

News Updates

Rodent Enrichment Gets Green Light

Providing laboratory mice with enriched housing does not affect the standardization of research results, according to recent work by a group of Swiss and German researchers. Mice housed in standard cages show abnormal brain development and a myriad of behavioral problems, including stereotypies, but some scientists hesitate to provide enrichment, fearing that it may confound test results. Now, in the 16 December 2004 issue of *Nature*, a research team led by Hanno Würbel of the University of Giessen in Germany suggests that this is not the case. They raised female mice in small, barren cages or large, enriched cages, which included devices such as tunnels and shredded paper. At adulthood, Würbel's group subjected the animals to four behavioral tests commonly used in drug screening and behavioral phenotyping of genetically engineered mice. Environmental enrichment was not associated with increases in variability of test results or with problems with replication of results. By debunking the most common argument against the use of environmental enrichment, this work may pave the way toward improved housing for laboratory mice.

Nursing High for Rats

Rat moms find nursing their pups more rewarding than cocaine, a finding that may lead to a better understanding of the mother-infant bond in humans. Nursing is beneficial to both mother and infant, promoting maternal behavior while providing nutrition to the neonate, but the physiological mechanisms underlying the mother's motivation to nurse are not clear. Craig F. Ferris of the University of Massachusetts Medical School (Worcester, MA) and his colleagues used magnetic resonance imaging (MRI) to measure brain activity of lactating dams exposed to their pups and virgin females that had been injected with a bolus of cocaine. Both groups showed increased activity in the same brain region—the dopamine reward system (*J. Neurosci.*, 5 January). When the researchers exposed lactating dams to cocaine, activity in this brain region dropped to below the level induced by suckling. These results suggest the evolution of a system to reward mothers that care for their young.

Stemming Hair Graying

Stem cell transplants may someday replace hair dye in the quest to conceal gray hair. A recent study by a group of Harvard researchers using melanocyte-tagged transgenic mice that show premature graying sheds some light on this mystery. The synthesis of melanin, the pigment that provides the hair's keratinocytes with color, is cytotoxic, leading some researchers to believe that the loss of differentiated melanocytes explains age-related graying of the hair. However, when David E. Fisher and his colleagues stained and analyzed mouse skin samples, they determined that graying is due to inefficient maintenance of the melanocyte stem cell population in the hair follicle (*Science*, published online 23 December 2004, doi: 10.1126/science.1099593). Analysis of human scalp tissue showed similar results. Aside from any potential cosmetic applications, understanding how melanocytes die naturally may be critical to devising ways to kill them in cases when they become melanoma, the deadliest form of skin cancer.

Arthritic Mice Reveal Molecule Involved in Disease Progression

Using a mutant mouse model, researchers have discovered a signaling molecule involved in the pathogenesis of osteoarthritis (OA). Inhibiting this molecule may be a way to treat this degenerative disease, which is the most common form of arthritis in humans. OA results from the breakdown of the cartilage that cushions joints, and causes pain and immobility. OA is often considered an inevitable result of aging, but it also has a genetic component, and mutations in type IX or XI collagens are associated with early-onset OA. A group led by Yefu Li of the Harvard School of Dental Medicine (Boston, MA) studied mice with a mutation in type XI collagen. These mice had increased levels of the protein discoidin domain receptor 2 (DDR2) in the cartilage cells of their knee joints (*J. Biol. Chem.*, 7 January). Because DDR2 stimulates the production of the cartilage-degrading enzymes, it may be possible to slow the progression of OA using inhibitors of DDR2.