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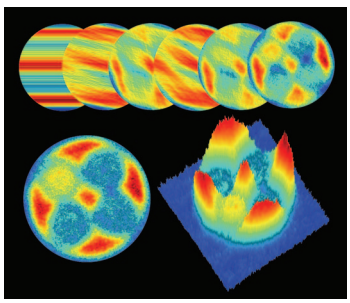
APPLIED PHYSICAL SCIENCES

NMR technique to expand MRI uses

MRI allows clinicians to noninvasively visualize the inside of the human body. Because the radio frequency (rf) pulses of MRI machines deposit heat in patients and medical staff, safety regulations that limit energy capabilities of such machines have long been established. Norbert Müller and Alexej Jerschow have devised a low-energy NMR technique that does not expose samples to rf irradiation and could help develop light, mobile, and safer MRI equipment. The authors' technique, which does not require external rf irradiation, relies on the detection of spontaneous proton spin noise in a tightly coupled rf cavity. To reconstruct spin noise images, the researchers used a commercial, liquid-state NMR spectrometer equipped with a cryogenically cooled probe to image a sample (glass capillaries filled with mixtures of water and heavy water). The sample was inserted into a standard NMR tube, and a magnetic field gradient was applied to acquire an image. Thirty one-dimensional images were collected, and, after applying a projection reconstruction algorithm, a two-dimensional image was obtained. Because of its low energy requirements, the imaging technique may enable new application areas for MRI microscopy. — F.A.



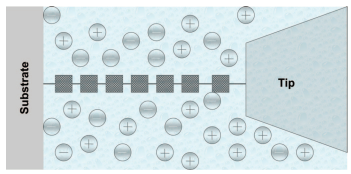
Low-energy NMR-based images.

"Nuclear spin noise imaging" by Norbert Müller and Alexej Jerschow (see pages 6790–6792)

CHEMISTRY

Electron transfer across nanojunctions

Bridge-assisted electron transfer processes are used in molecular electronics, such as scanning tunneling microscopes. Alexei Kornyshev *et al.* report a system that can provide current–voltage rectification (conversion from alternating to direct current) in single-molecule, bridge-mediated electronic nanojunctions, which can potentially be utilized in electronic devices. Kornyshev *et al.* employed a mathematical model to analyze the electronic conductivity of molecules in aqueous electrolyte solutions and confined in a gap between two metallic electrodes. The model revealed strong rectification in the presence of electrolyte for the intrinsically symmetric system. Higher electrolyte concentrations led to a shortening of the Debye length, the distance over which significant charge separation can occur, and to stronger rectification. Increases in the length of the bridge molecule amplified this effect. These findings may allow new experimental studies of bridge-mediated nanojunctions and new designs of single-molecule-based electronic devices. — F.A.



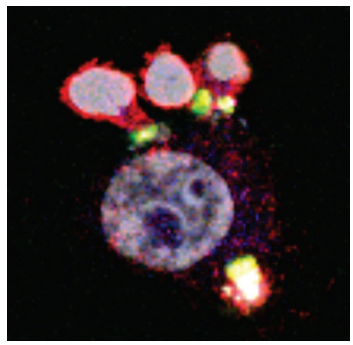
In situ bridge-mediated nanogap.

*"*In situ *superexchange electron transfer through a single molecule: A rectifying effect" by Alexei A. Kornyshev, Alexander M. Kuznetsov, and Jens Ulstrup* (see pages 6799–6804)

MICROBIOLOGY

Resting B cells transfer Epstein–Barr virus

Epstein–Barr virus (EBV) is an orally transmitted herpesvirus widespread among humans that has been associated with various cancer types. Claire Shannon-Lowe *et al.* report that EBV can infect epithelial cells while bound to the surface of resting B cells. EBV preferentially infects B lymphocytes via the interaction of the viral glycoprotein gp350 and the B cell receptor CD21. Shannon-Lowe *et al.* demonstrated a mechanism wherein the surface of the B cell acts as a transfer vehicle for infection. The authors showed that B cells and epithelial cells can form conjugates with gp350/CD21 com-



Conjugates between epithelial and EBV-infected B cells.

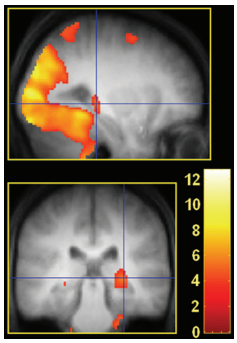
plexes, thereby promoting efficient EBV transfer. Transfer involves the viral glycoproteins gp85 and gp110, whereas gp42, which is essential for B cell infection, is not required. Surprisingly, a gp350 knockout virus, which cannot bind to B cells, had the capacity to infect epithelia directly. This finding suggests that gp350 is inhibitory to direct epithelial cell infection and that this inhibition is relieved upon gp350–CD21 interaction, thereby allowing simultaneous infection of two cell types. Thus, EBV appears to be able to concurrently access both lymphoid and epithelial compartments, shedding further light on the infective mechanisms of this cancer-associated virus. — N.Z.

"Resting B cells as a transfer vehicle for Epstein–Barr virus infection of epithelial cells" by C. D. Shannon-Lowe, B. Neuhierl, G. Baldwin, A. B. Rickinson, and H.-J. Delecluse (see pages 7065–7070)

NEUROSCIENCE

Sleep triggers relocation of spatial memories

A good night of sleep not only promotes the hard-wiring of spatial memories into the neural circuitry of the brain but also triggers the reorganization of where these memories are wired for long-term storage. Using a popular video game, Pierre Orban *et al.* trained two groups of human volunteers on place-finding missions in a virtual town and used functional MRI to map brain activity immediately after the task and again 3 days later. The first group of volunteers had regular sleep (RS) the first night after the training session, whereas the second group was totally sleep deprived (TSD). Both groups were then allowed two nights of regular sleep before the delayed retrieval test on the third day. The authors found that, during both immediate and delayed retrieval of navigation memories, the hippocampus was particularly active in both the RS and TSD subjects. In addition, the RS group also displayed more activity in the striatum during the delayed retrieval. Performance on the navigation tests was essentially equivalent for both groups, suggesting that sleep deprivation did not necessarily lead to poor memory acquisition. Rather, it hindered the relocation of these memories to another region of the brain, the striatum. — B.T.



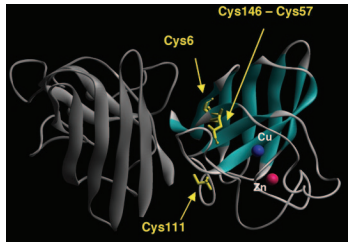
Navigation accuracy and place-finding-related brain network.

"Sleep after spatial learning promotes covert reorganization of brain activity" by Pierre Orban, Géraldine Rauchs, Evelyne Balteau, Christian Degueldre, André Luxen, Pierre Maquet, and Philippe Peigneux (see pages 7124–7129)

NEUROSCIENCE

Protein aggregation mechanisms for ALS

Mutations in the Cu, Zn-superoxide dismutase (SOD1) gene are responsible for ≈20% of familial amyotrophic lateral sclerosis (fALS) cases, yet with >100 known SOD1 mutations associated with fALS, the molecular mechanisms behind this disease remain unresolved. A pair of related studies provides evidence that links disease onset with the formation of intermolecular aggregates. First, Han-Xiang Deng *et al.* observed that overexpression of wild-type human SOD1 in transgenic mice carrying SOD1 mutants hastened disease progression, even converting one mutant that normally remains unaffected ($SOD1^{A4V}$) into a disease phenotype. The authors found that the disease-causing mutant proteins recruited wild-type SOD1 into insoluble aggregates that contained cross-linked multimers in the mitochondria of the affected mice. The high stability of most of the mature SOD1 mutants and the low solubility of the aggregates complicated this mechanism, but in a second study, Yoshiaki Furukawa *et al.* showed that the immature, disulfide-reduced forms of the SOD1 protein play a critical role. Intermolecular disulfide bonds were found to stabilize aggregated multimers of SOD1. Interestingly, a truncated SOD1 mutant lacking the final cysteine only formed dimers, indicating that higher-order aggregation is not a random process. The authors found that mild oxidation of purified mutant SOD1 protein could mimic this aggregation effect, and removal of the cysteines prevented aggregation. Taken together, these results suggest that oxidative stress can cause misfolded, immature SOD1 oligomers to stabilize via incorrect disulfide cross-links, leading to a toxic cascade of protein aggregation in fALS. — N.Z.



Crystal structure of human SOD1.

"Conversion to the amyotrophic lateral sclerosis phenotype is associated with intermolecular linked insoluble aggregates of SOD1 in mitochondria" by Han-Xiang Deng, Yong Shi, Yoshiaki Furukawa, Hong Zhai, Ronggen Fu, Erdong Liu, George H. Gorrie, Mohammad S. Khan, Wu-Yen Hung, Eileen H. Bigio, Thomas Lukas, Mauro C. Dal Canto, Thomas V. O'Halloran, and Teepu Siddique (see pages 7142–7147)

and

"Disulfide cross-linked protein represents a significant fraction of ALS-associated Cu, Zn-superoxide dismutase aggregates in spinal cords of model mice" by Yoshiaki Furukawa, Ronggen Fu, Han-Xiang Deng, Teepu Siddique, and Thomas V. O'Halloran (see pages 7148–7153)