This Week in The Journal

● Cellular/Molecular

Broccoli and the Blood–Brain Barrier

Jing Zhao, Anthony N. Moore, John B. Redell, and Pramod K. Dash

(see pages 10240–10248)

As if there weren't enough reasons to eat your vegetables, this week Zhao et al. report that a substance in broccoli helps to maintain the integrity of the blood–brain barrier (BBB) following a cortical contusion injury. Systemic administration of sulforaphane, contained in broccoli and other cruciferous vegetables, increased activity of NF-E2-related factor-2 (Nrf2). Nrf2 binds to the antioxidant response element (ARE), influencing expression of so-called cytoprotective proteins. Sulforaphane treatment of uninjured and brain-injured rats increased cortical expression of Nrf2-driven genes. Infusion of NR decoy oligonucleotides containing the ARE binding site for Nrf2 prevented sulforaphane-induced, Nrf2-driven gene expression. Tight junction proteins are key to maintaining BBB integrity, and they decline after brain injury. Sulforaphane attenuated the loss of these proteins as well as the loss of endothelial cells and also reduced the injury-related increase in BBB permeability and brain edema. OK, OK, pass the broccoli.

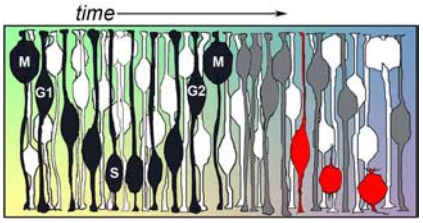
▲ Development/Plasticity/Repair

Nuclear Position and Neurogenesis in the Retina

Lisa M. Baye and Brian A. Link

(see pages 10143–10152)

Proliferating cells of the developing retina divide either symmetrically into two daughter cells that remain proliferative or neurogenically with at least one of the daughter cells exiting the cell cycle to become a postmitotic neuron. This week, Baye and Link took a close look at interkinetic nuclear migration, the apical–basal movements of progenitor cell nuclei. The authors tracked nuclear movements in zebrafish retinal neuroepithelial cells. As expected, nuclear position was apical in cells during M-phase, but varied in G_1- or S-phase. Progenitor cells varied in total cell cycle period, basal-most nuclear position, and time spent in that position. To watch nuclear movements of single cells, the authors used time-lapse confocal images of transgenic zebrafish in which GFP was expressed at cell cycle exit. The nuclei of cells that produced postmitotic neurons showed greater maximum basal migration and shorter cell cycle periods.



The schematic shows the interkinetic nuclear migration of neuroepithelial cells in the retina and the location of the cell nuclei during phases of the cell cycle. See the article by Baye and Link for details.

■ Behavioral/Systems/Cognitive

Feature Maps in the Ferret Visual Cortex

Brandon J. Farley, Hongbo Yu, Dezhe Jin, and Mriganka Sur

(see pages 10299–10310)

Neurons of the visual cortex are organized into multiple feature maps according to parameters including receptive field, ocular dominance, orientation selectivity, and spatial frequency. Each of these complex maps is spatially interrelated, but it is unclear what guides the development of these relationships. Experimental and modeling data point to a "dimension-reduction" system in which feature maps obey the principles of continuity and uniform coverage. This week, Farley et al. removed the ocular dominance map from the equation by removing sensory input from one eye of newborn ferrets. They reasoned that if the maps were interdependent, removal of the ocular dominance map would affect the shape and relatedness of the other maps. Indeed, modeling of a three-feature as opposed to a four-feature map predicted these shifts. *In vivo* optical imaging showed that enucleation eliminated the ocular dominance map but not orientation or spatial frequency maps. However, the structure and spatial relationships of the remaining maps was altered.

♦ Neurobiology of Disease

PARP Activity and Photoreceptor Death in the rd1 Mouse

François Paquet-Durand, José Silva, Tanuja Talukdar, Leif E. Johnson, Seifollah Azadi, Theo van Veen, Marius Ueffing, Stefanie M. Hauck, and Per A. R. Ekström

(see pages 10311–10319)

Retinitis pigmentosa (RP) is a general term for photoreceptor neurodegeneration that results from any of >100 genetic mutations. This week, Paquet-Durand et al. make a case for poly(ADP-ribose) polymerase (PARP) in the photoreceptor cell death. In the rd1 mouse model of RP, a loss-of-function mutation in phosphodiesterase 6 (PDE6-β) leads to cGMP accumulation and excessive calcium entry through cGMP-gated channels. PARP normally kick-starts DNA repair, but overactive PARP may promote cell death. Although PARP expression was similar in rd1 and wild-type mouse retinas, activated PARP and its by-product PAR were elevated considerably in rd1 mice by postnatal day 11. Apoptotic markers revealed a cell-death pattern that matched the central-to-peripheral retinal gradient of PARP activity. Some cells with elevated PARP activity also displayed signs of DNA damage and expression of apoptosis inducing factor. In retinal explants, a PARP inhibitor reduced PARP activity and the ensuing photoreceptor cell death.