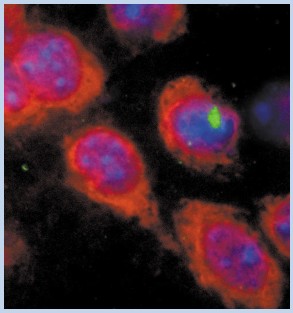
Blood into brains



Cells generated from bone marrow transplants can migrate from the circulation into the brain and differentiate into neurons, according to a report in *Science* (290, 1779–1782, 2000). Spurred on by evidence that bone marrow cells can transform into glial cells, Éva Mezey and her coworkers looked to see if blood cells could also metamorphose into neurons. The researchers injected bone marrow from adult male mice into one-day-old female pups of a strain of mouse genetically devoid of bone marrow. Cells derived from the donated marrow could then be detected using fluorescent *in situ* hybridization of the Y chromosome. After one to four months, donor-derived cells not only took up residence in the bone marrow, spleen, and liver of the mice, but also in the CNS. Furthermore, many of the cells in the brain expressed two neuron-specific markers, suggesting that differentiation had taken place. The next task, says Mezey, is to determine if the process occurs naturally in humans, and what role it might play physiologically. Some answers could come from studies of postmortem brain tissue from women who received bone marrow transplants from males. Nevertheless, Mezey's research raises the exciting possibility that an individual's own bone marrow could be used for CNS transplantation procedures, circumventing the current ethical and practical obstacles associated with embryonic stem cells. *LF*

The first complete sequence of a plant genome was published last month, bringing to an end a four-year effort by the *Arabidopsis* sequencing consortium. Last month's *Nature* (408, 816–826, 2000) provides the genome sequences of chromosomes 1, 3, and 5 of thale cress (*Arabidopsis thaliana*), completing the set of five chromosomes (two of which were published in 1999; *Nature* 402, 761–777, 1999). The complete ~119 million bp sequence contains a large amount of genetic redundancy (at least 70% of the genome is duplicated), which is thought to be due to duplication at the genome (polyploidy) or gene level. Of the 26,000 genes identified in the sequence so far, only 15,000 appear to be unique (compared with 13,601 in *Drosophila*). In contrast to their animal counterparts, *Arabidopsis* genes also appear compact with short noncoding regions (introns) closely spaced (around 4.6 kb apart). According to Virginia Walbot of Stanford University, the *Arabidopsis* sequence will be an invaluable resource for crop biotechnologists, "making much simpler" identification of crop genes involved in vegetative growth and reproduction. ***AM***

Catalytic DNA lead probe

Nanomolar concentrations of lead can be harmful to health, but current forms of detection are relatively insensitive and require sophisticated off-site analysis. Now, Jing Li and colleague Yi Lu have developed a DNA-based lead "probe" that can detect levels of the toxic metal ion between 10 nM and 4 μM using a simple fluorescence assay (*J. Am. Chem. Soc.* 122, 10466–10467, 2000). Double-stranded DNA forms a helix, but single strands can form folded structures with catalytic activity. Thousands of such catalytic DNA sequences were screened to find one (called 17E) that was sensitive to lead. When lead is present, the catalytic DNA cleaves a DNA–RNA substrate linked to a fluorophore, enhancing fluorescence. 17E was more than 80-fold more sensitive to lead than to other metal ions, and the signal was not reduced when equal quantities of other metal ions were present. Lu says that the next step is to create an array of DNA lead probes, each with differing affinities for lead, thereby improving the probe's sensitivity and reducing the risk of saturation. Practically, microfluidic technology or fiber optics could be used as platforms for the probes, offering robust and transportable detectors for this and other toxic ions. *LF*

Chlorobenzene degrader

Scientists have traditionally relied on consortia of bacteria to degrade nasty mixtures of chlorinated hydrocarbons that often are recalcitrant environmental contaminants. Now, a team of German researchers has isolated a bacterium that is capable of degrading chlorobenzenes in isolation (*Nature* 408, 580–583, 2000). The researchers obtained the strain, designated CBDB1, from a bioreactor containing a mixture of bacteria that were capable of dechlorinating chlorobenzenes. According to Lorenz Adrian, lead author on the paper, the bacterium cannot survive in the absence of the chlorobenzene, and is strictly anaerobic, requiring a hydrogen source and chlorobenzene as an electron donor. It is thought that CBDB1 forms a new bacterial cluster together with *Dehalococcoides ethenogenes*. The German team believes that isolation of the bacterium will facilitate research to define more precisely the conditions that favor rapid breakdown of the chlorinated solvent. Ultimately, this should enable more effective bioremediation of waste sites contaminated with chlorinated hydrocarbons. *AM*

Insulin mimics

A new gene therapy approach may overcome the problem of cleavage of proinsulin into active insulin for treatment of autoimmune type I diabetes. Researchers from Korea and Canada have engineered a liver-targeting recombinant adeno-associated virus (rAAV) that expresses a single-chain insulin analog (SIA), which does not require enzymatic conversion to act as biologically active insulin (*Nature* 408, 483–488, 2000). Because insulin secretion from the pancreas is induced mainly by glucose, the SIA gene was placed under the control of the glucose-responsive L-type pyruvate kinase (LPK) promoter. One week after injecting diabetic rats and mice with rAAV-LPK-SIA, blood glucose levels became normal and remained so throughout the entire eight-month study period. According to corresponding author Hyun Chul Lee, the researchers are currently working to solve lower post-digestion glucose levels of SIA "through protein engineering techniques." In another approach, genetically engineered K cells from the gut, which are naturally responsive to glucose, have been engineered to express glucose-dependent polypeptide, successfully protecting mice from developing diabetes after destruction of beta cells (*Science* 290, 1959–1962, 2000). *AJB*

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