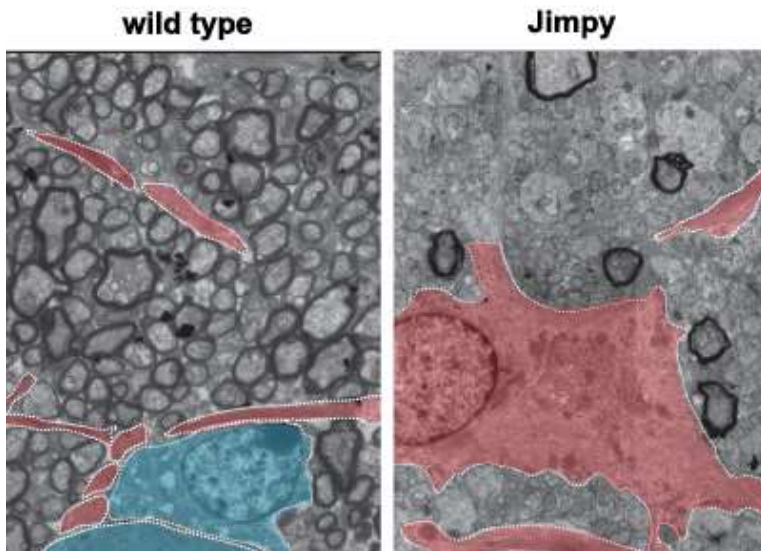# "Effects of the Jimpy mutation on mouse retinal structure and function"

wild type | Jimpy

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3:00 – 4:00 PM

Science 2, room 109

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The Jimpy mutant mouse has a point mutation in the proteolipid protein gene (*plp1*). The resulting misfolding of the protein leads to oligodendrocyte death, myelin destruction and failure to produce adequately myelinated axons in the central nervous system (CNS). It is not known how the absence of normal myelination during development influences neural function. We characterized the Jimpy mouse retina in order to find out if lack of myelination in the optic nerve during development has an effect on normal functioning and morphology of the retina. Optokinetic reflex measurements showed that Jimpy mice had, in general, a functional visual system. Both PLP1 antibody staining and RT-PCR for *plp1* mRNA showed that *plp1* is not expressed in the wild type retina. However, in the optic nerve, *plp1* is normally expressed, and consequently, in Jimpy mutant mice, myelination of axons in the optic nerve was mostly absent. Nevertheless, neither axon count nor axon ultrastructure in the optic nerve was affected. Physiological recordings of ganglion cell activity using microelectrode arrays revealed a decrease of stimulus-evoked activity at mesopic light levels. Morphological analysis of the retina did not show any significant differences in the gross morphology, such as thickness of retinal layers or cell number in the inner and outer nuclear layer. The cell bodies in the inner nuclear layer, however, were larger in the retina of Jimpy mutant mice. Antibody labeling against cell type specific markers showed that the number of rod bipolar and horizontal cells was increased in Jimpy mice. In conclusion, while the Jimpy mutation has dramatic effects on the myelination of retinal ganglion cell axons, it has moderate effects on retinal morphology and function