PNAS Plus Significance Statements

Modeling integrated photovoltaic–electrochemical devices using steady-state equivalent circuits

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This article (pp. E1076–E1082) extends the construction of direct solar-to-fuels devices, such as the artificial leaf based on crystalline silicon. Because a single Si junction has insufficient potential to drive water splitting, it cannot be used for direct solar-to-fuels conversion. This paper performs an equivalent circuit analysis for multiple series-connected devices. The predictive utility of the model is demonstrated in the case of water oxidation at the surface of a Si solar cell, using a cobalt–borate oxygen evolving catalyst. Considering recent cost reductions of Si solar cells, this paper offers a path to the construction of low cost solar-to-fuels devices.

Structural model for the protein-translocating element of the twin-arginine transport system

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The twin-arginine transport system (Tat) has the remarkable ability of transporting folded proteins across membranes while avoiding uncontrolled ion leakage. Tat is essential for plant photosynthesis and is required for bacterial pathogenesis. The mechanism by which folded proteins are translocated is poorly understood. We have determined the structure of the TatA oligomer, which is responsible for the translocation step, and evaluated its impact on lipid bilayers. The results (pp. E1092–E1101) suggest a mechanism of protein translocation involving thinning and perturbing the membrane bilayer. The approach used here will be useful for structural analysis of other oligomeric proteins that weakly assemble in the membrane.

Arf tumor suppressor and miR-205 regulate cell adhesion and formation of extraembryonic endoderm from pluripotent stem cells

Chunliang Li, David Finkelstein, and Charles J. Sherr

The *Arf* tumor suppressor gene is not expressed in most normal tissues but when activated by oncogenic stress signals engages a p53-dependent transcriptional program that prevents tumor formation. Surprisingly, expression of the $p19^{Arf}$ protein in mouse embryoid bodies is required for the timely formation of extraembryonic endoderm (ExEn). Inactivation of *Arf* down-regulates a single microRNA, miR-205, which can "rescue" ExEn formation in *Arf*-null embryonic or induced pluripotent stem cells. During ExEn formation, miR-205 regulates a suite of genes that govern cell migration and adhesion, suggesting a conceptual basis for linking the roles of *Arf* in ExEn differentiation and tumor metastasis. (See pp. E1112–E1121)

Host DNA released in response to aluminum adjuvant enhances MHC class II-mediated antigen presentation and prolongs CD4 T-cell interactions with dendritic cells

Amy S. McKee, Matthew A. Burchill, Michael W. Munks, Lei Jin, John W. Kappler, Rachel S. Friedman, Jordan Jacobelli, and Philippa Marrack

Alum has been used to improve the efficacy of vaccines since the 1930s. Here we show (pp. E1122–E1131) that alum acts in part via host DNA to increase the interaction time between T cells and APCs.

Effects of insufficient sleep on circadian rhythmicity and expression amplitude of the human blood transcriptome

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Insufficient sleep and circadian rhythm disruption are associated with negative health outcomes, but the mechanisms involved remain largely unexplored. We show (pp. E1132–E1141) that one wk of insufficient sleep alters gene expression in human blood cells, reduces the amplitude of circadian rhythms in gene expression, and intensifies the effects of subsequent acute total sleep loss on gene expression. The affected genes are involved in chromatin remodeling, regulation of gene expression, and immune and stress responses. The data imply molecular mechanisms mediating the effects of sleep loss on health and highlight the interrelationships between sleep homeostasis, circadian rhythmicity, and metabolism.

Electroencephalogram signatures of loss and recovery of consciousness from propofol

Patrick L. Purdon, Eric T. Pierce, Eran A. Mukamel, Michael J. Prerau, John L. Walsh, Kin Foon K. Wong, Andres F. Salazar-Gomez, Priscilla G. Harrell, Aaron L. Sampson, Aylin Cimenser, ShiNung Ching, Nancy J. Kopell, Casie Tavares-Stoeckel, Kathleen Habeeb, Rebecca Merhar, and Emery N. Brown

Anesthesiologists reversibly manipulate the brain function of nearly 60,000 patients each day, but brain-state monitoring is not an accepted practice in anesthesia care because markers that reliably track changes in level of consciousness under general anesthesia have yet to be identified. We found (pp. E1142–E1151) specific behavioral and electrophysiological changes that mark the transition between consciousness and unconsciousness induced by propofol, one of the most commonly used anesthetic drugs. Our results provide insights into the mechanisms of propofol-induced unconsciousness and establish EEG signatures of this brain state that could be used to monitor the brain activity of patients receiving general anesthesia.

Retrograde monosynaptic tracing reveals the temporal evolution of inputs onto new neurons in the adult dentate gyrus and olfactory bulb

Aditi Deshpande, Matteo Bergami, Alexander Ghanem, Karl-Klaus Conzelmann, Alexandra Lepier, Magdalena Götz, and Benedikt Berninger

New neurons are constantly added to the hippocampus and the olfactory bulb. These neurons are believed to fulfill unique functions during their early life compared with mature neurons, which may depend on the way they are connected. Here we studied the stepwise integration of new neurons within these two brain areas using a rabies-virus–based synaptic tracing tool. Our study (pp. E1152–E1161) revealed that in both areas integration follows a similar logic, with adult-born neurons incorporating first into the local circuit before becoming innervated by long-range connections. This changing pattern of presynaptic connectivity likely contributes to adult-born neurons' functions.

Xerocytosis is caused by mutations that alter the kinetics of the mechanosensitive channel PIEZO1

Chilman Bae, Radhakrishnan Gnanasambandam, Chris Nicolai, Frederick Sachs, and Philip A. Gottlieb

Familial xerocytosis in humans, which causes dehydration of red blood cells and hemolytic anemia, was traced to mutations in the mechanosensitive ion channel, PIEZO1. The mutations slowed inactivation and introduced a pronounced latency for activation. Loss of inactivation and increased latency for activation could modify groups of channels simultaneously, suggesting that they exist in common spatial domains. The hereditary xerocytosis mutants affect red cell cation fluxes: slow inactivation increases them, and increased latency decreases them. These data (pp. E1162–E1168) provide a direct link between pathology and mechanosensitive channel dysfunction in nonsensory cells.

An RNA recognition motif-containing protein is required for plastid RNA editing in *Arabidopsis* and maize

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Transcripts in plant organelles are altered by conversion of cytidines to uridines in a process termed RNA editing. Members of two protein families have been identified in the plant editosome, but its complete composition is unknown. Now a unique protein that contains an RNA recognition motif has been found to be essential for editing of multiple plastid transcripts in both *Arabidopsis* and maize. Phylogenetic analysis (pp. E1169–E1178) indicates that this protein belongs to a sub-family of RNA recognition-motif proteins predominantly predicted to be targeted to organelles and that are thus likely to play roles in organelle RNA metabolism.