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Program Cover
This image shows dentate granule cells in the hippocampus of an adult mouse that lacks TRIM9 ubiquitin ligase. These cells, labeled with red and green florescent proteins, exhibit occasional ectopic migration into the molecular layer. **Courtesy, with permission:** Cortney C. Winkle, Reid H. J. Olsen, Hyojin Kim, Sheryl S. Moy, Juan Song and Stephanie L. Gupton, 2016, *The Journal of Neuroscience* 36(18): 4940-4958.

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Confocal micrograph of cultured hippocampal neurons stained for neuronal nitric oxide synthase (nNOS, grey-blue) and the postsynaptic protein gephyrin (red). nNOS activity modulates the clustering of gephyrin at GABAergic synapses. **Courtesy, with permission:** Borislav Dejanovic and Guenter Schwarz, 2014, *The Journal of Neuroscience* 34(23) 7763-7768.

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Thalamic axonal arbors from corticothalamic neurons of the primary somatosensory (S1) cortex. Colorized fluorescent image from an *in vitro* slice containing EYFP-expressing corticothalamic fibers originating from a small injection of virus transducing channelrhodopsin2-EYFP into deep S1 cortex. **Courtesy, with permission:** Seung-Chan Lee, Saundra L. Patrick, Kristen A. Richardson and Barry W. Connors, 2014, *The Journal of Neuroscience* 34(39) 13170-13182.

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Longitudinal sections of sciatic nerves from neuropathic PMP22-null mice were double-labeled with the lipid raft marker cholera toxin subunit β (in red) and phalloidin (in green) to illustrate the severe disruption of lipid raft and actin network in myelinating Schwann cells lacking PMP22. Nuclei are shown in blue. **Courtesy, with permission:** Sooyeon Lee, Stephanie Amici, Hagai Tavori, Waylon M. Zeng, Steven Freeland, Sergio Fazio and Lucia Notterpek, 2014, *The Journal of Neuroscience* 34(48) 16140-16152.

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This watercolor by Biuse Guivernau was inspired by immunocytofluorescence images of cultured hippocampal mouse neurons treated acutely with β-amyloid. β-amyloid oligomers (and nitrated beta-amyloid oligomers) not only induce neuronal death, but also impair neuronal function, by binding to dentritic spines and synapses. The image represents the functional isolation of neurons resulting from amyloid buildup in a brain affected by Alzheimer's disease. **Courtesy, with permission:** Cortney C. Winkle, Barbara A. Sorg, Sabina Berretta, Jordan M. Blacktop, James W. Fawcett, Hiroshi Kitagawa, Jessica C.F. Kwok and Marta Miquel, 2016, *The Journal of Neuroscience* 36(45): 11459-11468.

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This composite illustration shows oligodendrocytes (labeled with antibodies against myelin basic protein, white), which were induced to differentiate in culture by treatment with an antibody against the membrane protein LINGO-1. This image is superimposed on an image of a demyelinated brain lesion from autopsy tissue of a multiple sclerosis (MS) patient, which shows myelin (myelin basic protein, pink), axons (neurofilament, red), and LINGO-1 (green). LINGO-1 is upregulated in MS lesions, and blocking LINGO-1 function promotes remyelination in animal models of MS. **Courtesy, with permission:** Zhaohui Shao, Xinhua Lee, Guanrong Huang, Guoqing Sheng, Christopher E. Henderson, Daniel Louvard, Jiho Sohn, Blake Pepinsky and Sha Mi, 2017, *The Journal of Neuroscience* 37(12): 3127-3137.

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This confocal image shows a retina prepared from a GLT1-EGFP mouse. The widely used astrocyte marker GLT1 (green) is highly expressed by neurons in the outer nuclear layer, while SOX9 (purple) is most prominent on Müller glial cells, as identified by glutamine synthetase (white). Cell nuclei are labeled by DAPI (blue). **Courtesy, with permission:** Wei Sun, Adam Cornwell, Jiashu Li, Sisi Peng, M. Joana Osorio, Nadia Aalling, Su Wang, Abdellatif Benraiss, Nanhong Lou, Steven A. Goldman and Maiken Nedergaard, 2017, *The Journal of Neuroscience* 37(17): 4493-4507.

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A thalamocortical slice from a 4-dayold mouse brain in which neurons in the ventrobasal thalamus express Cre recombinase and tdTomato, allowing visualization of thalamocortical axons (red) innervating the barrel cortex. Layer 6 corticothalamic neurons (green) were labeled by an antibody to the transcription factor TBR1, and all other cell bodies were counterstained with ToPro (blue). The same Cre line was crossed with a channelrhodopsin reporter for optogenetically guided dual recording experiments from connected thalamic and cortical neurons, as described in the article by Hu and Agmon. **Courtesy, with permission:** Hang Hu and Ariel Agmon, 2016, *The Journal of Neuroscience* 36(26) 6906-6916.

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Fluorescence micrograph of a glial microdot island containing reciprocally connected hippocampal neurons, a GABAergic (blue) and a glutamatergic (red) neuron. MPTS (red) or Alexa-568 (blue) was infused during double whole-cell recordings. Both neurons were transduced with Synaptophysin-pHluorin, which allowed the identification of active glutamatergic and GABAergic synapses after train stimulation of either neuron (glutamatergic synapses: green spots; GABAergic synapses: white spots). Comparing the number of active synapses to the rate of mEPSC and mIPSC showed that innervation by a GABAergic neuron downregulates spontaneous release rates in glutamatergic neurons. **Courtesy, with permission:** Keimpe D. B. Wierda and Jakob B. Sørensen, 2014, *The Journal of Neuroscience* 34(6) 2100-2110.

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Confocal micrograph of a day 6 coculture of oligodendrocytes and dorsal root ganglion neurons, depicting a control and a mutant (Ilk–/–) oligodendrocyte. The control cell extends arbors that contact neighboring neuronal processes and produce membranous leaflets. In contrast, the Ilk-null oligodendrocyte is deficient in this ability. Green fluorescent protein (green) is expressed upon loss of Ilk, thereby labeling mutant cells. The sample was labeled with antibodies against neurofilament-200 (blue), myelin-basic protein (red), and stained with 4′,6-diamidino-2-phenylindole (white). **Courtesy, with permission:** Ryan W. O'Meara, John-Paul Michalski, Carrie Anderson, Kunal Bhanot, Peter Rippstein and Rashmi Kothary, 2013, *The Journal of Neuroscience* 33(23) 9781-9793.

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Layer III pyradimal cell of cerebral cortex of mouse from an original preparation of Santiago Ramón y Cajal impregnated with the Golgi method (P80001). Z-projection (32 sections; z-step, 2.072 µm). Objective, 20×; numerical aperture, 0.75 (ImageJ). **Courtesy, with permission:** Pablo García-López, Virginia García-Marín and Miguel Freire, 2006, *The Journal of Neuroscience* 26(44): 11249-11252.

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This confocal micrograph shows an olfactory bulb slice from a postnatal day 14 mouse. Newborn interneurons were labeled by EGFP (green) and gap-mCherry (blue). The dendritic branching of interneurons was seen from the granule cell layer to the external plexiform layer. **Courtesy, with permission:** Hiroo Takahashi, Yoichi Ogawa, Sei-ichi Yoshihara, Ryo Asahina, Masahito Kinoshita, Tatsuro Kitano, Michiko Kitsuki, Kana Tatsumi, Mamiko Okuda, Kouko Tatsumi, Akio Wanaka, Hirokazu Hirai, Peter L. Stern and Akio Tsuboi, 2016, *The Journal of Neuroscience* 36(31): 8210-8227.

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This hippocampal neuron, 14 d *in vitro,* lacks NMDA receptor subunit GluN2B. It was immunostained for the AMPA receptor subunit GluA1 (green), the vesicular glutamate transporter VGLUT1 (red), and the microtubule-associated protein MAP2 (blue). An edge-detect filter was used to enhance color and cluster contour. In the absence of the GluN2B subunit, synaptic clustering of AMPA receptors is increased as a result of impaired anchoring of the synaptic proteasome. **Courtesy, with permission:** Joana S. Ferreira, Jeannette Schmidt, Pedro Rio, Rodolfo Águas, Amanda Rooyakkers, Ka Wan Li, August B. Smit, Ann Marie Craig and Ana Luisa Carvalho, 2015, *The Journal of Neuroscience* 35(22): 8462-8479.