EDITORS' CHOICE

edited by Stella Hurtley

ECOLOGY/EVOLUTION

Sons and Daughters

Male springbok.

Biases in the ratio of males to females occur in many polygynous mammal species. According to the mother's condition, investment in sons or daughters may have different fitness benefits in terms of the quality of offspring and hence quantity of grandoffspring produced. In many cases, such as red deer in Scotland, mothers in good condition differentially invest in sons, because males are more costly to rear. However, the reverse may sometimes be true. Kruger *et al*. studied sex-ratio variation over 30 years in a population of springbok in the southern Kalahari region of South Africa. Females in better condition produced more daughters than sons. It seems that the faster onset of sexual maturity in females will produce greater fitness returns in the unpredictable Kalahari environment. Rainfall may be an important controlling factor: Daughters were differentially produced earlier in the wet season, giving them a greater chance of reaching maturity in good condition themselves. The mechanism of sex-ratio adjustment probably lies either in an ability on the mother's part to discriminate between X- and Y-bearing sperm or condition-dependent selective implantation of male or female embryos. — AMS

Proc R. Soc. Lond. B **272**, 375 (2005).

MATERIALS SCIENCE

Nylon-Nanotube Fibers

One potential application of carbon nanotubes is as a reinforcing agent for polymer fibers, and direct mixing has led to some significant improvements in tensile strength and Young's modulus. However, incomplete dispersion of the nanotubes, which tend to bundle together, and a lack of direct bonding to the polymer, which helps prevent pullout, have limited performance. Gao *et al*. have overcome some of these difficulties by using caprolactam as both solvent and monomer for incorporating single-walled nanotubes (SWNTs) into a nylon-6 matrix. Nitric acid–treated SWNTs, which are terminated with carboxylic acid groups, are well solvated by amide-containing compounds such as caprolactam. After nylon-6 is formed by the ring-opening polymerization of caprolactam, the amino end of the nylon chain can couple to the SWNTs via an amide linkage. The tensile strength and Young's modulus of nylon-6 improved by about a factor of 2 to 3 for SWNT loadings of 0.5 to 1.5 weight %. — PDS

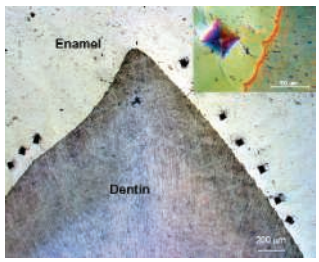
J. Am. Chem. Soc. 10.1021/ja446193 (2005).

MATERIALS SCIENCE

Broken Teeth

Teeth are made up of two calcified tissues that have very different properties: enamel and dentin. The outer coating of enamel is harder but more brittle than the dentin it surrounds. The interface zone between these two structures has been thought to prevent cracks in the enamel from traversing into the dentin, which would cause the fracture and complete failure of a tooth. Using interfacial fracture mechanics, Imbeni *et al*. show that the thin interface layer is not responsible for crack arrest. By creating a series of Vickers microhardness indents in polished sections of healthy extracted teeth, they were able to observe the angle and depth penetration of the cracks that formed. In a majority of the cases, the crack penetrated into the dentin, where it was stopped by the bridging links that form between its mineral and biological components. Although the interface itself is not that strong, the dentin near the interface has collagen fibers that are preferentially oriented perpendicular to the interface and also has a lower mineral content relative to the bulk material, and it is this combination of factors that stops the cracks in their tracks. — MSL

Nature Mat. **4**, 229 (2005).



Cracks induced at the enamel-dentin boundary.

VIROLOGY

Virus-Directed Damage Control

Viruses are successful pathogens because of the many and varied ways they usurp host proteins for their own gain.

Uracil DNA glycosylase (UNG2) is part of the base-excision repair (BER) machinery that helps preserve the integrity of cellular DNA. UNG2 is packaged into the virions of human immunodeficiency virus (HIV) type 1, but the enzyme's role in this context is unclear. Priet *et al*. now show that the virion-associated UNG2 is essential to the viral life cycle. UNG2 counteracts the misincorporation of uracil into viral DNA, an event that could be deleterious to the virus. Intriguingly, in experiments exploring the effect of HIV on host BER, Aukrust *et al*. find that $CD4^+$ T cells from HIV-infected patients exhibit a decline in DNA glycosylase activity and are impaired in their capacity to repair cellular DNA damage. Both abnormalities were ameliorated by antiretroviral drugs.

Whether or not these effects on BER are mechanistically linked, it's clear that in both scenarios the advantage goes to the virus. — PAK

Mol. Cell **17**, 479 (2005); *Blood* 10.1182/blood-2004-11-4272 (2005)

BIOMEDICINE

Presentable Enough for Entry

In the autoimmune condition multiple sclerosis, demyelination and axonal damage ultimately result in impaired motor function. The disease is thought to be caused by invading T cells that react against self components of the central nervous system (CNS), although the identity and location of antigen-presenting cells (APCs) that activate pathogenic T cells is a matter of speculation.

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Published by AAAS

Greter *et al.* studied a multiple sclerosis system in which T cells reactive to a myelin antigen induce experimental autoimmune encephalomyelitis (EAE) upon transfer to mice. Animals lacking organized central lymphoid tissue developed EAE as quickly and with the same severity as control animals, suggesting that pathogenic T cells do not need to be reactivated in peripheral lymphoid organs in order to migrate to the CNS. Resident APCs of the CNS—microglial cells and astrocytes—did not appear to be important for causing disease. Instead, a subset of nonresident dendritic cells was required for disease to progress. In the model and in multiple sclerosis lesions, similar dendritic cells were associated with microvessels of the CNS, suggesting that activation and entry of autoreactive T cells may occur through the presentation of antigen at the blood-brain barrier. — SJS

Nature Med. **11**, 328 (2005).

GEOPHYSICS

The Sum of the Parts

Quantifying how emissions of any particular greenhouse gas affect the radiative forcing of climate is difficult, because of the complexity of the chemical interactions between different species and the wide range of spatial and temporal scales of atmospheric processes. Current assessments of climate change assume that a particular amount of radiative forcing cannot be attributed to any specific emissions species, and instead rely on calculations based on the atmospheric abundance of each species. Shindell *et al.* use a coupled chemistry-aerosol-climate model to hindcast atmospheric composition from preindustrial times to the present, caused by increased emissions of methane and the precursors of tropospheric ozone (NOx, CO, and volatile organic compounds, excluding methane). The global annual average composition response to all emission changes is nearly the same as that of the sum of the responses to individual emissions. Thus, emission figures can be used to calculate the radiative effects of these species. This emissions-based view indicates that the relative importance of various emissions is significantly different than suggested by current abundance-based assessments: Methane, in particular, is almost twice as important as previously suggested. — HJS

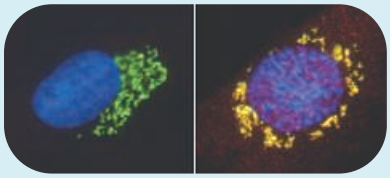
Geophys. Res. Lett. **32**, L04803 (2005).

HIGHLIGHTED IN *SCIENCE*'S SIGNAL TRANSDUCTION KNOWLEDGE ENVIRONMENT

Science's stke www.stke.org

Checkpoint Control at the Golgi

Organelles, such as the Golgi apparatus, must disperse equally during cell division. However, it is not clear whether checkpoints exist for sensing organelle integrity during mitosis. Preisinger *et al.* examined the link between Golgi morphology and cell cycle control. GRASP65, a structural component of Golgi membranes, is required for Golgi fragmentation before entry into mitosis. The C terminus of GRASP65 is phosphorylated primarily by the mitotic kinase Cdk1–cyclin B and to a lesser extent by polo-like kinase 1 (Plk1), an enzyme required for normal mitotic spindle function. Phosphorylation of Golgi-associated GRASP65 on the Cdk1–cyclin B consensus sites correlated with entry into mitosis. Plk1 was detected in a complex with GRASP65 and the Golgi protein GM130 in mitotic cell extracts, but only if GRASP65 was phosphorylated by Cdk1–cyclin B, suggesting that the mitotic kinase creates docking sites on GRASP65 for Plk1. When cells were depleted of Plk1, mitotic fragmentation of the Golgi into clusters was decreased. Overexpression of the GRASP65 C terminus delayed entry into mitosis. However, cells expressing a GRASP65 C terminus harboring a mutant that cannot bind Plk passed through mitosis normally. Passage through mitosis may thus depend largely on the influence of GRASP65-associated Plk1 on the Golgi, where it may help to ensure appropriate Golgi fragmentation and thereby equal partitioning into daughter cells. — LDC



In interphase (left) GRASP (green) labels the Golgi; at the onset of mitosis (right) phosphorylated GRASP (red) also accumulates at the Golgi (yellow) as it starts to disassemble.

CREDIT: FRANCIS BARR, MPI DRESDEN

EMBO J. **24**, 753 (2005).

Checkpoint Control at the Golgi

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