

narcotic properties are recorded for chemically unreactive N<sub>2</sub> gas, giving rise to “l’ivresse des grandes profondeurs” (the “rapture of the deep”—the intoxication experienced by divers) (8). An early explanation for xenon anesthesia was given by Pauling, who suggested that clathrate hydrate structures encapsulating the rare gas atoms were formed near synapses, impeding inter-neuronal transmission (9). Recent results suggest a more interesting solution. The protein complexes that form transmembrane ion pumps associated with neurotransmitters contain hydrophobic regions. It is thought that neutral species such as xenon and N<sub>2</sub> might enter these regions, especially at high pressure, and interfere with the neuronal process to result in anesthesia and narcosis (10, 11).

Highly oxidizing conditions are usually needed to activate xenon into true chemical reactivity and to form bonds with species such as oxygen. However, Sanloup *et al.* have used optical spectroscopy and synchrotron x-ray diffraction combined with chemical analysis to show that xenon can react with natural silicate materials, including SiO<sub>2</sub>, to form xenon oxide species under the high-pressure, high-temperature conditions found in Earth’s crust (1). This is an important result, because the noble gases form useful geochemical tracers. Formed by radioactive decay processes or encapsulated within deep Earth materials since the formation of the planet, the “inert” gaseous elements are thought to diffuse out from the mantle, core, and crust at well-defined rates. Xenon is particularly important in this regard. If it does undergo redox reactions and enter into chemical combination with silicates and other oxides, this could explain the apparent “xenon deficit” in the atmospheres of Earth and Mars, remarked upon by geochronologists and geophysicists (12). If this is true for xenon, then perhaps it also occurs for the radioactive rare gas, radon, that is formed by radioactive decay processes in crustal rocks.

The physical properties of xenon in deep Earth environments are as strange as its possible chemical behavior. Jephcoat (13) has shown that under lower mantle and core conditions, the melting point of xenon exceeds that of iron, as does its density. This means that if xenon did not react chemically with mantle or core materials, it would fall as “hail” toward the center of Earth, through the molten outer core. However, the results of Sanloup *et al.* suggest that it can also react with silicates, oxidizing them to metallic alloys or replacing silicon in mineral structures. There might be new geochemical partitioning equilibria to be considered within the deep crust, mantle, and core, involving xenon physics and chemistry.

It is of interest that Sanloup *et al.* carried out their experiments in platinum capsules,

because reactions involving platinum were how Bartlett initiated his first xenon chemistry experiments (5). Perhaps the capsules are not completely innocent in the high-pressure and high-temperature experiments, because they might act as reaction sites for xenon in addition to providing the host for platinum-silicon alloys formed during the presumed silicate reduction reactions. However, even if that is the case, it does not rule out the potential importance of metal-silicate reactions involving xenon within Earth. These results could presage a new area in xenon solid-state chemistry under high-pressure conditions, which might be extended to other noble gases that have not yet been chemically awakened, if the conditions are made right.

## PALEONTOLOGY

# Dinosaurs Dined on Grass

Dolores R. Piperno and Hans-Dieter Sues

Grasses (family Poaceae or Gramineae), with about 10,000 extant species, are among the largest and most ecologically dominant families of flowering plants, and today provide staple foods for much of humankind. Dinosaurs, the dominant megaherbivores during most of the Mesozoic Era (65 to 251 million years ago), are similarly one of the largest and best known groups of organisms. However, the possible coevolution of grasses and dinosaurs has never been studied. Now, Prasad *et al.* (1) report on page 1177 of this issue their analysis of phytoliths—microscopic pieces of silica formed in plant cells—in coprolites that the authors attribute to titanosaurid sauropods that lived in central India about 65 to 71 million years ago. Their data indicate that those dinosaurs ate grasses.

Part of the difficulty in studying the question of dinosaur-grass coevolution results from the poor quality of the fossil record for early grasses. The earliest unequivocal grass fossils date to the Paleocene-Eocene boundary, about 56 million years ago (2, 3), well after the demise of nonavian dinosaurs at the end of the Cretaceous Period. Pollen and microfossils of Poaceae are uncommon in sedimentary strata until the middle Miocene, about 11 to 16 million years ago, when the family is thought to have undergone considerable evolutionary diversification and ecological expansion (2). Thus, dioramas in museums

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have long depicted dinosaurs as grazing on conifers, cycads, and ferns in landscapes without grasses. The work of Prasad *et al.* (1) is the first unambiguous evidence that the Poaceae originated and had already diversified during the Cretaceous. The research shows that phytoliths, which have become a major topic of study in Quaternary research over the last 20 years (4–8), can provide a formidable means for reconstructing vegetation and animal diets for much earlier time periods when early angiosperms were diversifying. These remarkable results will force reconsideration of many long-standing assumptions about grass evolution, dinosaurian ecology, and early plant-herbivore interactions.

Scientists have long known that grasses make distinctive kinds of phytoliths in the epidermis of their leaves and leaflike coverings that surround their flowers (9). More recent work has examined in greater detail phytolith characteristics from a large set of grasses comprising taxa representing the entire range of diversification within the family, showing that discriminations at the subfamily, tribe, and genus levels are often possible (1, 4–8, 10). In addition, publication of a well-resolved consensus phylogeny of the Poaceae by the Grass Phylogeny Working Group (GPWG) (11) considerably advances our overall understanding of the evolutionary history of grasses and leads to improved interpretations of the early grass fossil record. For example, by mapping the phytolith characters that discriminate clades and subfamilies of extant taxa onto this phylogenetic tree, we can infer how phytolith morphology changed at

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major, diverse effects on each group (12). The documentation by Prasad *et al.* of a range of poacean taxa in the Late Cretaceous of India makes the possibility real that titanosaurid sauropods were not the only grass eaters of the era, and that coevolutionary interactions between grasses and diverse vertebrate herbivores may have greater antiquity than previously believed. For example, the enigmatic gondwanatherian mammals with their high-crowned (hypsodont) cheek teeth could have eaten grasses (13). It has often been argued that the intense consumption of vegetation by herd-forming herbivorous dinosaurs led to the diversification of angiosperms during the Cretaceous (12). Prasad *et al.* identified the silicified remains, including trichome phytoliths, of a variety of nongrass angiosperms in the coprolites, providing direct evidence that the dinosaurs were generalist herbivores.

Phytoliths are common in an array of extant basal angiosperms, monocotyledons, and eudicotyledons in addition to grasses and are among the few substances capable of inducing morphological changes to animal mouthparts (14). It is believed that they constitute an important type of mechanical plant defense against both insect and vertebrate herbivory (14). By 65 million years ago, therefore, angiosperms may have experienced considerable herbivore pressure such that some had evolved simple, inexpensive mechanical defenses that involved impregnating their structures with silica. The new data provided by Prasad *et al.* are certain to help resolve these and other important issues in Mesozoic terrestrial ecology.

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## GENETICS

## Two Genes Link Two Distinct Psychoses

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During the past decade, the tools of molecular genetics have begun to bear fruit in searches for selective genes involved in the major psychotic illnesses, schizophrenia and affective disorder. The most direct evidence involves the gene *DISC1* (*disrupted in schizophrenia 1*) (1). On page 1187 of this issue (2), Millar *et al.*, who pioneered the discovery of *DISC1* (3), now report a chromosomal translocation in schizophrenia involving the gene encoding phosphodiesterase 4B, *PDE4B*. *DISC1* binds *PDE4B1*, but an increase in cellular cyclic adenosine monophosphate (cAMP) dissociates the proteins and activates the phosphodiesterase. This discovery affords a molecular explanation of cognitive and affective dysfunction and may clarify the relationship between schizophrenia and affective illness, thus potentially leading to new therapeutic strategies.

Genetic linkage and association studies have implicated several candidate genes in schizophrenia, including those encoding neuregulin 1, dysbindin, and regulator of G protein signaling 4 (4). In contrast to these suggestive findings, *DISC1* is the first gene

whose chromosomal aberrations clearly segregate with psychotic disturbance. This includes both schizophrenia and affective disorder, with direct involvement in at least two distinct pedigrees (5, 6). Furthermore, linkage and association studies establish *DISC1* as a candidate susceptibility gene in large populations of patients with schizophrenia or affective disorder (4). The intriguing relationship of *DISC1* to both of these disorders, long thought to be distinct, has been strengthened by a link to chromosome 1q42, the region of *DISC1*, for schizoaffective disorder. As the name implies, this mental disorder shares the characteristic of both cognitive and mood-related illnesses (7).

How *DISC1* influences mental function is beginning to be clarified. In schizophrenic patients, *DISC1* variants are linked to specific neurocognitive impairments (8–11). A single-nucleotide polymorphism in *DISC1* that leads to an amino acid change (Ser<sup>704</sup> → Cys) is associated with schizophrenia and correlates with variations in hippocampal size and function during cognitive tasks in normal subjects (8) as well as cognitive variations in aged normal subjects (11).

Cellular functions of *DISC1* have been revealed through the identification of various proteins that interact with it: microtubule-centrosome-associated proteins such as NUDEL (see the first figure); actin-

related proteins such as FEZ-1; postsynaptic density-related proteins such as citron; and nuclear proteins such as activating transcription factor 4 (12–14). *DISC1* is critical for maintaining a complex containing NUDEL and the microtubule-associated motor protein dynein at the centrosome. Mutations in *DISC1*, such as those that produce truncated forms of the protein, prevent dynein-centrosome interaction in some schizophrenic patients (15). In addition, reduced expression of *DISC1* in mice disturbs proper neuronal migration and arborization of dendritic neuronal processes in the developing cerebral cortex (15). These findings have suggested that localization of dynein to centrosomes through *DISC1* is critical for dynein signaling that regulates the growth of neuronal processes and development of the cerebral cortex. The *DISC1* mutation found in the Scottish pedigree could lead to a truncation of *DISC1* at its carboxyl terminus and/or degradation of the protein. Failure to detect the truncated *DISC1* in lymphoblasts of a few schizophrenics from the Scottish pedigree may also be attributed to variations in *DISC1* protein isoforms (see the second figure). Such isoforms could exhibit differences in their metabolism in the brain and lymphoblasts as well as in the developing and adult brain (1, 16). Loss of *DISC1* and/or actions of the truncated mutant *DISC1* can cause dysfunctions that would fit with substantial evidence that schizophrenia is a disorder of neural development (17–19).

Millar *et al.* (2) now report a patient with a chromosomal translocation involving the gene encoding *PDE4* that leads to a 50% reduction in expression of *PDE4B1*, one subtype of *PDE4B*. They detect binding of *PDE4B1* with *DISC1* (see the second fig-

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