MATERIALS SCIENCE

Solar cells go round the bend

With high oil prices sparking a surge of interest in alternative energy sources, solar cells have become the subject of intense research. Much of this effort focuses on finding new designs that open up fresh applications. John Rogers and colleagues now report just such a development (J. Yoon *et al. Nature Mater.* doi:10.1038/nmat2287; 2008) — tiny, ultrathin cells made of silicon that, when fixed in arrays on a flexible substrate, create large, bendy solar cells (pictured).

The authors carve their microcell arrays from a rectangular block of silicon. They begin by etching the outlines of the microcells (the tops and sides) onto the upper surface of the silicon block. They then make electronic junctions and electrical contacts by 'doping' the silicon, adding boron and phosphorus, and using an inert mask to define the regions to be doped. A further round of etching exposes the final three-dimensional shape of the microcells, retaining a thin sliver of silicon to anchor the cells to the block. Finally, the base of the wafer is doped with boron, to yield functioning solar microcells.

To make bendable, large-scale solar cells, Rogers and colleagues use a printing technique. They press a flat stamp onto the arrays of microcells on the silicon block, breaking the anchors that tether them to the silicon. The microcells stick to the soft surface of the stamp, and are transferred to a flexible substrate simply by pressing the stamp onto the substrate. The authors then construct electrodes to connect the microcells to each other, using one of various established methods.

The resulting devices have several desirable properties. First, they are remarkably light, which, along with their flexibility, allows them to be transported and installed more easily than existing solar cells. Second, they work just as efficiently when bent as they do when flat, so they could be fixed to curved or irregular surfaces. Furthermore, they can be made to be transparent, which would allow them to be used on windows. And because the microcells are so thin, less silicon is used, minimizing costs.

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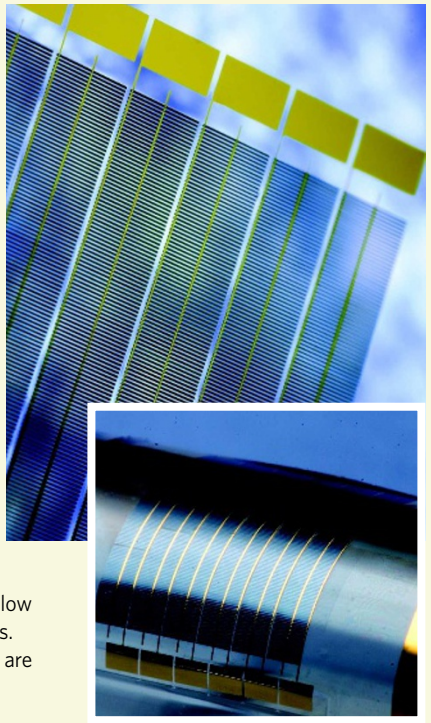

J. YOON *ET AL.*

in the nucleus accumbens — a primary brain region in the addiction neurocircuitry. So Pulipparacharuvil and colleagues[1] hypothesized that such cocaine-induced structural neuroplasticity might also be regulated by MEF2. Indeed, they demonstrate that this transcription factor, which is highly expressed in medium spiny neurons and is predominantly active under normal conditions[1], is affected by chronic cocaine administration. Specifically, long-term exposure to cocaine seems to prevent MEF2 dephosphorylation by a calcium/calmodulin-dependent phosphatase enzyme known as calcineurin, thereby suppressing its activation.

Cocaine-induced MEF2 inhibition also seems to involve enhanced phosphorylation of this transcription factor by a kinase known as Cdk5. In the nucleus accumbens, Cdk5 activity — which modulates behavioural responses to cocaine, such as motivation to consume the drug[5] — increases after chronic exposure to cocaine[6]. Together with Pulipparacharuvil and colleagues' data, these observations[4–6] strongly suggest that repeated exposure to cocaine inhibits MEF2 activity through both enhanced phosphorylation by Cdk5 and attenuation of dephosphorylation by calcineurin. The reduction in MEF2's transcriptional activity in turn promotes increases in the number of dendritic spines.

The basic mechanism underlying experience-dependent synaptic plasticity is often described by the phrase "Neurons that fire together, wire together"[7]. Reward-related associative learning is a form of such 'Hebbian plasticity', in which synaptic connections are enhanced by the improved strength of existing synapses and/or by an increase in the number of such connections. So a logical conclusion would be that cocaine-induced increases in spine density reflect an activity-dependent strengthening of synaptic connectivity, which presumably underlies addictive behaviour. Surprisingly, however, Pulipparacharuvil and colleagues' observations[1] do not support this inference. By manipulating MEF2 activity, they inhibited cocaine-induced increases in spine density. However, this did not seem to prevent increases in the behavioural response to this drug, and might even promote it. So increases in spine density resulting from MEF2 inhibition seem to be associated with reduced behavioural sensitivity to cocaine.

If bulk increase in spine density within the nucleus accumbens does not contribute to enhanced behavioural responses to cocaine, then what is its function, and how can it be reconciled with the processes of experience-dependent associative learning? One confounding aspect of Hebbian plasticity is that, when allowed to proceed unchecked, activity-dependent changes in synaptic connections can destabilize neural networks[8]. In self-defence, the brain uses homeostatic-plasticity mechanisms to oppose such destabilizing effects.

Homeostatic plasticity tends to occur on a large scale to maintain the overall firing activity of a neuron. This allows synapse-specific remodelling of neuronal circuits to proceed through Hebbian mechanisms while maintaining stability of the overall neural network. A major excitatory input reaching medium spiny neurons originates from the prefrontal cortex, but chronic exposure to cocaine markedly alters the output of prefrontal cortical neurons projecting to the nucleus accumbens[9]. So, as Pulipparacharuvil *et al.* suggest, an intriguing possibility is that cocaine-induced increases in spine density in the nucleus accumbens, which are mediated by MEF2 inhibition, may represent a homeostatic response to altered excitatory input from the prefrontal cortex.

These findings[1] provide a direct challenge to the view that increased spine density induced by repeated exposure to psychostimulants underlies maladaptive plasticity. Moreover, they agree with previous observations[10] that identified compensatory drug-induced neuroadaptations. Future research into the neuroplasticity induced by addictive drugs must therefore consider competition between activity-dependent remodelling of synaptic connections and homeostatic adaptations that maintain overall stability in neuronal networks. ■

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