

# This Week in The Journal

## ● Cellular/Molecular

### *A Two-Pore K<sup>+</sup> Channel Complex in C. elegans*

*sup-9*, *sup-10*, and *unc-93* May Encode Components of a Two-Pore K<sup>+</sup> Channel that Coordinates Muscle Contraction in *Caenorhabditis elegans*

Ignacio Perez de la Cruz, Joshua Z. Levin, Claudia Cummins, Philip Anderson, and H. Robert Horvitz (see pages 9133–9145)

Two-pore domain potassium channels comprise a large family of “leak” channels that contribute to the cell membrane conductance near the resting potential. They are modulated by pH, neurotransmitters, and other factors. Now de la Cruz et al. have identified possible regulatory protein subunits for these channels in *Caenorhabditis elegans*. Previous genetic studies in *C. elegans* found that gain of function mutants of *sup-9*, *sup-10*, and *unc-93* cause a “rubberband,” or uncoordinated (*unc*) phenotype. The authors cloned *sup-9* and *sup-10*. Sequence analysis revealed that *sup-9* encodes a member of the two-pore K<sup>+</sup> channel family, whereas SUP-10 and UNC-93 appear to associate with SUP-9, presumably as regulatory subunits. Green fluorescent protein-tagged SUP-10 and UNC-93 colocalized with SUP-9 in muscle cells. The authors speculate that the *unc* phenotype results from an increased potassium channel activity, because it was mimicked opening chloride channels in wild-type worms with muscimol. The structure of SUP-9 is similar to the mammalian TASK channels. *Sup-10* and *unc-93* are also conserved in *Drosophila* and humans, and may represent a new class of regulatory proteins that associate with and influence the activity of two-pore K<sup>+</sup> channels *in vivo*.

## ▲ Development/Plasticity/Repair

### *Sema4D and Inhibition of Axonal Regeneration*

The Transmembrane Semaphorin Sema4D/CD100, an Inhibitor of Axonal Growth, Is Expressed on Oligodendrocytes and Upregulated after CNS Lesion

Caroline Moreau-Fauvarque, Atsushi Kumanogoh, Emeline Camand, Céline Jaillard, Gilles Barbin, Isabelle Boquet, Christopher Love, E. Yvonne Jones, Hitoshi Kikutani, Catherine Lubetzki, Isabelle Dusart, and Alain Chédotal (see pages 9229–9239)

The failure of CNS axons to regenerate after injury stands in contrast to the more robust regeneration of peripheral axons. Oligodendrocytes, the central myelin-forming glial cells, are a major source of inhibitory factors that can prevent neuronal regeneration. Only a handful of these factors, the inactivation of which can enhance neurite outgrowth, have been characterized so far. The article by Moreau-Fauvarque et al. adds the homodimeric transmembrane protein Sema4D to the list of myelin-derived inhibitory factors. First identified in immune cells, the authors report that Sema4D is selectively expressed in mouse CNS white matter by myelinating oligodendrocytes. Using a



DRG explants were cultured on alternating stripes of Sema4D (asterisks) or laminin (L). As shown in *B*, the axons avoided Sema4D. See Figure 6 of Moreau-Fauvarque et al. for details.

“stripe” assay, Sema4D repelled mature DRG and granule cell axon growth. Moreover, after spinal cord lesions, the factor was transiently upregulated in oligodendrocytes surrounding the lesion site. These data provide further support for the idea that axon guidance molecules such as the semaphorins can also inhibit axonal regeneration.

## ■ Behavioral/Systems/Cognitive

### *The Musician’s Brain*

Brain Structures Differ between Musicians and Non-Musicians

Christian Gaser and Gottfried Schlaug (see pages 9240–9245)

Those of us with little or no aptitude (or, if you like, talent) for playing a musical instrument now have an excuse: our brains aren’t built for it. Such is the finding of Gaser and Schlaug in this week’s *Journal*. They found that a subject’s musical experience, as a non-musician, an amateur, or a professional, was correlated with gray matter volume in brain areas that are activated while reading and playing music, including auditory, visual–spatial, and motor areas. No wonder some people are better able to identify a tone without reference, read those funny little marks on a page and translate them, fingers flying, into a recognizable melody. But were they born that way, or is it the years of diligent practice that reshaped their cortex? The data suggest the latter: a voxel-by-voxel morphometric analysis showed that brain structural differences increased with the practice intensity and years of training of a musician. Might the musically adroit have some pre-existing disposition, however? This remains to be seen. Some might argue that the subjects, right-handed male keyboardists, are not entirely representative of professional musicians, but the study seems to make one thing clear: your brain (and your talent) won’t grow without practice.