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"*C1ql1* expression in oligodendrocyte progenitor cells promotes oligodendrocyte differentiation"

Abstract: Myelinating oligodendrocytes arise from the stepwise differentiation of oligodendrocyte progenitor cells (OPCs). Approximately 5% of all adult brain cells are OPCs. Why would a mature brain need such a large number of OPCs? New myelination is possibly required for higher-order functions such as cognition and learning. Additionally, this pool of OPCs represents a source of new oligodendrocytes to replace those lost during injury, inflammation, or in diseases such as multiple sclerosis (MS). How OPCs are instructed to differentiate into oligodendrocytes is poorly understood, and for reasons presently unclear, resident pools of OPCs are progressively less utilized in MS. The complement component 1, q subcomponent-like (C1QL) protein family has been studied for their functions at neuron-neuron synapses, but we show that OPCs widely express *C1ql1*. We create OPC-specific conditional knockout mice and show that C1QL1 promotes the differentiation of OPCs into oligodendrocytes and enhances myelin production both during development and during recovery from cuprizone-induced demyelination. We further use primary cultured OPCs to show that recombinant C1QL1 causes enhanced OPC differentiation *in vitro*. Our results suggest that C1QL1 may initiate a previously unrecognized signaling pathway to promote new oligodendrocyte production with relevance for possible novel therapies of demyelinating diseases and may illuminate a novel mechanism to regulate myelination's function in cognition and learning.