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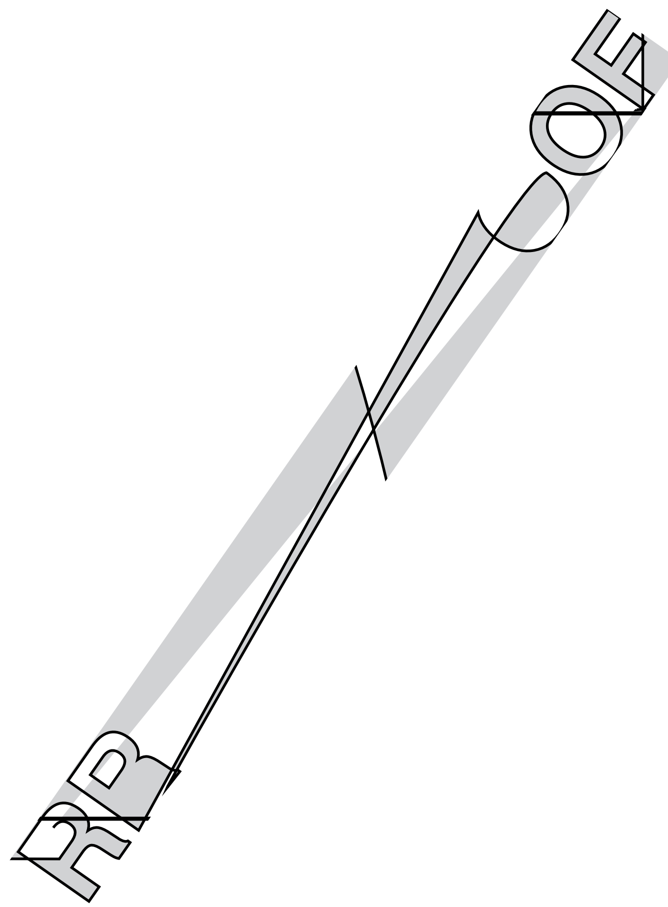

delay in the appearance of differentiated OLs but no long-term myelin deficit. However, a subsequent study by Xin et al. [6], who crossed the original line with FLP-expressing mice to remove the Pgk-Neo selection cassette (leaving behind Olig1-Cre), found a severe myelination defect leading to early postnatal lethality. Apart from this contested role in OL lineage development, Olig1 is known to be required for remyelination of experimentally-induced demyelinated lesions in the mouse spinal cord [7].

Given the central role of the Oligs in OL lineage development, it is important to try to settle the controversy over the developmental requirement for Olig1. This might have added significance because the Olig1 null locus [1,6] contains an expressed Cre cassette under Olig1 transcriptional control and these Olig1(+/Cre) mice are being used to delete floxed genes specifically in OL lineage cells. For example, conditional deletion of Dicer1 (flox/flox) using Olig1 (+/Cre) [6] caused severe impairment of myelination and death around P21 [8], whereas analogous experiments using Olig2(+/Cre) or Cnp(+/Cre) resulted in only slightly delayed myelination with full recovery by P60 [9]. In another ex[illegible]