendroglial NMDA receptors *in vivo* impairs axonal energy metabolism and causes late-onset axonopathy. Their study reveals a novel aspect of neuronal energy metabolismin which activity-dependent glutamate release enhances oligodendroglial glucose uptake and glycolytic support of fast spiking axons.