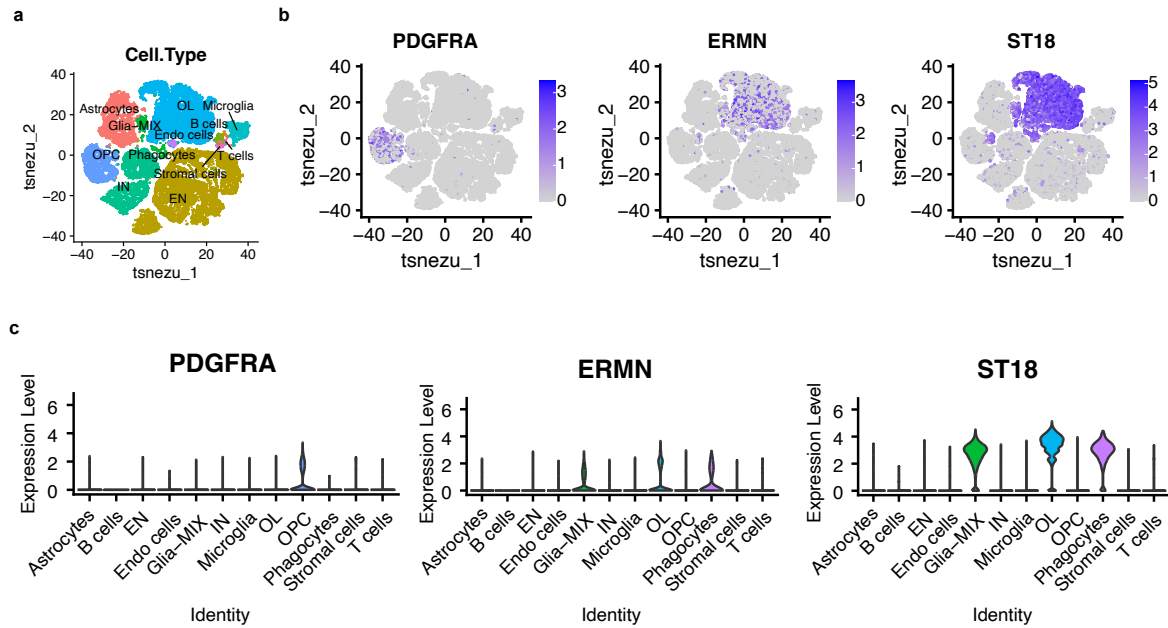
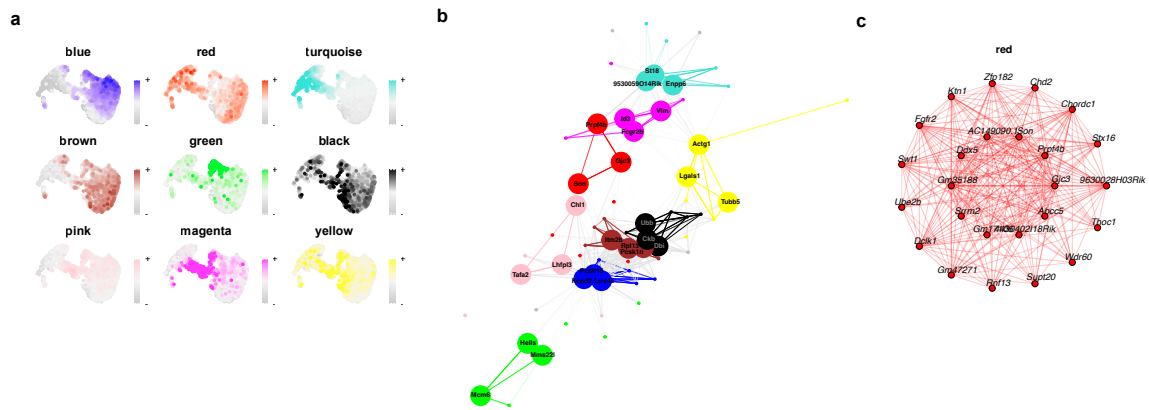




Extended figure S1.

ST18 expression is elevated in oligodendrocyte lineage cells in human MS tissue.

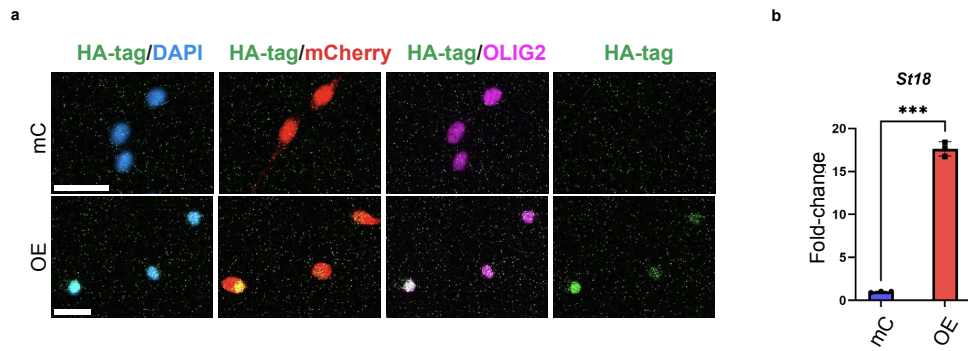
a, t-SNE projection of all cell types captured by Schirmer *et al.* (2019) in white- and grey-matter MS brain lesions. EN, excitatory neurons; IN, inhibitory neurons; OL, oligodendrocytes (65,217 nuclei). **b-c**, Expression of ST18, PDGFRA, BCAS1 and ERMN represented as UMAP projection (b) or violin plots (c).



Extended figure S2.

Co-expression network analysis and module hub gene identification during OPC-to-NFOL transition.

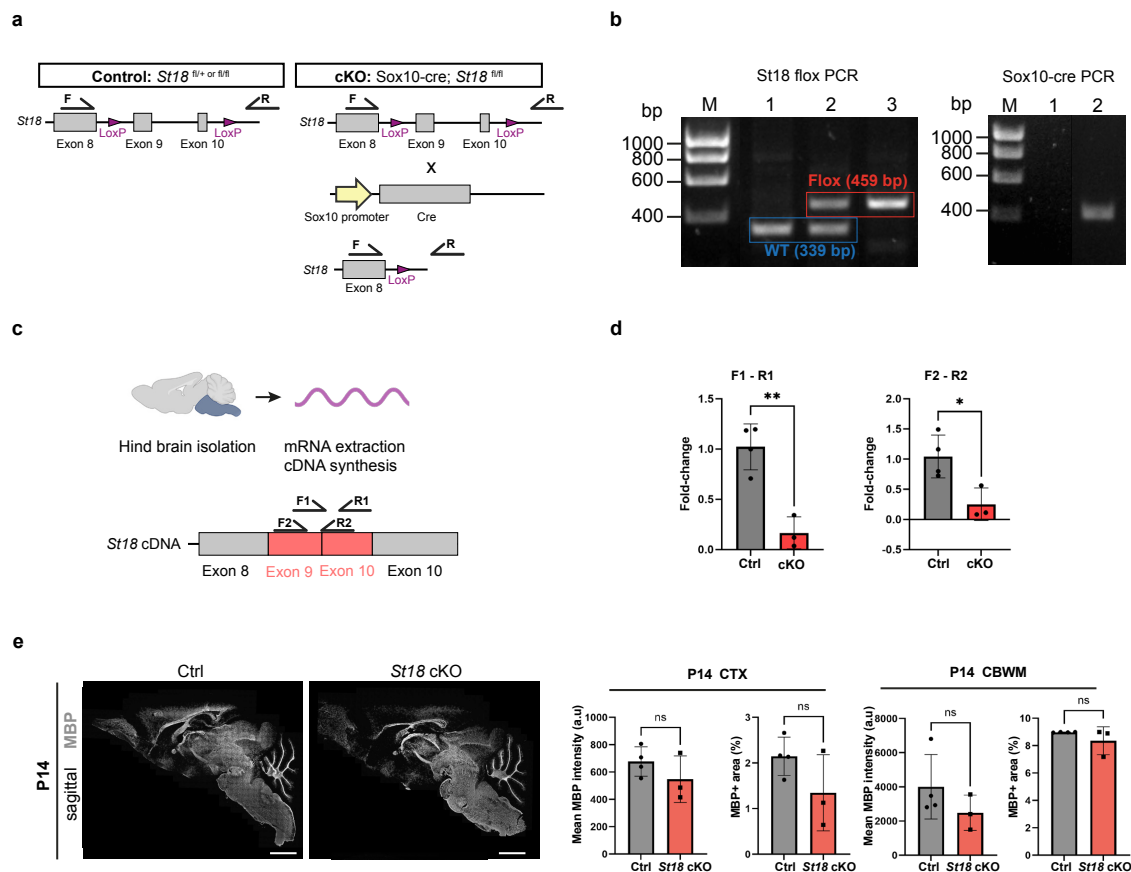
a, UMAPs coloured by module eigengene (ME) scores reveal spatial module expression across OPC/NFOL states. **b**, integrated gene co-expression network combining all modules, with nodes coloured by module identity; top three hub genes per module are labelled. **c**, Module-specific co-expression networks for the red modules. The top 10 hub genes, ranked by kME, are positioned at the centre.



Extended figure S3.

Validation of lentiviral-mediated *St18* overexpression in primary rat oligodendrocytes.

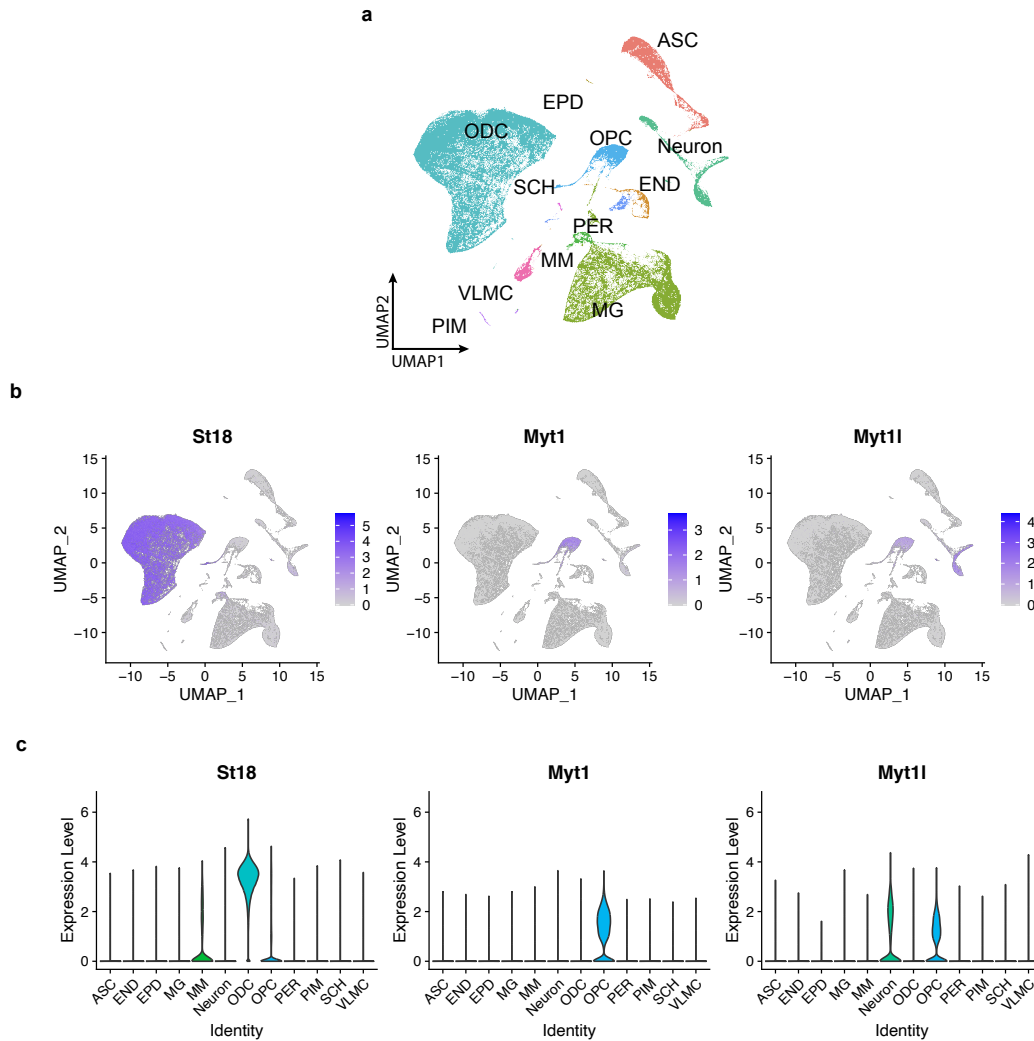
a, Immunofluorescence imaging of HA-tag together with Olig2, mCherry and DAPI of primary rat oligodendrocytes overexpressing *St18* (OE) or mCherry control (mC). **b**, Quantification of *ST18* expression level in primary rat oligodendrocytes overexpressing *St18* (OE) or mCherry control (mC), measured by RT-qPCR relative to actin (n=3). Data are represented as mean $\pm$ SEM. Significance was assessed using the two-tailed unpaired Student's t-test. *** $p < 0.001$. Scale bar: 25 μ m.



Extended figure S4.

Generation and validation of oligodendrocyte-specific *St18* knockout mice.

a, Schematic strategy of *St18* conditional knockout (cKO) using Cre-lox recombination. Cre recombination results in the deletion of exon 9 and 10 resulting in a frameshift mutation. Forward (F) and reverse (R) primer binding sites for genotyping are indicated. **b**, Representative genotyping PCR results from isolated genomic DNA showing amplification of the wild-type (WT, 339 bp) and floxed (459 bp) *St18* alleles (left), and the presence or absence of the Sox10-cre transgene (right). **c**, Schematic of hindbrain dissection and workflow for mRNA isolation, cDNA synthesis, and RT-qPCR. Primers were designed to bind the floxed exons in *St18* cKO mice. **d**, RT-qPCR results showing fold-change in *St18* expression for each primer pair illustrated in (c) in Control mice or *St18* cKO mice. **e**, MBP Immunofluorescence imaging of brain sagittal section of *St18* cKO and control P14 mice with quantification within the cortex (CTX) and cerebellum white matter (CBWM) (n=4). Scale bar: 1 mm. Data represent mean $\pm$ SD. * $p < 0.05$, ** $p < 0.01$; ns, not significant; unpaired t-test.



Extended figure S5.

Expression analysis of *Myt1*, *Myt1l* and *St18* across spinal cord cell populations.

a, UMAP projection of all spinal cord cell types. ASC, astrocytes; END, endothelial cells; EPD, ependymal cells; MG, microglia; MM, monocyte-derived macrophage; ODC, oligodendrocytes; OPC, oligodendrocyte progenitor cells; PER, pericytes; PIM, perivascular infiltrating macrophages; SCH, Schwann cells; VLMC, vascular and leptomeningeal cells (157,563 nuclei). **b-c**, Expression of *Myt1*, *Myt1l* and *St18* represented as UMAP projection (**b**) or violin plots (**c**).