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Neuron–oligodendroglial interactions in health and malignant disease

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Abstract

Experience sculpts brain structure and function. Activity-dependent modulation of the myelinated infrastructure of the nervous system has emerged as a dimension of adaptive change during childhood development and in adulthood. Myelination is a richly dynamic process, with neuronal activity regulating oligodendrocyte precursor cell proliferation, oligodendrogenesis and myelin structural changes in some axonal subtypes and in some regions of the nervous system. This myelin plasticity and consequent changes to conduction velocity and circuit dynamics can powerfully influence neurological functions, including learning and memory. Conversely, disruption of the mechanisms mediating adaptive myelination can contribute to cognitive impairment. The robust effects of neuronal activity on normal oligodendroglial precursor cells, a putative cellular origin for many forms of glioma, indicates that dysregulated or 'hijacked' mechanisms of myelin plasticity could similarly promote growth in this devastating group of brain cancers. Indeed, neuronal activity promotes the pathogenesis of many forms of glioma in preclinical models through activity-regulated paracrine factors and direct neuron-to-glioma synapses. This synaptic integration of glioma into neural circuits is central to tumour growth and invasion. Thus, not only do neuron-oligodendroglial interactions modulate neural circuit structure and function in the healthy brain, but neuron-glioma interactions also have important roles in the pathogenesis of glial malignancies.