# *This Week in The Journal*

## ● Cellular/Molecular

### *Fueling the Olfactory Glomerulus*

Janna C. Nawroth, Charles A. Greer, Wei R. Chen, Simon B. Laughlin, and Gordon M. Shepherd

(see pages 9790–9800)

In an era that promotes energy efficiency and conservation, it is only appropriate that brain parts are also evaluated for their energy consumption. This week, Nawroth et al. evaluated the energy required to run an olfactory bulb. The authors calculated the energy contribution of each cellular component. Specifically, they accounted for the thousands of axons that converge on a glomerulus as well as presynaptic and postsynaptic dendrites of mitral, tufted and periglomerular cells, and glial processes. As expected, maintaining membrane potential was the predominant resting energy cost. With activation by a single sniff of odorant, axodendritic synapses were the main energy hogs, with dendrodendritic synapses contributing a much smaller component. As a result, 2-deoxyglucose and fMRI measurements mainly reflect the activity of afferent input and axodendritic synapses. Thus, metabolic imaging provides a good view of the odorant map but an incomplete assessment of olfactory bulb function.

## ▲ Development/Plasticity/Repair

### *To Fasciculate or to Defasciculate*

Marc A. Wolman, Ann M. Regnery, Thomas Becker, Catherina G. Becker, and Mary C. Halloran

(see pages 9653–9663)

Even growing axons have choices, with one of the big ones being whether or not to fasciculate. Many signaling proteins and cell adhesion molecules contribute to this bundling process. Semaphorin3D (Sema3D) is generally considered a repulsive molecule, but Wolman et al. show this week that it can promote axon fasciculation through another mechanism. In the zebrafish medial longitudinal fascicle, Sema3D expression supported fasciculation, but not by surrounding the axons with a repulsive force. Rather, Sema3D interacted genetically with the cell adhesion molecule L1. Knockdown of Sema3D expression led to reduced axonal L1, particularly in defasciculated axons. Conversely, overexpression of Sema3D increased L1 levels without altering fasciculation. Reduction of either molecule disrupted interactions between leader and follower axons. Pathfinding was also compromised in axons with reduced Sema3D expression. In loosely fasciculated anterior commissure neurons, overexpression of Sema3D caused hyperfasciculation via this regulation of L1.

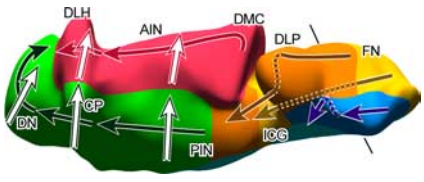
## ■ Behavioral/Systems/Cognitive

### *Mapping Projections to the Cerebellar Nuclei*

Izumi Sugihara and Yoshikazu Shinoda

(see pages 9696–9710)

The central vermis, pars intermedia, and the lateral hemispheres, along with the three deep nuclei, form the traditional divisions within the cerebellum. In addition, longitudinal cortical stripes, visualized by aldolase C labeling of Purkinje cells, specify five functional compartments according to termination patterns of climbing fibers from the inferior olive. This week, Sugihara and Shinoda looked to see if olivonuclear fibers show similar compartmentalization. The authors compared the patterns of tracer-labeled inferior olive projections with aldolase C labeling of Purkinje cell axon terminals. Labeled inferior olive fibers projected to five compartments within the cerebellar nuclei equivalent to the compartments in cerebellar cortex. Aldolase C labeled the caudoventral parts of the cerebellar nuclei, whereas the rostrodorsal parts were aldolase C negative. The organization suggests olivocortical-nuclear modules that transcend the traditional borders of the individual cerebellar nuclei.




The panel shows a three-dimensional view of the rat left cerebellar nuclei. The colors indicate the compartments created by groups of projections in the olivonuclear pathway. See the article by Sugihara et al. for details.

## ♦ Neurobiology of Disease

### *Cilia Proteins and Cerebellar Morphogenesis*

Victor V. Chizhikov, James Davenport, Qihong Zhang, Evelyn Kim Shih, Olga A. Cabello, Jannon L. Fuchs, Bradley K. Yoder, and Kathleen J. Millen

(see pages 9780–9789)

What do formation of cilia and formation of the cerebellum have in common? The answer, according to Chizhikov et al., is at least the *Kif3a* and *IFT88* genes. Previous studies have revealed that mutations in genes expressed in cilia also cause malformations of the cerebellum. The authors investigated how cilia-related proteins mostly associated with non-neuronal cells participate in cerebellar development. A mouse expressing lacZ under control of the *IFT88* promoter was expressed in Purkinje cells, granule cell progenitors, and Bergmann glia of developing and mature mouse cerebella. With conditional deletion of *IFT88* from brain, cilia were absent from the reduced population of granule cells. The mutant mice were ataxic, and cerebella were smaller with abnormal folia and Purkinje cell morphology. The phenotype resulted from disrupted granule progenitor cell proliferation in the external granular cell layer, which resembled impaired sonic hedgehog signaling. Deletion of *Kif3a*, another gene required for cilia formation, had similar deleterious effects.