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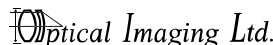
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Portion of lobule VII of cerebellum from the mouse model of the lysosomal disease, late-infantile neuronal ceroid lipofuscinosis (CLN2 disease). Immunofluorescence labeling shows calbindin-positive Purkinje cells (red) and DAPI-labeled nuclei (blue) in relation to aberrant accumulation of the macroautophagy adapter protein p62/Sqstm1 (green) in protein aggregates. p62 responds to lysosomal membrane permeability in CLN2 disease by sequestering released lysosomal content in intraneuronal aggregates. Image generated by M. C. Micsenyi.
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Large numbers of retinal ganglion cells (red) project aberrantly to the opposite eye in developing mutant mouse embryos lacking the heparan sulfotransferase enzyme Hs6st1.

Courtesy, with Permission: Thomas Pratt, Christopher D. Conway, Natasha M. M.-L. Tian, David J. Price, and John O. Mason, 2006, *The Journal of Neuroscience*, 26: 6911-6923.

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A merged image taken by confocal microscopy showing the extent of infiltration (Hoechst 33258; blue), demyelination (myelin basic protein; green), and axonal loss (neurofilament 200; red) in a lesion site of the lumbar spinal cord of a WldS mouse with induced experimental autoimmune encephalomyelitis (EAE). Many axons are preserved within the EAE lesions in WldS but not wild-type mice.

Courtesy, with permission: Shinjiro Kaneko, Jing Wang, Marie Kaneko, Glenn Yiu, Joanna M. Hurrell, Tanuja Chitnis, Samia J. Khoury, and Zhigang He, 2006, *The Journal of Neuroscience*, 26: 9794-9804.

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Choroid plexus epithelial cells (CPECs) differentiated from human embryonic stem cell-derived neuroepithelial progenitors. CPEC markers TTR and ZO1 are marked in red and green, respectively, with Hoechst nuclear counterstaining in blue.

Courtesy, with permission: Momoko Watanabe, Young-Jin Kang, Lauren M. Davies, Sanket Meghpara, Kimbley Lau, Chi-Yeh Chung, Jaymin Kathiriyi, Anna-Katerina Hadjantonakis, and Edwin S. Monuki, 2012, *The Journal of Neuroscience*, 32: 15934-15945.

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