

edited by Gilbert Chin

GEOPHYSICS

Hawaiian Carats

Diamonds, which require high pressures and generally moderate temperatures in order to form, are found primarily in volcanic rocks (kimberlites) that erupt through old, thick continental crust. The geothermal gradient means that these locations are much cooler, and the volcanic rocks derive from several hundred kilometers or more beneath the continents. A few diamonds have been found to be derived from crustal carbon in subduction zones (which are cool, too). Only one diamond has been found in an oceanic setting: an ancient oceanic plateau in the southeast Pacific.

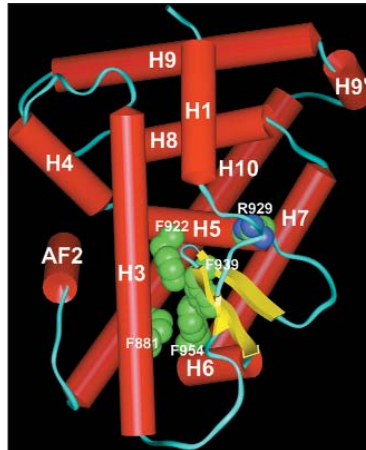
Wirth and Rocholl report the recovery of microdiamonds from a recent volcanic eruption in the Hawaiian Islands (on Oahu). Their occurrence is enigmatic because the mantle beneath Hawaii is hot and Hawaiian lavas are thought to come from outside the regions where diamond can exist stably. These microdiamonds may have formed in a reducing fluid deep in the mantle beneath Hawaii, followed by subsequent transport of this mantle upward to the region where Hawaiian lavas originate. — BH

Earth Planet. Sci. Lett. 10.1016/S0012-821X(03)00204-8 (2003).

CELL BIOLOGY

Safe in the Womb

Mammalian embryos are surrounded by the trophoblast giant cell layer, an epithelial layer that forms a barrier between maternal and embryonic tissues. Jaquemar *et al.* examined mutant mice deficient in an intermediate filament protein, keratin 8. The majority of mouse embryos lacking keratin 8 fail to develop beyond midgestation. However, chimeric concepti in which the embryonic tissues lacked ker-



Four phenylalanine residues (green) occupy the pocket that might otherwise bind a steroid ligand.

atins 8 but contained extraembryonic tissue derived from tetraploid wild-type cells could develop, suggesting that a failure in the trophoblast giant cell layer could be responsible for the embryonic deaths. The growth factor TNF (tumor necrosis factor) contributed to breaching epithelial integrity because, in the absence of maternal TNF, more keratin 8-deficient embryos were able to survive. When TNF-dependent apoptosis was induced by the addition of concanavalin A, mutant embryos lacking keratin 8 were more likely to fail because of the formation of hematomas at sites of trophoblast giant cell layer barrier breakage. Thus, this epithelial keratin plays an important role in protecting trophoblast giant cells from maternal TNF-induced apoptosis and enabling the embryo to develop to term. — SMH

J. Cell Biol. 161, 749 (2003).

DEVELOPMENT

A Receptor Without a Ligand

The insect hormone α -ecdysone is considered to be the major trigger of morphogenic change during *Drosophila* larval development and entry into metamorphosis. Its actions are mediated through a heterodimer complex consisting of two nuclear receptors: the ecdysteroid receptor (EcR) and Ultraspiracle (USP). Nevertheless, the presence of other ecdysteroids in insect hemolymph at specific developmental stages has raised the question of whether and how these other compounds might function.

Only one other *Drosophila* nuclear receptor, DHR38, is known to form a complex with USP. Baker *et al.* now show that a DHR38-USP complex governs a second ecdysteroid signaling pathway. Several naturally occurring *Drosophila* ecdysteroids stimulated DHR38-dependent transcription in cultured *Drosophila* cells, but only when the receptor formed heterodimers with a constitutively activated form of USP. Hence, unlike EcR, DHR38 requires coactivation of its heterodimer partner in order to be competent to respond to ecdysteroids. The structure of the "ligand binding-domain" of DHR38 revealed the lack of well-defined ligand-binding and conventional coactivator binding sites, explaining why no such interactions were detected in biochemical assays. Although both EcR and DHR38 require heterodimerization with USP to be ecdysteroid-responsive, these receptors appear to use alternate mechanisms for transactivation of gene expression. — LDC

Cell 10.1016/S0092867403004203 (2003).

NEUROSCIENCE

Practice Makes Imperfect

Musicians can be at risk of developing focal hand dystonia because they practice repetitive fine movements of their hands and fingers. The impairment in motor control and the difficulty in making coordinated noncramping movements are thought to reflect an abnormal and induced disorganized representation of the fingers in the somatosensory cortex or motor cortex or both. Physical therapy to regain sensory discrimination and motor fitness has been employed, and Candia *et al.*

describe evidence indicating that behavioral improvement is accompanied by neural changes. They find that the topographic maps of the fingers in the somatosensory cortex return to their normal order in which the distance between digits 1 and 2 (D1-D2) is less than the distance D2-D5, which, in turn, is less than D1-D5. — GJC

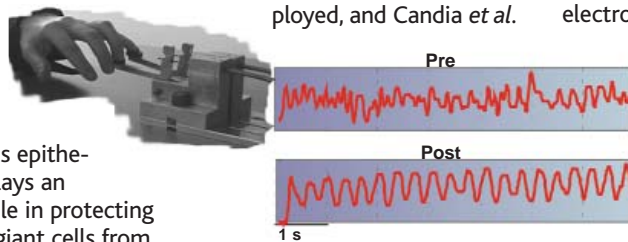
Proc. Natl. Acad. Sci. U.S.A. 10.1073/pnas.1231193100 (2003).

CHEMISTRY

Layered Repair

Depositing alternating layers of charged polymers (polyelectrolytes) is one way to build up surfaces in a controllable fashion, and Thierry *et al.* have applied this layer-by-layer approach to protect the insides of blood vessels. During operations, such as the reopening of blocked arteries, the

CONTINUED ON PAGE 1625



Movement of a dystonic right middle finger during a trill pre- and post-therapy as recorded on a dexterity device.

vessel walls are often damaged and lose portions of the protective endothelial cell lining. These vessels are then susceptible to the accumulation of debris, which leads to a narrowing of the artery (restenosis). Alternating layers of hyaluronan and chitosan were used to coat the walls of aortic porcine arteries; the growth of blood clots was substantially inhibited in comparison to uncoated, damaged arteries. Both of these polysaccharides are known to be biocompatible and to have healing capabilities and anti-inflammatory properties. Some of the hyaluronan was replaced with a hyaluronan-arginine complex in order to see if the multilayer coating could mimic the localized drug delivery aspect of the latest generation of stents. Although most of the arginine was released in an initial burst, it did help to reduce the adhesion of platelets to the artery walls. — MSL

J. Am. Chem. Soc. 10.1021/ja034321x (2003).

ranging from <1 to 12% in comparison to the phytoplankton carbon fixation measured during normal ozone concentrations. Most regional estimates of ozone-related productivity losses have been based either on spatial extrapolations of these local values or on calculations that assume a spatially uniform ozone concentration (an idealized hole).

In order to avoid these assumptions of homogeneity, Arrigo *et al.* use numerical modeling and accurate maps of ozone abundance to assess the large-scale effects of stratospheric ozone depletion on oceanic primary production. They take into account the complicating effects of vertical mixing, spatial and temporal variations in ozone abundance, sea ice cover, cloudiness, wind speed, ocean mixed layer depth, and phytoplankton chlorophyll *a*. They conclude that ultraviolet radiation-induced losses of surface phytoplankton production are substantial under all ozone conditions, mostly due to ultraviolet A,

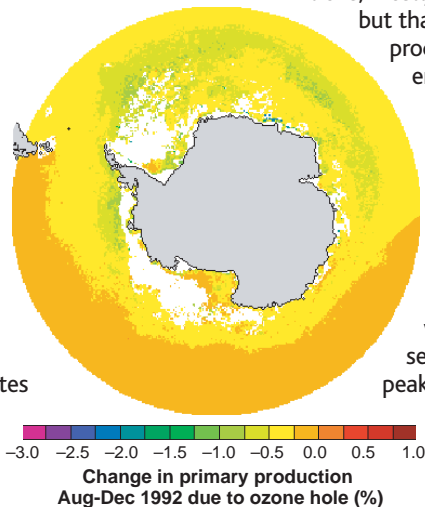
but that the loss of primary production resulting from enhanced fluxes of ultraviolet B (integrated to 0.1% light depth) was less than 0.25%. The loss of primary production is minimized by the strong attenuation of ultraviolet radiation within the water column and by sea ice, which is at its peak extent during the months when ozone is lowest. — HJS

J. Geophys. Res. 108, 3154 (2003).

OCEAN SCIENCE

Light Sunburn

Springtime depletion of stratospheric ozone over Antarctica (the ozone hole) allows more ultraviolet B radiation to reach the sea surface, which decreases phytoplankton productivity. In localized studies, estimates of the impact on oceanic photosynthesis have varied, with daily losses



Change in primary production Aug-Dec 1992 due to ozone hole (%)

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Mitotic Regulator on the Move

The cyclin-dependent kinase Cdc2 is usually thought of as a promoter of mitosis. Manes *et al.* describe a possible role for Cdc2 in regulating cell migration. Expression of the membrane protein $\alpha V\beta 3$ integrin is associated with migratory and invasive behaviors of some cancer cells, and Cdc2 levels were elevated in a prostate cancer cell line overexpressing $\alpha V\beta 3$ integrin. Ectopic expression of Cdc2 increased migration in cell lines; conversely, inhibition of Cdc2 activity by expression of a dominant negative or with pharmacological inhibitors reduced migration of cultured cells. Prostate cancers often show increased expression of $\alpha V\beta 3$ integrin, and increased expression of Cdc2 in prostate cancer correlates with more aggressive cancers. Thus, the authors note that key actions of Cdc2 in this instance may occur through promotion of migration and invasion rather than through the more customary role of promoting cell proliferation. — LBR

J. Cell Biol. 161, 817 (2003).

Safe in the Womb

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