This Week in The Journal

● Cellular/Molecular

Adenosine Enhances Sweet Responses

Robin Dando, Gennady Dvoryanchikov, Elizabeth Pereira, Nirupa Chaudhari, and Stephen D. Roper

(see pages 322–330)

Taste buds contain clusters of three types of cells that interact to process tastes. Type II cells express receptors for sweet, umami, or bitter tastes, but they do not form typical synapses: instead, receptor activation causes these cells to secrete ATP through hemichannel pores. This ATP activates afferent gustatory nerve fibers, but it also provides positive feedback to type II cells and excites type III cells, which form canonical synapses that release norepinephrine and/or serotonin. Serotonin activates gustatory afferents and inhibits type II cells. ATP-dependent signaling is terminated by conversion of ATP first to AMP—via ATPases secreted by type I cells—and then to adenosine—by ectonucleotidases produced by type III cells. Dando et al. show that adenosine also provides positive feedback to sweet-sensing type II cells, enhancing calcium transients and ATP release produced by sweet, but not umami or bitter tastes. These effects are primarily mediated by A2B adenosine receptors.

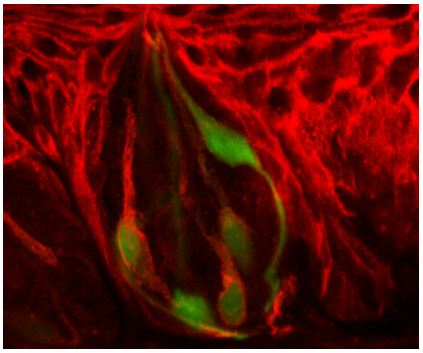
▲ Development/Plasticity/Repair

NGF-Induced Turning Requires ERM Function

Bonnie M. Marsick, Jose E. San Miguel-Ruiz, and Paul C. Letourneau

(see pages 282–296)

Neuronal growth cones explore the environment via actin-based filopodia. Diffusible guidance cues promote growth and turning by increasing filopodial protrusions, but retrograde flow of actin filaments causes protrusions to retract unless membrane receptors contact substrate-bound molecules. In the latter case, linkages form between substrate, receptors, and the actin cytoskeleton, so forces that otherwise cause retraction instead cause



Ecto-5′-nucleotidase (red), which generates adenosine from AMP, is expressed in epithelial cells and connective tissue surrounding taste buds, as well as in type III cells (green) within the taste bud. See the article by Dando et al. for details.

cytoskeletal rearrangements leading to neurite growth. Marsick et al. show that ezrin/radixin/moesin (ERM) family proteins help form such linkages in chick dorsal root ganglion (DRG) neurons. Gradients of nerve growth factor (NGF) increased filopodial protrusions, local activation of ERMs, and expression of the cell adhesion molecule L1—which binds to ERMs—along filopodia shafts. Reducing ERM function increased retrograde actin flow and reduced filopodial protrusion, axon growth, and turning. NGF-induced expression of L1 was also reduced when ERM function was blocked, and conversely, L1 knock-down reduced activation of ERMs and reduced filopodial protrusions and neurite growth.

■ Behavioral/Systems/Cognitive

Rats Show Transformation-Tolerant Visual Recognition

Sina Tafazoli, Alessandro Di Filippo, and Davide Zoccolan

(see pages 21–34)

People have a remarkable ability to recognize objects when they appear in different contexts or when they are viewed from different angles or distances. Transformation-tolerant recognition also occurs in non-human primates, but the underlying neural mechanisms are poorly understood. Hoping to extend our ability to investigate these mechanisms, Tafazoli et al. sought to determine whether rats exhibit transformation-tolerant recognition. To do so, they first trained rats to discriminate two computer-generated objects, then presented objects formed by blending the two prototypes. Flashing one of the prototypes before presenting a morphed object primed the rats to perceive the morph as similar to the flashed object. More importantly, this priming occurred even if the flashed prototype was shown at a previously unseen angle or size, indicating that the rats recognized the transformed object without training. Nonetheless, training increased the ability of transformed objects to bias responses.

♦ Neurobiology of Disease

Plant Extract Attenuates the Effects of Alcohol

Yi Shen, A. Kerstin Lindemeyer, Claudia Gonzalez, Xuesi M. Shao, Igor Spigelman, et al.

(see pages 390 – 401)

Fruit of the Japanese raisin tree, *Hovenia dulcis*, has long been used in East Asia to reduce alcohol intoxication and treat alcohol poisoning. It does this partly by speeding alcohol catabolism: extracts of the fruit increase the activity of alcohol dehydrogenase and acetaldehyde dehydrogenase. Additional effects of *H. dulcis* may make it useful in treating alcohol withdrawal. Chronic alcohol use reduces GABAergic inhibition, thus producing tolerance and causing withdrawal symptoms such as anxiety and increased seizure susceptibility. These symptoms are currently treated with benzodiazepines, which enhance GABA currents. Shen et al. found that dihydromyricetin, a flavonoid purified from *H. dulcis*, also enhanced GABA currents in cultured rat neurons. Moreover, dihydromyricetin reduced tolerance, the duration of alcohol intoxication, and withdrawal-induced anxiety-like behaviors and seizure susceptibility. A benzodiazepine antagonist reduced the protective effects of dihydromyricetin, indicating that dihydromyricetin and benzodiazepines might act via the same site on $GABA_A$ receptors.