In damaged blood vessels, the extracellular matrix becomes stiffer around the site of damage. Smooth muscle cells that surround blood vessels then proliferate to repair the damage. Bae et al. found that stiff surfaces stimulated cell proliferation through a signaling pathway that included the enzyme Rac. Mice that lacked Rac in their smooth muscle cells were unable to repair damaged blood vessels efficiently. — WW

**MULTIPLE SCLEROSIS**

**A surprising culprit attacks the brain**
The brain deterioration in mice with a multiple sclerosis (MS)—like disease is exacerbated by a signaling molecule called interleukin-17 (IL-17), raising hopes that MS patients could be treated with drugs that target this cytokine. In an unexpected twist, however, Noster et al. find that in humans the culprit is a different cytokine, GM-CSF, a small molecule that promotes inflammation in many autoimmune diseases. What’s more, in human patients IL-17 blocked GM-CSF production, a striking contrast to its effect in mice. These data suggest a new rationale for a therapeutic approach in MS patients: decreasing GM-CSF. — AC

**DISEASE MECHANISMS**

**An airborne agent of heart disease?**
Kawasaki disease is the most common cause of acquired heart disease in children, but even now—40 years after its discovery—doctors still don’t know its cause. Infectious and environmental agents are both possibilities. Rodó et al. compared daily Kawasaki disease case records in Japan with models of regional air trajectories. Spikes in disease incidence, they found, occurred when the wind blew from an agricultural region in northeastern China. Aerosol samples identified a high abundance of Candida, a fungus. Although the results are only a correlation, they support an existing model suggesting that genetically susceptible children may develop the disease when a windborne toxin or environmental agent triggers an aberrant immune response. — BJ
Nat. Genet. 10.1038/ng.2982 (2014)

**METABOLIC GENETICS**

**Genetic variation affects blood metabolites**
Genetic variation influences human metabolism dramatically. Understanding the process could help researchers identify drugs and develop better treatments for complex diseases. In a study of over 7000 adults, Shin et al. reconstructed metabolic

**IN OTHER JOURNALS**

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**ION TRANSPORT**
Solving the mystery of iodine uptake

The thyroid gland produces iodine-containing hormones that regulate metabolism. The cell membrane protein NIS (sodium/iodine symporter) transports iodine into thyroid cells, but because iodine concentrations outside of the cell are so low, how it does so is a mystery. The key? Moving two sodium ions along with the iodine ion, Nicola et al. found. NIS also does not bind sodium very tightly, but the high concentrations of sodium outside the cell allow one sodium ion to bind. This binding increases the affinity of NIS for a second sodium ion and also for iodine. With the three ions bound, NIS changes its conformation so that it opens to the inside of the cell, where the sodium concentration is low enough for NIS to release its sodium ions. When the sodium goes away, so does NIS’s affinity for iodine, leading NIS to release it. — VV
Nat. Commun. 10.1038/ncomms4948 (2014).