Science AAAS

Paradigm Shifts in Dye-Sensitized Solar Cells

Michael D. McGehee

Science **334**, 607 (2011);
DOI: 10.1126/science.1212818

This copy is for your personal, non-commercial use only.

If you wish to distribute this article to others, you can order high-quality copies for your colleagues, clients, or customers by clicking here.

Permission to republish or repurpose articles or portions of articles can be obtained by following the guidelines here.

The following resources related to this article are available online at www.sciencemag.org (this infomation is current as of January 11, 2012):

Updated information and services, including high-resolution figures, can be found in the online version of this article at:
http://www.sciencemag.org/content/334/6056/607.full.html

A list of selected additional articles on the Science Web sites **related to this article** can be found at:
http://www.sciencemag.org/content/334/6056/607.full.html#related

This article **cites 9 articles**, 1 of which can be accessed free:
http://www.sciencemag.org/content/334/6056/607.full.html#ref-list-1

This article appears in the following **subject collections:**
Physics, Applied
http://www.sciencemag.org/cgi/collection/app_physics

Downloaded from www.sciencemag.org on January 11, 2012

Science (print ISSN 0036-8075; online ISSN 1095-9203) is published weekly, except the last week in December, by the American Association for the Advancement of Science, 1200 New York Avenue NW, Washington, DC 20005. Copyright 2011 by the American Association for the Advancement of Science; all rights reserved. The title *Science* is a registered trademark of AAAS.

repressor." Thus, in the SCA1 mouse model, both a Cic loss of function and gain of function appear to occur simultaneously, albeit at distinct transcriptional targets. Because the partial loss of Cic function cannot explain the disease rescue that Fryer *et al*. observed, they propose that polyQ-expanded ataxin-1 also confers a gain-of-function effect on Cic, and the relief of this gain of function is enough to rescue the disease phenotypes even in the face of further loss of Cic function. At the molecular level, it appears that polyQ-expanded ataxin-1 causes Cic to bind more tightly to the promoters of some genes (gain of function), while it concomitantly causes Cic to bind less well to the promoters of other genes (loss of function). How ataxin-1 converts Cic at certain loci to a hyper-repressor while inhibiting the function of Cic at other loci remains to be determined. Nonetheless, these findings underscore the complexity of protein interactions in mediating neurodegenerative disease.

The study by Fryer *et al*. suggests the exciting possibility that exercise, perhaps early in disease, or even before disease onset (this could be determined in SCA1 and other trinucleotide repeat diseases by genetic testing for risk alleles), could help delay the progression. And if the effects of exercise are attributed to a decrease in Cic, then lowering the amount of Cic might be an effective therapeutic approach. Given the encouraging results with exercise and SCA1, it will be of immediate interest to test the effects of exercise on other neurodegenerative diseases, for which effective treatments have been elusive and are desperately needed.

References

1. J. D. Fryer *et al*., *Science* **334**, 690 (2011).
2. M. S. Forman, J. Q. Trojanowski, V. M. Lee, *Nat. Med.* **10**, 1055 (2004).
3. H. T. Orr *et al*., *Nat. Genet.* **4**, 221 (1993).
4. J. R. Gatchel, H. Y. Zoghbi, *Nat. Rev. Genet.* **6**, 743 (2005).
5. H. Y. Zoghbi, H. T. Orr, *J. Biol. Chem.* **284**, 7425 (2009).
6. P. Fernandez-Funez *et al*., *Nature* **408**, 101 (2000).
7. K. Watase *et al*., *Neuron* **34**, 905 (2002).
8. Y. C. Lam *et al*., *Cell* **127**, 1335 (2006).
9. J. Lim *et al*., *Nature* **452**, 713 (2008).
10. L. Duvick *et al*., *Neuron* **67**, 929 (2010).
11. K. Watase *et al*., *PLoS Med.* **4**, e182 (2007).
12. C. W. Cotman, N. C. Berchtold, L. A. Christie, *Trends Neurosci.* **30**, 464 (2007).
13. Y. Rolland, G. Abellan van Kan, B. Vellas, *J. Am. Med. Dir. Assoc.* **9**, 390 (2008).
14. L. E. Dibble, O. Addison, E. Papa, *J. Neurol. Phys. Ther.* **33**, 14 (2009).
15. A. Chiò, G. Benzi, M. Dossena, R. Mutani, G. Mora, *Brain* **128**, 472 (2005).

10.1126/science.1214714

APPLIED PHYSICS

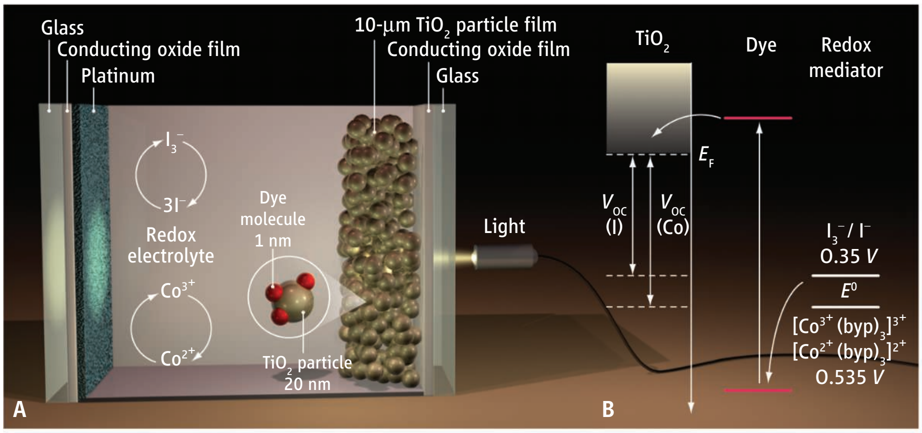
Paradigm Shifts in Dye-Sensitized Solar Cells

Efficiency limitations from the iodine-based electron shuttle have been overcome by replacing it with cobalt compounds and by using dyes that block recombination.

Michael D. McGehee

Dye-sensitized solar cells (DSCs) have captured the imagination of a wide range of people, from middle-school students who make their first solar cell by sensitizing films of titania nanocrystals with berry juice to scientists and engineers who are striving to solve the world's energy problem. A schematic of a DSC is shown in the figure, panel A (*1*, *2*). Until recently, practical DSCs—ones with efficiencies exceeding 11%—have had to rely on dyes that are expensive in that they contain the noble metal ruthenium (Ru). Efforts to increase cell efficiency by boosting the output voltage of the cell, and to decrease costs by eliminating the use of Ru, have run afoul of the enemy of all solar cells—the recombination of charge carriers before they are delivered to the electrodes. On page 629 of this issue, Yella *et al*. (*3*) report on a specially designed redox mediator containing cobalt (Co) complexes that enables a DSC efficiency of 12.3% under 1 sun of illumination.

To understand how the redox mediator improves DSC efficiency, it is useful to review how these electrochemical cells work. Titania nanocrystals—white paint pigments that reflect most of the incident solar energy—are bonded into a film on a transparent electrode. To absorb sunlight, titania is dyed with a light-absorbing material, the dye sensitizer (e.g., a Ru compound bearing bipyridyl ligands that help absorb light). The cell is completed with an electrolyte solution with a redox mediator, typically iodide ions, and a counterelectrode. Light absorption by a dye molecule provides the energy to inject one of its electrons into the titania, which provides a pathway to the external circuit for energy extraction. The mediator donates an electron to the now electron-deficient sensitizing dye. The now-oxidized mediator diffuses across the cell to the counterelectrode to be reunited with an electron passed through the external circuit.



Shuttle differences. (**A**) Schematic of a DSC solar cell. In a conventional cell, the dye is a Ru complex, and the redox shuttle is based on iodine ions. In the work of Yella *et al*., the dye contains an electron donor and acceptor connected by a conjugated bridging group, and the redox shuttle is based on a cobalt compound. (**B**) The open-circuit voltage of the cell, V_{OC}, is the difference between the Fermi level E_F of the titania and the redox potential E^0 of the redox shuttle. The cobalt-based shuttle boosts V_{OC} by 185 mV versus iodine ions. TiO_2, titanium dioxide; byp, bipyridyl.

CREDIT: P. HUEY/SCIENCE

Department of Materials Science, Stanford University, Stanford, CA 94305, USA. E-mail: mmcgehee@stanford.edu

Published by AAAS

Downloaded from www.sciencemag.org on January 11, 2012

DSCs are extremely good at separating photogenerated charge carriers and taking them to the electrodes. If an incoming photon is absorbed by the dye in a state-of-the-art DSC, the probability of the charge carriers reaching the electrodes is nearly 100%. Until recently, the world record for power conversion was 11.1% (*4*). Although the sensitizing molecules in these cells have a relatively large energy gap, the cells do not generate as high a voltage as one could reasonably expect because the redox potential of the most commonly used redox couple, which is based on iodide ions, is too low for the best sensitizing dyes (see the figure, panel B). Energy is wasted as the redox couple reduces a charged-sensitizing dye back to its neutral state.

Many researchers have been trying for more than a decade to find a redox couple with a more ideally suited redox potential in order to increase the voltage of DSCs, but the process has been slow and frustrating. The iodide mediator is special as its oxidized form, I_3^-, does not readily accept electrons from the titania surface, which would be an unwanted recombination process (*5*). The I_3^- ions can persist for 1 ms or more before undergoing an electron-transfer reaction in solution, which gives them sufficient time to diffuse to the counterelectrode for reduction. Several alternative metal complexes with better redox potentials have been found, but solar cells made with them typically have unacceptably high recombination rates and lower open-circuit voltages.

Last year, Boschloo, Hagfeldt, and coworkers achieved a power-conversion efficiency of 6.7% in DSCs with iodide-free electrolytes using Co complexes (*6*). The key to making good cells was adding just enough electrically inactive bulk to the periphery of the sensitizing dyes and the Co complexes to slow down recombination without blocking the necessary electron-transfer processes.

The approach Yella *et al*. took to slowing down recombination was to use a relatively new family of dyes that connect an electron donor (D) moiety to an electron acceptor moiety (A) through a conjugated (π−bonded) bridge (D−π−A) provided by a zinc porphyrin complex. These D−π−A dyes do not contain any expensive, rare metal atoms, such as Ru, and tend to absorb light more strongly. Most important, they attached alkoxy chains to the sides of these molecules to provide a very effective barrier to recombination between electrons in the titania and holes in the Co complexes.

One of the shortcomings of the Co complexes is that they diffuse through the electrolyte more slowly than the conventional iodide ions because they are larger. Yella *et al*. found that they could get efficiency as high as 13.1% by reducing the illumination intensity by 50% because it is less important for the ions to diffuse to the electrode quickly when the carrier density is lower. This efficiency might be obtained under normal solar lighting by reducing the distance the ions need to diffuse through the use of thinner films. Complete absorption in these thinner films could be achieved by using even better D−π−A dyes that absorb more strongly or advanced light-trapping techniques such as plasmonics (*7*, *8*). Long-term stability studies must be performed on Co complexes in dye-sensitized solar cells to determine if they are as stable as iodide-based electrolytes.

For many years, dozens of researchers around the world tried to develop new sensitizing dyes and redox couples for DSCs but inevitably concluded that the best sensitizing dyes needed to contain Ru and the best mediators needed to contain iodide. These paradigms are now shattered. One of the next developments that needs to occur is reducing the energy gap of sensitizing dyes so that light can be harvested efficiently further into the infrared region of the spectrum. One approach would be to use sensitizing dyes to absorb infrared light and energy relay dyes that absorb visible light, and then transfer energy to the sensitizing dyes (*9*). As scientists around the world develop new D−π−A dyes and redox couples and combine them with energy relay dyes and new light-trapping techniques, we can expect to see the efficiency climb toward 15%, which could make DSC technology competitive with other kinds of solar cells.

References

1. B. O'Regan, M. Gratzel, *Nature* **353**, 737 (1991).
2. A. Hagfeldt, G. Boschloo, L. Sun, L. Kloo, H. Pettersson, *Chem. Rev.* **110**, 6595 (2010).
3. A. Yella *et al.*, *Science* **334**, 629 (2011).
4. Y. Chiba *et al.*, *Jpn. J. Appl. Phys.* **45**, L638 (2006).
5. G. Boschloo, A. Hagfeldt, *Acc. Chem. Res.* **42**, 1819 (2009).
6. S. M. Feldt *et al.*, *J. Am. Chem. Soc.* **132**, 16714 (2010).
7. H. A. Atwater, A. Polman, *Nat. Mater.* **9**, 205 (2010).
8. I.-K. Ding *et al.*, *Adv. Energy Mater.* **1**, 52 (2011).
9. B. E. Hardin *et al.*, *Nat. Photonics* **3**, 406 (2009).

10.1126/science.1212818

Downloaded from www.sciencemag.org on January 11, 2012

NEUROSCIENCE

Synaptic Switch and Social Status

Matthieu Maroteaux and Manuel Mameli

The strength of synaptic connections in the mammalian brain can influence social status.

In 1859, Charles Darwin introduced the key concept of natural selection—that in the struggle for survival and reproduction, individuals compete for the same resources. Hence, animals living in a social environment can establish dominance hierarchies within a short time, which remain stable during the existence of the group (*1*). This ranking within social communities has a fundamental advantage—it eliminates conflict in the group, which minimizes energy expenditure and violence, thereby allowing resource sharing (*2*). On page 693 of this issue, Wang *et al*. (*3*) demonstrate that encoding of social dominance in mice involves specific synapses in cortical regions of the brain.

Wang *et al*. used a behavioral experimental model (*4*) that provides a quantitative measure of aggressiveness without allowing physical contact between competing mice, thereby preventing injuries. In each trial, one mouse forces its opponent outside of a neutral area, permitting identification of a dominant and a subordinate mouse. The authors show that in a cohort of four mice, a social hierarchy was quickly organized and once established, persisted over time. Another important aspect is the complete independence of social dominance from other factors, including sensorimotor and learning skills. This makes hierarchy formation a unique and distinct trait of animal behavior.

When and where does nature decide that an individual will dominate in a community? Early in life, mice as well as humans are embedded in a community that in the simplest scenario consists of siblings. An interesting possibility is that innate traits and genetic programming that shape neuronal circuits from the postnatal period are responsible for the allocation of dominance or subor-

INSERM UMR-S 839, Institut du Fer à Moulin, Université Pierre et Marie Curie, F75005 Paris. E-mail: manuel.mameli@inserm.fr

Published by AAAS