**IMMUNOLOGY**

**SREBPs for T Cell Expansion**

Responding to infections is energetically demanding, especially for cytotoxic CD8+ T cells. Once these cells recognize an infection, they blast, which requires lipid biosynthesis, and then undergo metabolic reprogramming so that they rely primarily on glycolysis to meet their high energetic demands. The specific mechanisms that allow for this transition are not well elucidated. Kidani et al. identify a role for the sterol regulatory element–binding proteins SREBP1 and SREBP2, transcription factors that regulate lipid homeostasis, in this process. The expression of SREBPs is up-regulated in response to T cell activation and is required for the induction of a lipid synthesis program. CD8+ T cells from mice with a T cell–specific deficiency in SREBPs exhibited poor blastogenesis and proliferation upon activation, altered lipid homeostasis, and did not undergo the typical activation-induced metabolic reprogramming. Impaired responses of SREBP-deficient mice to infection with lymphocytic choriomeningitis virus underscored the physiological relevance of this pathway. — KLM

_Nat. Immunol._ 14, 10.1038/ni.2570 (2013).

**GEOCHEMISTRY**

**Lost N Found**

Humans are adding new reactive forms of nitrogen (N) into the environment, which has the potential to cause a range of problems, including eutrophication and the formation of dead zones in lakes and coastal waters. The microorganisms responsible for these “N loss” pathways, known as denitrification or anaerobic ammonium oxidation (anammox), often reside in sediments, but a variable and limiting supply of organic matter makes it difficult to determine which reaction dominates. Babbin and Ward set out to address this problem in the lab by constructing a series of mesocosms out of sediments from the Chesapeake Bay, United States, with large amounts of organic matter added to some of the columns. Over 7 weeks of incubation and monitoring, the proportion of each pathway was dictated more by the relative N content of the organic matter than by the total organic matter content. Moreover, the microbial communities in the sediments were able to quickly adjust to high N loading, such as sewage effluent or fertilizer runoff, so that most of the reactive N would be removed from the ecosystem and potentially released back to the atmosphere. — NW


**CELL BIOLOGY**

**Haste Makes Waste**

mTORC1 is a protein kinase complex that regulates many biological processes, including cell growth and proliferation, and that has a primary role in the control of protein synthesis. Inhibition of mTORC1 increases life span, indicating that effects of mTORC1 on life span might be related to effects on protein synthesis. Conn and Qian present a mechanism by which reduced rates of protein synthesis might extend life span. They found essentially that haste makes waste. When mTORC1 activity was high and cells synthesized polypeptides rapidly, the stability of a newly synthesized fluorescent reporter protein was diminished. Inhibition of mTORC1, on the other hand, slowed protein synthesis through effects on translation initiation and elongation, and improved the fidelity of the process. Slower translation may allow more time for correct tRNA pairing or for cotranslational processes that promote proper folding and may thus result in fewer misincorporated amino acids or misfolded proteins, either of which would tend to reduce protein stability. — LBR

BIO MEDICINE

A Delicate Balance in Fragile X

Endocannabinoids are lipids that modulate cognition, anxiety, mood, and pain sensation—all behaviors that are deficient in patients with fragile X syndrome (FXS). FXS is a genetic disorder in which fragile X mental retardation protein (FMRP), an RNA-binding protein that controls protein synthesis, is not expressed in neurons. Treatments for the condition are focused on alleviating the symptoms. Busquets-Garcia et al. suggest that targeting the endocannabinoid system could be a new therapeutic approach for FXS. When rimonabant, a drug that blocks endocannabinoid receptor CB1R, was injected into the hippocampus of FMRP-deficient mice (a good model for FXS), the animals showed improved memory and sensitivity to pain. In pyramidal neurons, activation of the receptor mGluR5 by excitatory glutamnergic neurons triggers the mTor signaling pathway to control gene expression and synaptic plasticity. Activation of this pathway is elevated in FXS, and rimonabant normalized the phosphorylation of pathway components Akt and p70S6K in the FXS mouse model. Genetic deletion of CB1R blunted the therapeutic effects of the drug. Furthermore, an antagonist of the CB2R endocannabinoid receptor specifically normalized anxiety-like behavior in the FXS mouse model. These observations suggest that modulating endocannabinoids in FXS patients could improve cognitive abilities and anxiety, among other symptoms. — LC

Nat. Med. 19, 10.1038/nmm.3127 (2013).

PHYSICS

A Very Dilute Superconductor

Whether a given material conducts electricity, or even does so without any resistance (as in a superconductor below its transition temperature), depends sensitively on the density of electronic carriers. This density can be manipulated in several ways, the most straightforward one being chemical doping. For a semiconductor (such as Si) to start superconducting, doping with an element (such as B) at a level of a few percent is normally needed. Lin et al. found that in the bulk material SrTiO$_3$, a tiny departure from the stoichiometric composition, achieved by removing $1 \times 10^8$ oxygen atoms, is sufficient to support superconductivity. Through thermoelectric measurements, they deduced that at this lowest carrier concentration the Fermi surface is almost spherical, with only a slight anisotropy, and that the Fermi temperature, a measure of the carrier concentration, is an order of magnitude lower than the Debye temperature, which reflects the lattice dynamics energy. This unusual hierarchy of scales, as well as the character of the Fermi surface, may present challenges to theoretical models of superconductivity in this compound. — JS


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