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In This Issue

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End-Permian mass extinction explored

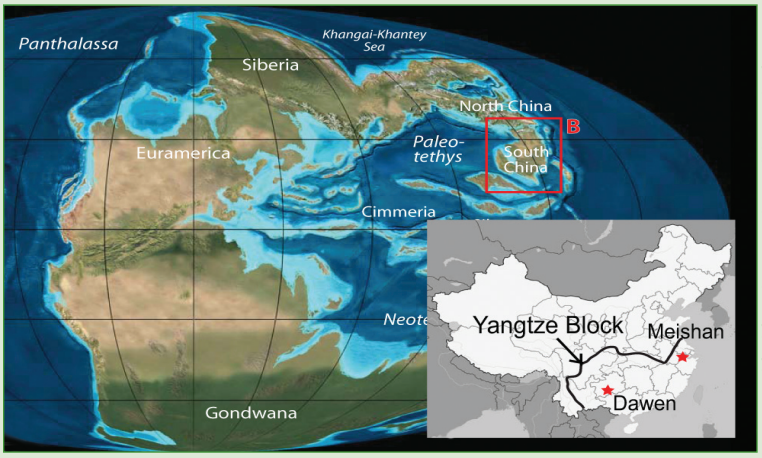
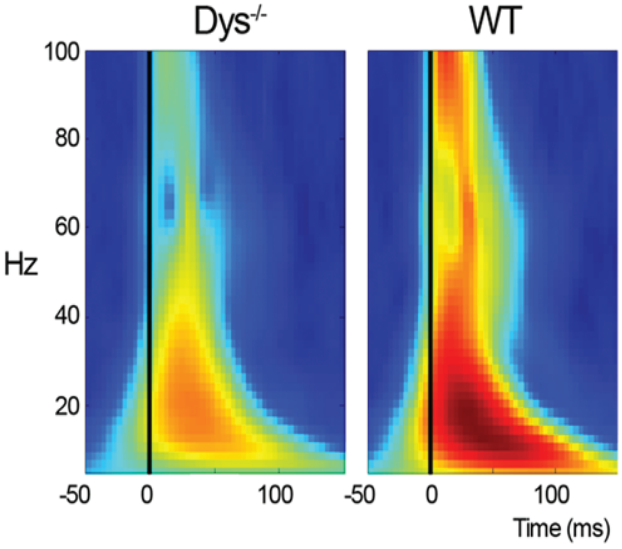


Image modified from base map by R. Blakey and ref. 14 in Brennecka et al., pp. 17631–17634.

Oceanic oxygen levels and mass extinction.

Researchers have long debated the cause of the end-Permian mass extinction, one of Earth's most catastrophic prehistoric extinctions that wiped out 90% of marine species. Gregory Brennecka et al. (pp. 17631–17634) performed a uranium isotope analysis of carbonate rocks deposited in what is now southern China to explore potential links between the end-Permian mass extinction and the timing and extent of changes in the oxygen levels in the ocean; carbonate rocks contain a snapshot of the isotopic signatures of the seawater into which they are deposited. Previous studies had suggested that oxygen-depleted conditions in the deep ocean led to a gradual build-up of noxious gases, such as hydrogen sulfide, and eventually poisoned many organisms. Contrary to those findings, the authors' analysis suggests that the intensification of anoxia, or a lack of oxygen, in the ocean either coincided with or shortly preceded the previously established start of the extinction, and likely persisted for at least 40,000 years afterward. The findings call into question previous hypotheses that suggest an extended period of oceanic anoxia before the extinction. According to the authors, the findings do not refute the possibility of hydrogen sulfide as a potential culprit in the extinction, but they support a scenario where the gas accumulated relatively close to the ocean surface, not in much deeper zones. — P.N.

Modeling schizophrenia in mice

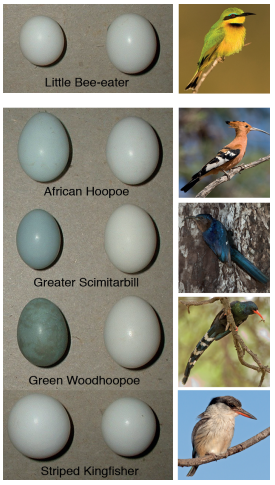


Dysbindin-mutant mice have abnormal brain activity in response to auditory stimuli.

Clinical studies and animal models suggest that loss of inhibitory neuron activity plays a key role in schizophrenia. It is further known that the disease is more common among people harboring a genetic variation that reduces levels of the protein dysbindin in the brain, but how this protein is linked to the clinical features of the disease remains unclear. Gregory Carlson et al. (pp. 17595–17596) reveal a functional association between dysbindin downregulation and inhibitory neuron impairment in mice. The authors found that dysbindin-mutant mice recapitulated a broad set of phenotypes associated with schizophrenia, including abnormalities in high-frequency brain activity in response to auditory stimuli and reduced levels of the protein parvalbumin (PV), a marker for a type of inhibitory neuron. These changes were further associated with deficits in the inhibitory responses of neurons, as monitored by voltage-sensitive dye imaging of a neuronal circuit in mouse brain tissue slices. Together, the findings suggest a possible mechanistic link between an established genetic liability for schizophrenia and the biochemical underpinnings of the disease, implicating disrupted inhibitory neuron activity involving PV-positive cells as the molecular mechanism involved in schizophrenia, according to the authors. — N.Z.

Ancient host specificity of a parasitic bird

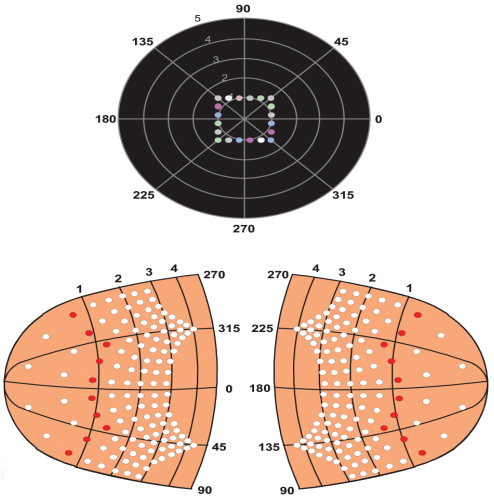
The coevolution of parasitic species and their hosts can result in multiple parasitic strains, each adapted to a specific host. Claire Spottiswoode et al. (pp. 17738–17742) studied the host-specific adaptations of parasitic Greater Honeyguides, small tropical birds that leave their eggs to be reared by other species. Data from fieldwork in Zambia and a museum collection amassed over 3 decades revealed that the size and shape of honeyguide eggs vary in accordance with egg traits in their hosts, tasked with rearing the abandoned eggs. The researchers analyzed DNA from cell organelles called mitochondria, transmitted maternally from mothers to their daughters.



Parasitic honeyguide eggs (*right*) resemble those of their hosts (*left*) in size and shape.

They found two female lineages associated with two distinct groups of hosts: those nesting in terrestrial burrows and those nesting in tree cavities. Deep genetic divergence between these honeyguide lineages indicates they have been faithful to their respective hosts for millions of years. In marked contrast, data from several nuclear genes, which are inherited from both parents, indicated that interbreeding between male and female honeyguides reared by different hosts prevents the strains from diverging into two separate species. The study provides an unusual example of the maintenance of long-standing adaptive divergence within a single species, the authors suggest. The findings raise puzzling questions about the genetics of host-specific adaptation in these and other parasitic birds, according to the authors. — S.R.

Placenta sacrifices for brain development



Array of electrodes (red) in the monkey brain, activated by a camera and computer system.

A prosthetic device capable of restoring sight to the blind would require a camera and computer system to convert visual signals to electrical stimulations in the primary visual cortex, known as area V1. Researchers have previously shown that electricity activates neurons in the monkey brain. Peter Schiller et al. (pp. 17809–17814) inserted microelectrodes into the primary visual cortex of two rhesus monkeys to determine the type of mental images created by this process. The microelectrodes permitted the researchers to electrically stimulate the monkeys' brains and also to record neuronal activity as the primates performed two visual tasks. Analysis of the monkeys' neuronal activity as well as eye movements throughout the experiments determined the location, size, color, and contrast of the image created by the stimulation. Stimulation of single sites within area V1 of the monkey brain allowed the monkeys to see a small dark spot composed of various low-contrast colors, the authors found. The findings could help researchers test neural network models aimed at understanding the features of area V1, possibly contributing to the development of a prosthetic device for the blind, according to the authors. — J.V.