This Week in The Journal

● Cellular/Molecular

Immature Neurons in Postnatal Rat Spinal Cord

Nicolás Marichal, Gabriela García, Milka Radmilovich, Omar Trujillo-Cenóz, and Raúl E. Russo

(see pages 10010–10024)

If a turtle's spinal cord is injured, severed axons can regrow and produce some functional recovery. Part of this regrowth might stem from neurogenic precursor cells that are present in the central canal region of the turtle spinal cord. These cells can differentiate into neurons, and they might also promote axon regeneration by bridging the lesion. Marichal et al. now report that similar cells might be present in the ependymal layer of neonatal rat spinal cord. Some cells in this region extended a single process to the central canal, expressed proteins characteristic of immature neurons, and did not express common markers of mature neurons. The morphological and electrophysiological properties of these cells suggested that they comprise neurons at various stages of maturation. Postnatal proliferation of neurogenic precursors was not detected, however, and their functional role *in vivo*, including any possible role in recovery from injury, has yet to be determined.

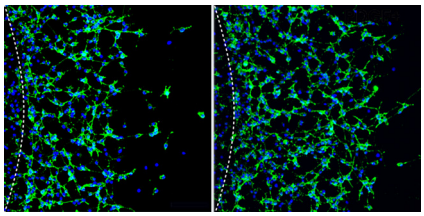
▲ Development/Plasticity/Repair

Regulation of Oligodendrocyte Precursors by Endothelin-1

Ana Gadea, Adan Aguirre, Tarik F. Haydar, and Vittorio Gallo

(see pages 10047–10062)

Oligodendrocyte progenitor cells (OPCs) are born in restricted ventricular regions of the CNS, and subsequently migrate throughout the white matter where they divide, lose their ability to proliferate and migrate, and then form myelin. This process is regulated by several molecular cues, some of which—such as platelet-derived growth factor (PDGF)—are produced by neurons and astrocytes. Gadea et al. identify the peptide endothelin-1 as another astrocyte-derived molecule that regulates OPCs. Specifically, endothelin-1 enhanced OPC migration in the presence of PDGF or fibroblast growth factor (FGF). Endothelin-1 did not, however, stimulate migration in the absence of PDGF or FGF, nor did it stimulate proliferation, as it does in astrocytes. On the other hand, endothelin-1 delayed the maturation of preoligodendrocytes to oligodendrocytes, which normally occurs after migration ceases. The effects of endothelin-1 were mediated by endothelin receptors A and B, both of which are expressed in mature oligodendrocytes as well as OPCs.



Endothelin-1 increases PDGF-stimulated migration of OPCs. Left, PDGF alone; right, PDGF + endothelin-1. See the article by Gadea et al. for details.

■ Behavioral/Systems/Cognitive

Evidence for Human Mirror Neurons

James M. Kilner, Alice Neal, Nikolaus Weiskopf, Karl J. Friston, and Chris D. Frith

(see pages 10153–10159)

Electrophysiological recordings in macaques have identified mirror neurons, which fire both when a specific action is performed and when the same action is observed. The low spatial resolution of functional magnetic resonance imaging (fMRI) makes it difficult to demonstrate the existence of analogous neurons in humans: although regions of the human inferior frontal gyrus (IFG) are active during both observation and execution, it is impossible to determine whether the same neurons are active. To circumvent this problem, scientists rely on repetition suppression, the reduction of neuronal activity by sequential stimulation. If mirror neurons are present, repetition suppression should occur when a specific action is observed then executed, but not when observation of one action is followed by execution of another. Kilner et al. suspected that failure to reveal mirror neurons in previous experiments using this paradigm resulted partly from the use of pantomimed actions. Using optimized stimuli, they now report evidence of human mirror neurons.

◆ Neurobiology of Disease

Regulation of Neuronal Excitability by a Noncoding RNA

Jun Zhong, Shih-Chieh Chuang, Riccardo Bianchi, Wangfa Zhao, Heekyung Lee, et al.

(see pages 9977–9986)

Synaptic activity can modulate neuronal excitability by altering protein synthesis at both the transcriptional and translational levels. This process must be tightly regulated to avoid producing hyperexcitability. One translational regulator is BC1, a noncoding RNA that prevents initiation of translation by interacting with the translation machinery. Zhong et al. show the importance of such translational suppression in preventing hyperexcitability by knocking out BC1 in mice. In hippocampal slices from these mice, activation of metabotropic glutamate receptors (mGluRs) caused larger increases in synthesis of synaptic proteins than in wild-type mice. In addition, whereas blocking $GABA_A$ receptors induced short synchronous bursting in wild-type pyramidal cells, these bursts gradually became prolonged in BC1 knock-outs. Moreover, EEG recordings showed increased neural activity in knock-out mice, and these mice were more susceptible to seizures induced by loud noise than wild-type mice. These three indicators of hyperexcitability were reduced or eliminated by inhibiting mGluRs, their specific downstream kinases, or protein synthesis.