Page 9, 85

This watercolor by Biuse Guvernau 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 build-up 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.

Page 21

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, Guangrong Huang, Guoqing Sheng, Christopher E. Henderson, Daniel Louvard, Jiho Sohn, Blake Pepinsky and Sha Mi, 2017, *The Journal of Neuroscience* 37(12): 3127-3137.

Page 22

This confocal image shows a retina prepared from a GLT-1:EGFP mouse. The widely used astrocyte marker GfAP (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 synthase (white). Cell nuclei are labeled by DAPI (blue). **Courtesy, with permission:** Wei Sun, Adam Cornwell, Jiashu Li, Sisi Peng, M. Joana Wei, Mamiko Okuda, Kouko Tatsumi, Akio Wanaka, Hirokazu Hirai, Peter L. Stern and Akio Tsuboi, 2016, *The Journal of Neuroscience* 36(31): 8210-8227.

Page 38

A thalamocortical slice from a 4-day-old 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 corticocortical 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.

Page 41

Fluorescence micrograph of a glial microtubid on isolated 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 transfected with Synaptophysin-pHLucifer, 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. Sorensen, 2014, *The Journal of Neuroscience* 34(6) 2100-2110.

Page 42

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 arbor 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 Banot, Peter Rippstein and Rashmi Kohathy, 2013, *The Journal of Neuroscience* 33(23) 9781-9793.

Page 43

Layer III pyramidal 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, 20x; numerical aperture, 0.75 [image]. **Courtesy, with permission:** Pablo Garcia-Lopez, Virginia Garcia-Marin and Miguel Freire, 2006, *The Journal of Neuroscience* 26(44): 11249-11252.

Page 75

This confocal micrograph shows an olfactory bulb slice from a postnatal day 14 mouse. Newborn interneurons were labeled by EGFP (green) and gap-gMcherry (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, Seiichi Yoshihara, Ryo Asahina, Masahito Kinoshita, Tatsuhiro Kitano, Michika Kitisu, Kana Tatsumi, Mamiko Okuda, Kaukou Tatsumi, Akio Wanaka, Hirokazu Hirai, Peter L. Stern and Akio Tsuboi, 2016, *The Journal of Neuroscience* 36(31): 8210-8227.

Page 85

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:** Joanna S. Ferreira, Jeanette Schmidt, Pedro Rio, Rodolfo Aguas, Amanda Rooyakkers, Ka Wan Li, August B. Smit, Ann Marie Craig and Ana Luisa Carvalho, 2015, *The Journal of Neuroscience* 35(22): 8462-8479.