EDITORS' CHOICE

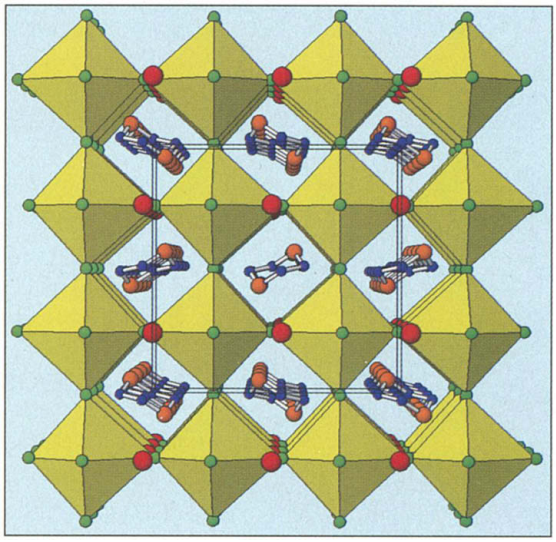
edited by Stella Hurtley

CHEMISTRY

Metal-Free Perovskites

Molecular analogs of perovskites, which includes important mantle minerals and high-temperature superconductors, have been synthesized. Bremner *et al.* crystallized cubic and 2-H hexagonal variants of a perovskite in which $(NH_4)Cl_6$ octahedra form the building blocks of a corner-sharing network. In the cubic form, the cation is the doubly hydrated piperazinium cation $C_4N_2H_{12}^{2+}$, which occupies its site along with a water molecule that helps create a stabilizing hydrogen-bonding network not seen in inorganic perovskites. The hexagonal variant, whose prototype is $BaNiO_3$, is formed by the larger doubly protonated Dabconium cation $C_6N_2H_{14}^{2+}$ and is also stabilized by hydrogen bonds. The ease of synthesis suggests that it should be possible to create optically active noncentrosymmetric as well as chiral analogs by using other templating cations. — PDS

J. Am. Chem. Soc. 10.1021/ja027484e (2002).



The cubic molecular perovskite. The orange atoms are the N atoms of the piperazinium cations, and the red atoms are the O atoms of the water molecules.

CREDIT: BREMNER *ET AL., J. AM. CHEM. SOC.* 10.1021/JA027484E (2002)

IMMUNOLOGY

Killing with Impunity

To deliver swift death to their targets, cytotoxic T lymphocytes (CTLs) and natural killer (NK) cells secrete granules containing two classes of protein that collaborate in a lethal partnership. The first, perforin, generates polymeric channels in the target cell membrane, which kill by allowing the access of the second component, pro-apoptotic granzymes. A long-standing question about this mode of cytotoxicity has been how killer cells are able to prevent their own demise via the same mechanism.

Kithiganahalli *et al.* reasoned that because granule exocytosis is polarized toward the target cell–killer cell interface, protection might be mediated by a component of the secretory granule itself, possibly a membrane-associated protease. To test this idea, CTLs were induced to deploy their cytotoxic granules in the presence of inhibitors of the membrane protease cathepsin B. In this situation, CTLs from normal mice, but not those deficient in perforin, became highly susceptible to their own killing activity. Activation of CTLs led to the cell surface accumulation of an active form of cathepsin B, capable of specifically cleaving monomeric perforin. Thus, by degrading perforin before it can polymerize in their own membranes, activated killer cells prevent granzyme reentry and avoid suicide. — SJS

J. Exp. Med. **196**, 493 (2002).

PLANT SCIENCE

Dividing Links

The chloroplasts found within plant cells are thought to derive from ancient endosymbiotic cyanobacteria. Chloroplasts contain their own genome and must replicate their DNA and divide to provide new chloroplasts in growing and dividing plant cells.

A protein named ARTEMIS has now been discovered by Fulgosi *et al.* and appears to play a key role in chloroplast division. ARTEMIS is encoded in the plant cell nucleus, but the protein is found on the chloroplast inner envelope, where it is required for organelle division. Plants engineered to lack ARTEMIS contained chloroplasts that had failed to divide completely; their inner thylakoid membranes had been duplicated and separated, but organelle fission was incomplete. A similar protein found in a cyanobacterium, *Synechocystis*, was also required for cyanobacterial division, suggesting an evolutionarily conserved mechanistic link between chloroplast division and prokaryotic cell fission. — SMH

Proc. Natl. Acad. Sci. U.S.A. **99**, 11501 (2002).

APPLIED PHYSICS

Wired for Extended Lifetime

In the usual process of patterning the metal lines in microelectronics, copper is deposited into trenches etched into the native insulating layer. In order to minimize environmental damage to the delicate wiring, the wires are then encapsulated using a dielectric layer. Despite the care taken to protect the circuitry, a significant mechanism in the failure of such wiring remains electromigration, whereby the copper atoms react with and diffuse into the dielectric layer.

Now Hu *et al.* show that coating the copper surface with a 20-nanometer layer of CoWP, CoSnP, or Pd before depositing the encapsulating dielectric layer can significantly improve the lifetime of the wiring circuitry. As the demand for smaller and smaller sized circuits continues to rise, such an effective coating should prove useful in putting the brakes on electromigration. — ISO

Appl. Phys. Lett. **81**, 1782 (2002).

EARTH SCIENCE

Any Old Iron

The Hf-W system has been used as the basis for determining when iron cores formed in Earth and Mars and when the Moon may have formed. The idea is that ^{182}Hf decays to ^{182}W with a half-life of about 9 million years. Thus, essentially no ^{182}Hf should persist 40 to 60 million years after the formation of the solar system. Chemically, tungsten but not hafnium tends to be incorporated into an iron core, so W crustal abundances are low. Thus, if iron cores formed before the time that all of the ^{182}Hf became extinct—or similarly if the Moon formed because of a giant impact with Earth—an isotopic signal should be present in W isotope data from crustal rocks. An early analysis suggested that there was no difference between the isotopic values of W for Earth and chondritic meteorites, which provide a reference value, implying that Earth's core formed late.

Three new and independent analyses by Schoenberg *et al.*, Yin *et al.*, and Kleine *et al.* instead indicate that the value for chondrites needs revision. A difference was observed between

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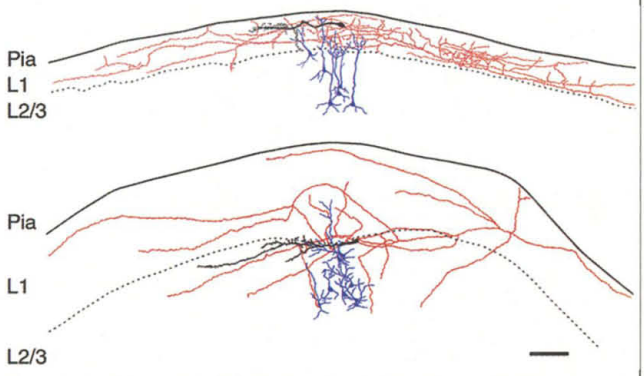
crustal values and those for carbonaceous chondrites, and crust samples from Earth and the Moon do contain evidence of the former presence of live ^{182}Hf. Together the data imply that the planets of the solar system accreted rapidly, within a few tens of millions of years, and formed their cores rapidly by internal segregation of iron. This notion is consistent with recent numerical models and other chemical and isotopic data. — BH



Camera lucida reconstructions of Cajal-Retzius cells (axons red, soma and dendrites black) and their connections with pyramidal neurons (blue) in the developing neocortex.

Geochim. Cosmochim. Acta **66**, 17 (2002); *Nature* **418**, 949; 952 (2002).

NEUROSCIENCE

Active Networks in the Early Brain

Among the earliest neurons generated in the central nervous system are the Cajal-Retzius cells. Morphological studies suggest that they play an essential role in the structural and functional organization of the neocortex.

To examine these cells in more detail, Radnikow *et al.* investigated Cajal-Retzius cells in the developing rat neocortex to establish their input-output relations and synaptic physiology. The neurons are active elements in an early neuronal network, and receive heavy GABAergic and glutamatergic synaptic input. The glutamatergic input is purely NMDA receptor–mediated. The Cajal-Retzius cells possess very long (1000- to 2000-μm) horizontal axons that form asymmetric synaptic contacts with dendritic shafts or spines of neocortical pyramidal cells. The total population of these cells provides a dense axonal network that establishes synaptic contacts over a wide range of the cortical surface. Cajal-Retzius cells thus constitute an integrative element of an early cortical network, and the concerted action of Cajal-Retzius cell activity with their partner neurons may play an important role during the early stages of synaptic circuit development. — PRS

J. Neurosci. **22**, 6908 (2002).

CREDIT: RADNIKOW *ET AL., J. NEUROSCI.* **22**, 6908 (2002)

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Autophagy and Receptor-Induced Neurodegeneration

Much attention has focused on the role of apoptosis in neurodegenerative diseases. An alternative form of death—autophagic death—may also be a critical factor. In autophagic death, organelles and cytoplasm are engulfed by autophagosomes and degraded. Lurcher mice (named for their ataxic behavior due to degeneration of the cerebellar cortex) have a mutation in the glutamate receptor subunit GluRδ2.

Yue *et al.* used a yeast two-hybrid screen to find proteins that interacted with the cytoplasmic tail of the GluRδ2 subunit and identified a protein called nPIST. The nPIST protein has properties that suggest it functions as an adaptor, possibly mediating protein-protein interactions at the receptor. A second two-hybrid screen, this time using the coiled-coil domains of nPIST, identified Beclin1, a protein previously implicated in control of membrane dynamics during autophagy. When transfected into cultured 293 cells, nPIST that lacked its PDZ domain (a likely site for interaction with other proteins) synergized with Beclin 1 to cause autophagy. Autophagy was also detected in dying Purkinje cells in the cerebellum of lurcher mice. Thus, inappropriate activation of glutamate receptors may lead to autophagic neurodegeneration through signals mediated by proteins clustered at the receptor, including nPIST and Beclin 1. Intriguingly, Beclin1 has also been shown to associate with members of the Bcl-2 family of pro-apoptotic proteins and could perhaps provide a mechanism for coordinated regulation of autophagy and apoptosis in the compromised Purkinje cells of lurcher mice. — LBR

Neuron **35**, 921 (2002).