Goodbye to fried green tomatoes?

The discovery of a gene that turns green tomatoes a ripe and rosy red could ensure that no more inedible fruits are found on supermarket shelves. Ripening in fruits does not occur through a single mechanism—tomatoes, for example, ripen by enhancing production of the gas ethylene, whereas strawberries ripen without doing this—which has confounded a broadly applicable means of control. To better understand the biochemical pathways involved in tomatoes, researchers in the plant research units of Texas (Houston, TX) and Cornell (Ithaca, NY) universities, and agbiotech company Syngenta (Bracknell, UK) studied how the *ripening-inhibitor* (*rin*) mutation kept fruit green (*Science* **296**, 343–346, 2002). The researchers found that the *rin* mutation lies between genes belonging to the MADS-box family of transcription factors, previously thought to be involved in flower development only. *Rin* mutants had reduced expression of two genes: *LeMADS-RIN*, which the team linked to ripening, upstream of ethylene production, and *LeMADS-MC*, which was linked to flower development. Interestingly, a *LeMADS-RIN*-like gene was also found in strawberries. Jim Giovannoni, lead author, says the next step is to confirm that *LeMADS-RIN* is involved in regulation of ripening "among diverse fruit-bearing species." Should this prove the case, researchers may have found a "master control switch" for fruit ripening. *LF*

The spinal trap

Following damage to the spinal cord, scar tissue forms, preventing damaged nerves from regrowing across the lesion. As a result, patients can suffer a permanent loss of movement and/or feeling. Now, researchers have shown that a bacterial enzyme, chondroitinase ABC, can clear one component of the scar tissue and partially restore function (*Nature* **416**, 636–640, 2002). Glial scar tissue contains a forest of large extracellular proteins—a key type being chondroitin sulfate proteoglycans (CSPGs)—which form a dense network of molecules that physically block the passage of nerves struggling to reconnect. Elizabeth Bradbury and colleagues at King's College London, the University of Cambridge (Cambridge, UK), and St. Bartholomew's Hospital (London) put the concept to the test, injecting the enzyme into rat spinal cord previously damaged by crushing. Chondroitinase ABC degraded the glycoprotein at the site of injury and promoted nerve regeneration. Moreover, the treated animals regained some lost movement, though no feeling was restored. The authors say that although the enzyme is no miracle cure, in combination with other therapies it could provide some hope for patients paralyzed by spinal cord injuries. *LF*

Research News Briefs written by Liz Fletcher, Christopher Martino, and Jessica Prokup.

Magnetic attraction

Miniature paramagnetic particles could create a novel matrix for DNA separation, offering a faster and less messy alternative to 2D agarose gel electrophoresis. Researchers at the Massachusetts Institute of Technology (Cambridge, MA) and the Institut Curie (Paris, France) created the matrix using a microchannel filled with a solution of paramagnetic beads and placed within a magnetic coil. When the magnetic field was turned on, the magnetic particles arranged themselves into "pillars" lying perpendicular to the force field. The researchers then added a test digest of λ DNA, showing that they could separate the fragments through the forest of columns (*Science* **295**, 2237, 2002). Video microscopy revealed how longer DNA molecules got "hooked" over the magnetic pillars, which slowed their passage down through the channels as compared with more mobile shorter fragments. The magnetic gel has several advantages over conventional agarose gels: it can separate digests faster, and can also handle DNA concatemers. Furthermore, by altering the size and concentration of the magnetic particles in solution, the "pore" size might be altered to be suitable for separating cells, proteins, or even organelles. Lead researcher Jean-Louis Viovy says that the next step will be finding a way to load the device with large DNA molecules without shearing them. *LF*

Herceptin's anticancer cocktail

Genentech's (S. San Francisco, CA) breast cancer drug herceptin—a monoclonal antibody targeted at the cell-surface receptor for human epidermal growth factor (HER-2)—may have more than one anti-cancer activity. Herceptin shrinks tumors by blocking activation of HER-2 and flagging cancer cells for elimination by the immune system. Now, Yotaro Izumi and colleagues at Massachusetts General Hospital (Boston, MA) have shown that herceptin also modulates tumor-induced growth of blood vessels—angiogenesis (*Nature* **416**, 279–280, 2002). In human breast tumors implanted in mice, herceptin shrank blood vessels by reducing the expression of the pro-angiogenic factors VEGF, TGF-α, Ang-1, and PAI-1 and increasing expression of the anti-angiogenic factor TSP-1. Herceptin proved to be less effective *in vivo* than *in vitro*, suggesting that the host cell might undergo some compensatory response to the inhibition of angiogenesis. As a single agent with dual targets, herceptin might be a simpler alternative to combination therapies. In the future, angiogenic profiles of a patient's tumor may provide clues on how to tailor treatments with agents able to modulate signal transduction. *CM*

Prostate cancer test upgrade

Current screens for prostate cancer check for elevated concentrations of prostate-specific antigen (PSA) in the blood, but this is not a fool-proof test: PSA levels rise when the prostate becomes enlarged, whether the cause is benign or sinister. Now, scientists have discovered a new biomarker that could improve the accuracy of prostate cancer tests. Using microarray technology, Mark Rubin and colleagues at the University of Michigan (Ann Arbor) loaded fluorescently labeled DNA from healthy and cancerous prostate tissue samples onto a microarray chip containing thousands of reference DNA. Through examination of over 80 microarrays, the researchers identified one gene, encoding α-methylacyl coenzyme A racemase (AMACR), that was overexpressed in cancerous prostate cells (*JAMA* **287**, 1662–1670, 2002). To confirm the clinical utility of AMACR as a biomarker for prostate cancer, Rubin *et al.* showed that AMACR protein concentrations were elevated in over 95% of tissue samples from prostate cancer patients. Screening for AMACR in the blood might provide physicians with a valuable supplement to PSA screens, reducing the number of unnecessary needle biopsies created by false positives, and highlights how gene profiling can uncover new biomarkers for disease. *JP*