EDITED BY GILBERT CHIN AND JAKE YESTON

CHEMISTRY

Upside of a Double Negative

Chemical bonds tend to form most easily by the attack of an ion on an oppositely polarized center, or by the neutral interactions of radical centers bearing unpaired electrons. Bisai *et al.* took the somewhat counterintuitive approach of preparing two negatively charged sites and then adding an oxidant to draw them together. The deprotonation of both a carbon and a nitrogen center came at a late stage in the synthesis of the polycyclic organic natural product lyconadin A. When more traditional approaches toward linking C and N to close the ring had failed, the authors relied on the likelihood that a lithium cation would stabilize a dianion through simultaneous coordination to both centers. Treatment with iodine then induced C-N bond formation in 80% yield, affording an impressive overall product yield of 10% over the 18 steps of the synthesis. — JSY

J. Am. Chem. Soc. **130**, 10.1021/ja8028069 (2008).

CLIMATE SCIENCE

At a Loss

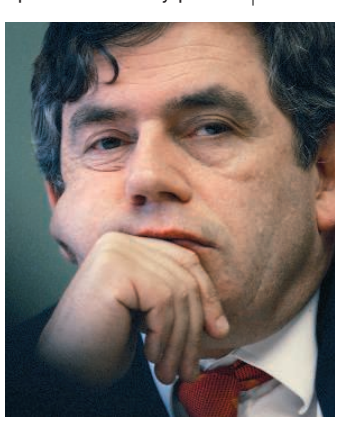
Sea-ice coverage in the Arctic plummeted in the summer of 2007 to levels never before observed, surprising even experts who had witnessed the decades-long decline and predicted that the ice pack would continue to shrink at an increasingly rapid rate. Why did so much ice disappear? Zhang *et al.* conducted a retrospective modeling study of the evolution of Arctic sea-ice coverage and found that preconditioning, anomalous winds, and ice-albedo feedback were responsible for most of the retreat. Years of warming climate there preconditioned the ice for disappearance by thinning it significantly, pushing it ever closer to the point of complete melting, while stronger than normal winds pushed unusually large amounts out of the Arctic basin. The ice thinning and exposure of open water that these processes caused left the remaining ice even more susceptible than normal to loss due to heating of the upper ocean, increasing the intensity of the positive ice-albedo feedback and accelerating the rate of ice loss. Once summer had passed and temperatures had dropped low enough for ice to begin to regrow, 10% more ice than usual had vanished, 70% of it due to melting and 30% due to ice advection. The large ice loss, coupled with prevailing climate trends, suggests that Arctic sea ice has become particularly vulnerable to anomalous atmospheric forcing. — HJS

Geophys. Res. Lett. 10.1029/2008GL034005 (2008).

NEUROSCIENCE

Out With the Old, In With the New

Might this adage, which some pundits have claimed as the basis for the vernal electoral calamities that have befallen the Labour Party in the United Kingdom, apply equally forcefully to the turnover of neurons in the brain? Adar *et al.* have performed a painstaking histological and immunofluorescence accounting of the survival likelihoods of newly born neurons in the brain of the zebra finch, a songbird that serves as an animal model for studying innate and learned influences on vocal communication. They focused on the nidopallium caudale (NC) region because it participates in auditory processing and is activated by social stimuli (other songbirds in this notably social species). By varying the complexity of the social environment, they found that the youngest cells—which had recently migrated from the site of their birth and were still becoming integrated, quite literally, as they established syn-aptic connections with existing NC neurons—were more likely to have survived if the bird had been exposed to a large group of male and female birds; conversely, in birds housed with only one other individual, the survival of older (though still relatively young) cells was enhanced. One interpretation of these data is that an increase in demand—in the form of an upturn in auditory/social inputs needing to be processed—acts as a selective pressure favoring the survival of new recruits. — GJC



J. Neurosci. **28**, 5394 (2008).

BIOMEDICINE

The School of Hard Knocks

The identification of a disease-causing gene mutation in humans is typically followed by a flurry of research aimed at elucidating the normal function of the gene and how disruption of that function produces the specific pathological features of the disease. These projects often rely on the phenotypic characterization of mice in which the murine ortholog of the gene has been inactivated (knock-out mice) or in which the specific disease-causing mutation has been introduced into the murine germline (knock-in mice).

Although such models are informative, a recent analysis serves as a reminder that mice are

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