O S

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spatial extent of OPC and astrocyte production in the ventral cord is limited by mutually antagonistic actions of SHH and BMPs.

The majority (approximately 80%) of OPCs in the spinal cord are generated in the ventral VZ. The remainder are generated in other progenitor domains, including dorsal domains dP3–5. The dorsally derived OPCs appear later in development than the pMN-derived OPCs (approximately E15 vs. E12.5) and they migrate less widely than their ventrally derived counterparts. It seems unlikely that these dorsal progenitor domains are under the influence of SHH from the floor plate, suggesting that there might be a SHH-independent route to OPC specification. Indeed, OPCs can arise in cultures derived from SHH null spinal cord or in the presence of cyclopamine, if fibroblast growth factor-2 (FGF-2) is also present in the culture medium. OPC generation in the dorsal spinal cord might therefore depend on FGF signaling, possibly combined with a decline in BMP expression in the dorsal cord during late embryogenesis. There might even be a biochemical overlap between SHH and FGF signaling because it has been shown that both pathways depend on MAP kinase activity.

It is not known whether or not ventrally and dorsally derived OPCs in the spinal cord are functionally specialized. However, there is evidence that ventrally and dorsally derived OPCs in the forebrain can substitute for one another, implying that they are functionally equivalent.

OPC G[illegible] [illegible] F [illegible]

The adult forebrain, including the cerebral cortex, develops from an embryonic structure called the telencephalon. Like the spinal cord, this starts as a simple neuroepithelial tube, although it becomes progressively more convoluted during development. There is no notochord underlying the telencephalon and no floor plate; however, the ventral neuroepithelial cells express SHH and its receptors, Patched (PTC) and Smoothened (SMO). At approximately E13.5, some cells in

Of course, there might be subtle differences that would not be detected without detailed behavioral analysis of the ablated mice, but this remains to be investigated.

R T a F a OPC D m : T OLIG G

A major step forward in understanding the molecular control of oligodendrocyte lineage development resulted from the discovery of transcription factors that orchestrate OPC specification and differentiation. Prime among these are the oligodendrocyte lineage (OLIG) transcription factors, OLIG1 and

oligodendrocytes are needed during normal healthy life, why large numbers of OPCs should survive in the adult has been puzzling.

It has been recognized that OPCs in the adult make contact with nodes of Ranvier (the gaps between adjacent myelin sheaths on an axon), they receive synaptic

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