

Self-assembly components during the reaction (above) and after cooling (below).

CHEMISTRY

Settling Down After It's All Over

Homogeneous catalysis maximizes the frequency and conformational flexibility of the collisions between catalyst and substrate, but a major shortcoming is the challenge of separating catalyst from product once the reaction is complete. Biphasic solvent systems can mitigate this problem, but often do so at the expense of reduced mixing efficiency.

Kim *et al.* demonstrate how to take advantage of molecular self-assembly in order to recover the catalyst in the dehydrogenative coupling of benzylic alcohols with olefins. The reaction is catalyzed by a phosphine-coordinated Rh complex and aminopyridine chelator (with one equivalent of olefin acting as the hydrogen acceptor). The phosphine and pyridine fragments are tethered to barbiturate derivatives that can assemble into a rigid network together with a third component—a triaminopyrimidine—by means of hydrogen bonding. When a dioxane suspension of the reagents and network-bound catalysts is heated to 150°C, the hydrogen bonds break, and a homogeneous solution forms. High yields are obtained in 2 hours, and cooling then regenerates the self-assembled network and precipitates the catalyst, allowing the product to be decanted. Catalysts were cycled eight times without significant loss in activity; moreover, switching substrates between cycles confirmed that none of the desired products partitioned into the solid phase. — JSY

Org. Lett. **8**, 10.1021/ol0608045 (2006).

APPLIED PHYSICS

Plasmons Go the Distance

The coupling of light with electronic surface excitations—specifically, surface plasmon polaritons—offers the opportunity to bridge the orders-of-magnitude difference in sizes between optical and electronic carriers. To develop schemes for coupling and transporting surface plasmons around a chip, the determination of their propagation lengths is particularly important. In this vein, van Wijngaarden *et al.* have excited surface plasmons using a focused beam of electrons and then detected the luminescence emitted as the plasmons decayed. Based on these cathodoluminescence intensity decay profiles, they could determine propagation lengths as a function of wavelength. Gold and silver thin films (on silicon and quartz substrates, respectively) were patterned with gratings to direct the emission, allowing the measurement of propagation lengths as short as several hundred nanometers. The resolution of the technique is limited by excitation volume and so should increase as film thickness decreases. The authors suggest that extensions to the characterization of more elaborate plasmonic nanostructures should also be possible. — ISO

Appl. Phys. Lett. **88**, 221111 (2006).

MOLECULAR BIOLOGY

Please Release Me

MicroRNAs (miRNAs) are small (20- to 22-nucleotide) RNAs that are encoded in the genomes of most plants and animals and that

regulate gene expression by pairing with complementary sequences in the 3' untranslated regions (UTRs) of target mRNAs. A perfect match between miRNA and target, as found in plants, generally results in cleavage and subsequent degradation of the target. An imperfect match, as often found in animals, generally results in repression of translation (of the mRNA into protein) and sequestration of the mRNA into cytoplasmic P bodies. Can such a repressed mRNA break free from its inhibitory miRNA and re-enter the pool of active mRNAs or is it doomed to stay silenced?

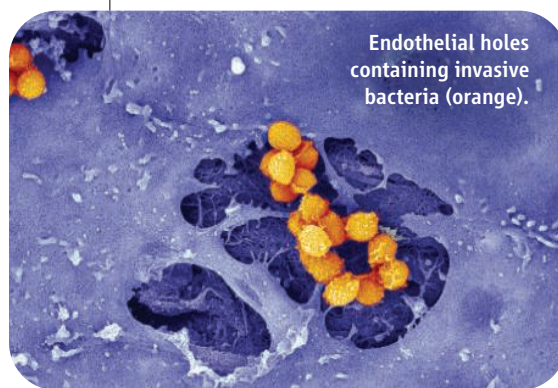
Bhattacharyya *et al.* investigate the dynamics of miRNA regulation by analyzing miR-122-directed repression of the human cationic amino acid transporter 1 (CAT-1). In Huh7 cells, CAT-1 translation is repressed by miR-122, and CAT-1 mRNA is found in P bodies. Stressing the cells by amino acid starvation results in the movement of CAT-1 mRNA from P bodies into actively translating ribosomes and in an increase of CAT-1 protein, brought about by the release of CAT-1 mRNA from the inhibitory action of miR-122. These effects are mediated by the interaction of the AU-rich element (ARE)-binding protein HuR with a segment of the CAT-1 3' UTR that is rich in A and U residues. Thus, miRNA-based down-regulation in animals is not all or none, as in plants, and can be reversed in response to changing conditions. — GR

Cell **125**, 1111 (2006).

CELL BIOLOGY

The Hole Story

The actin cytoskeleton is responsible for controlling cell shape and function. Small Rho-type GTPases regulate actin dynamics and are often the target of bacterial virulence factors that commandeer actin and use it to promote bacterial invasion strategies. Boyer *et al.* describe how *Staphylococcus aureus* exploits this cellular machinery. *S. aureus* produces a



Endothelial holes containing invasive bacteria (orange).

protein known as EDIN (epidermal cell differentiation inhibitor), which induces large, transient, transcellular holes in endothelial cell layers. These macroapertures are large enough to allow the passage of bacteria across the endothelium basement membrane. It seems that EDIN acts by inhibiting RhoA; this results in the disruption of actin cables and promotes

Continued on page 1719

Continued from page 1717

the production of actin-rich membrane waves, which open up the holes. — SMH

J. Cell Biol. **173**, 809 (2006).

IMMUNOLOGY

Another Function for AID

Activation-induced cytidine deaminase (AID) plays a pivotal role in the immune system, controlling antibody class switching and generating diversity through somatic hypermutation of immunoglobulin genes. AID is also part of a larger group of deaminases, which include the antiretroviral APOBEC family members.

Gourzi *et al.* explored the possibility that AID might possess a similar capacity for protection against retroviruses and found that cells from mice lacking AID were indeed less able to cope with a replication-deficient form of the transforming Abelson murine leukemia virus (Ab-MLV). In response to infection, AID activity was induced in the bone marrow, extending its territory beyond the B cell germinal center. Furthermore, mice succumbed to transformed B cell tumors more rapidly if they lacked AID, and showed a corresponding failure to control cellular proliferation.

AID activity induced phosphorylation of the cell cycle checkpoint kinase Chk1 and increased the sensitivity of host cells to killing by natural killer (NK) cells by up-regulating NK cell receptor ligands. These observations fit well with a model in which generalized DNA damage caused by widespread AID-induced mutations in transcribed genes prompts both checkpoint arrest and elimination by the immune system. It will now be interesting to see how broadly the scope for AID in protecting host from pathogen might extend. — SJS

Immunity **24**, 10.1016/j.immuni.2006.03.021 (2006).

BIOMEDICINE

Carbs Worth Remembering

The brains of patients with Alzheimer's disease (AD) show an aberrant buildup of oligomeric aggregates of amyloid β peptide ($A\beta$). These aggregates are neurotoxic and are believed by many researchers to be a central cause of the memory loss and cognitive decline that characterize the disease. Hence, interventions that inhibit $A\beta$ oligomerization would be expected to slow or prevent disease progression.

McLaurin *et al.* test this hypothesis in a mouse model of AD by administering cyclohexanehexols,

a group of small carbohydrate-like molecules that had been found in previous cell culture studies to stabilize $A\beta$ in a conformation that precluded its assembly into oligomers. The treated mice showed improved cognitive function and reduced neuropathology, and they lived longer than control mice. The cyclohexanehexols were effective not only in a prevention setting but even when given to mice after the onset of symptoms. These results underscore the pathogenic role of $A\beta$ oligomerization in AD and raise the possibility that derivatives of these compounds, which cross the blood/brain barrier and can be taken orally, may offer therapeutic benefit to patients with the disease. — PAK

Nat. Med. **12**, 10.1038/nm1423 (2006).

NANOTECHNOLOGY

All Wound Up

Nanohelices can be used in micro- and nanoelectromechanical systems as resonators, mechanical components, or sensors. One route for the controlled fabrication of nanohelices is to grow strained heterofilms on a substrate and to etch and release

the films, which then form coiled structures attached to the substrate at one end.

Previously, Zhang *et al.* developed such a method for SiGe and SiGe/Cr films on single-crystalline Si(100) substrates that was limited to helical angles of 45° or more (the maximum orientation mismatch). They now report that as the width of the stripes

is decreased below 1 μm , edge effects lead to tighter pitches and cause the handedness of the helices to reverse (from right to left, through a disordered transition regime); even concentric multiwall rings can be fabricated. Although the Cr layers are isotropic, they change the edge stresses and cause the onset of anomalous coiling (deviation from the preferred $\langle 100 \rangle$ scrolling direction) to occur at larger stripe widths. The authors map out the conditions for controlling helical angles to less than 10° and model the relaxation behavior of these films with finite-element simulations. — PDS

Nano Lett. **6**, 10.1021/nl053240u (2006).

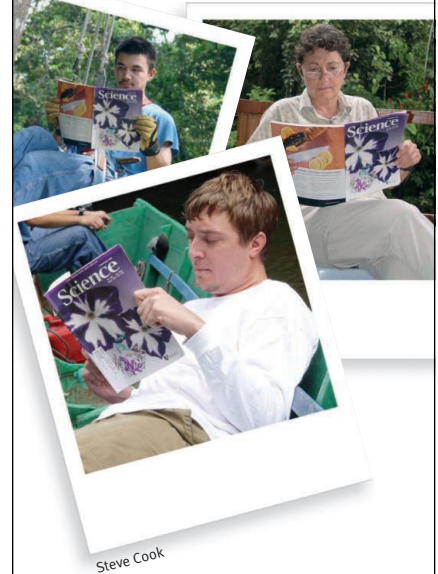


Scanning electron micrograph of Si/Cr bilayers; the similarity of coiling illustrates the dominant effect of the Cr layer over substrate direction.

Q Who's delivering science to every corner of the world?

Chris Bernau

Dr. Dinah Davidson



Steve Cook

“ Sharing one copy of *Science* around our research camp in Brunei requires a plan as systematic as the ants we're studying. On the boat, in a treetop, or on the deck after dinner, we all get our chance to catch up on what's new in science. ”

AAAS members Chris Bernau, Dr. Dinah Davidson, and Steve Cook

AAAS is committed to advancing science and giving a voice to scientists around the world. Helping our members stay abreast of their field is a key priority.

One way we do this is through *Science*, which features all the latest groundbreaking research, and keeps scientists connected wherever they happen to be.

To join the international family of science, go to www.aaas.org/join.



ADVANCING SCIENCE. SERVING SOCIETY

www.aaas.org/join