

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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