

Visual [impairment](#) in [diabetes](#) is a growing [public health](#) concern. Apart from the well-defined diabetic [retinopathy](#), disturbed [optic nerve](#) function, which is dependent on the [myelin sheath](#), has recently been recognized as an early feature of visual impairment in diabetes. However, the underlying cellular mechanisms remain unclear. Using a [streptozotocin](#)-induced diabetic mouse model, Wu et al. observed a [myelin](#) deficiency along with a disturbed composition of [oligodendroglial](#) lineage cells in the diabetic [optic nerve](#). They found that new myelin deposition, a continuous process that lasts throughout adulthood, was diminished during [pathogenesis](#). Genetically dampening newly generated myelin by conditionally deleting [olig2](#) in [oligodendrocyte](#) precursor cells within this short time window extensively delayed the signal transmission of the adult [optic nerve](#). In addition, [clemastine](#), an antimuscarinic compound that enhances myelination, significantly restored oligodendroglia and promoted the functional recovery of the [optic nerve](#) in diabetic mice. The results point to the role of new [myelin](#) deposition in [optic neuropathy](#) under diabetic insult and provide a promising therapeutic target for restoring [visual function](#) ¹⁾

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Wu H, Chen X, Yu B, Zhang J, Gu X, Liu W, Mei F, Ye J, Xiao L. Deficient deposition of new [myelin](#) impairs adult [optic nerve](#) function in a murine model of diabetes. *Glia*. 2023 Jan 20. doi: 10.1002/glia.24341. Epub ahead of print. PMID: 36661098.

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Last update: **2024/06/07 02:58**

