CELL BIOLOGY

Nuclear Membrane Mechanics

In the fission yeast *Schizosaccharomyces pombe*, the nucleus is tethered to the cytoskeleton by KASH domain–containing proteins in the outer nuclear membrane and SUN domain–containing proteins in the inner nuclear membrane. By exerting force on these SUN-KASH complexes, the cytoskeleton controls the position of the nucleus within the cell. Centromeric DNA inside the nucleus has been observed to cluster near SUN-KASH complexes during interphase, raising the possibility that this association mediates a functional connection to the cytoskeleton. King *et al.* have identified an inner nuclear membrane protein (Ima1) in *S. pombe* that links DNA to SUN-KASH complexes. They show that Ima1 binds to centromeric DNA in vitro and colocalizes with the SUN domain–containing protein Sad1 at the inner nuclear membrane; in Ima1-deficient yeast, colocalization between centromeric DNA and Sad1 was disrupted, and nuclei were frequently deformed and asymmetric. The authors propose that these protein-protein interactions may therefore be required to maintain nuclear shape and integrity in the face of cytoplasmic tensioners and provide a means by which cytoskeletal forces contribute to organizing DNA within the nucleus. — NM*

Cell **134**, 427 (2008).

GENETICS

Making a Meristem

Plant development is regulated by meristems, which give rise to all plant organs, including the root, shoot, and flowers. In *Arabidopsis,* the meristem is controlled primarily by a signaling cascade initiated by CLAVATA (CLA) receptors that are activated by CLE peptides. Suzaki *et al.* have examined homologs of the *Arabidopsis* CLAVATA3 protein (a CLE peptide), which controls meristem development, in rice. They found that two closely related rice genes together appear to reflect the general function of the single peptide in *Arabidopsis*, suggesting that development of the meristem is evolutionarily conserved despite the approximately 180 million years separating *Arabidopsis* and rice. However, they also find that within rice, the function of the two peptides has diverged, so that they appear to have undergone sub-functionalization. — LMZ

Plant Cell **20**, 10.1105/tpc.107.057257 (2008).

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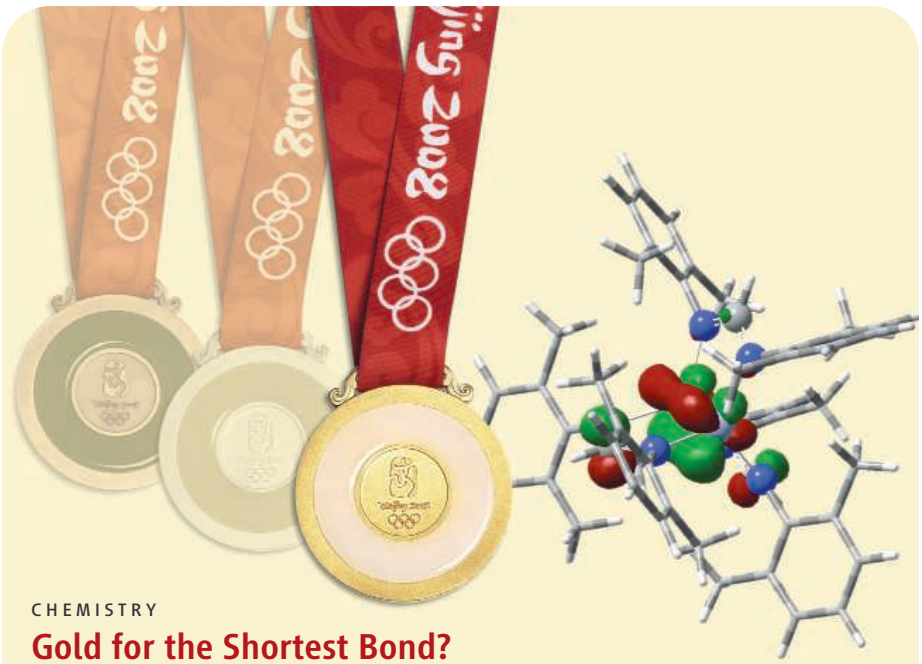


CHEMISTRY

Gold for the Shortest Bond?

As the Olympic Games come to a close, it's worth pointing out that chemists, like athletes, enjoy keeping records. What's the shortest bond? The longest? The weakest? The strongest? In the realm of metals, it turns out that chromium (Cr) has a special distinction. It has just enough electrons that when two Cr atoms come together in the gas phase, they can join in a sextuple bond. Unfortunately, making a compound you can handle in solution requires adding ligands, which bring with them extra electrons that lower the bond order to quintuple at most. After realization of a stable quintuply bonded Cr compound, the question shifted to how short the bond could be, and how equitably the five pairs of electrons were really being shared. Tsai *et al.* have now succeeded in pushing the Cr centers a little closer together, creating a Cr_2 anion protected by three bidentate amidinate ligands that x-ray crystallography revealed to have a central bond length of just under 1.74 Å. At nearly the same time, Noor *et al.* prepared a neutral Cr_2 complex, similarly flanked by bidentate nitrogen ligands (in this case, two amidopyridines), with a bond length of just below 1.75 Å. For comparison, the gas-phase sextuple comes in at 1.68 Å. — JSY

Angew. Chem. Int. Ed. **47**, 10.1002/anie.200801286; 10.1002/anie.200801160 (2008).

EVOLUTION

Adding Less or Substrating More?

Calibrating robust molecular phylogenies of clades of extant species against time offers a means of characterizing the tempo and mode of evolutionary radiations. Often, net diversification is rapid early in the history of a clade and declines later on. This "explosive-early" pattern could be produced either by a fall in speciation rates over time or by a rise in extinction rates—alternatives that support distinct ecological explanations for diversification.

Rabolsky and Lovette present an analytical model, based on the birth-death process, in which speciation and extinction rates vary continuously over time. They apply their framework to three published phylogenies (Australian agamid lizards, Australo-Papuan pythons, and North American wood warblers), and they use simulations to fit features of lineage accumulation curves to different modes of declining net diversification. They find

that the explosive-early pattern can be explained only by declining speciation rates and is not observed in scenarios with high ratios of extinction rates to speciation rates. Their results also show that an apparent excess of recently diverged lineages in lineage-through-time plots (typically seen as the result of increasing diversification or high relative extinction rates) can be produced when declining net diversification is driven by increasing speciation rates. — ShJS

Evolution **62**, 1866 (2008).

CHEMISTRY

Less Strain, More Force

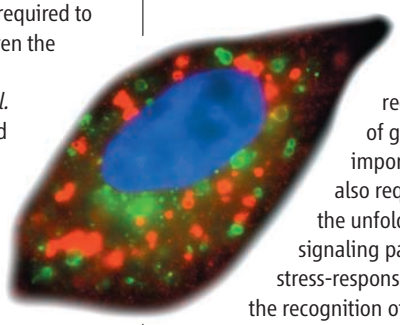
Many studies have probed the force required to pull apart double-stranded DNA. Given the interest in using pore structures to sequence nucleic acids, Ashcroft *et al.* have now measured the force needed to separate hairpins in a self-attracted single strand of DNA as it is pulled through such a pore, in this case a β-cyclodextrin ring. The ring was attached to an atomic force microscope tip and threaded onto a surface-immobilized polyethylene glycol molecule, to which a single strand of DNA that could form a hairpin was then linked at the free end. The force needed to pull the β-cyclodextrin ring through the hairpin was about 40 times greater than that typically needed to pull double-stranded DNA apart directly. The authors note that the transition state for destabilizing the hairpin occurs over a much smaller distance, and so more force must be applied. — PDS

Small **4**, 10.1002/smll.200800233 (2008).

VIROLOGY

Hitchhiking in Membrane Traffic

Autophagy is a process whereby cells rid themselves of defunct organelles and proteins by enclosing them in a double-membraned vesicle that then fuses with and is degraded by a lysosome. Autophagy is important in general cellular homeostasis, in development, and in certain aspects of pathology. In complementary papers, Sir *et al.* found that cells infected with the hepatitis C virus (HCV) accumulate autophagosomes but fail to increase autophagic degradation, probably because of a failure in autophagosome-lysosome fusion. The increase in morphologically distinguishable autophagosomes required the expression of genes known to be important in autophagy, but also required the activity of the unfolded protein response signaling pathway, which is a stress-response pathway involved in the recognition of aberrant proteins in the endoplasmic reticulum. Blocking HCV-dependent autophagosome accumulation by blocking autophagy or by blocking the unfolded protein response pathway suppressed virus replication. Thus, it seems that HCV exploits degradation-defective autophagosomes during its replication cycle. — SMH



HCV-infected cell: autophagosomes, green; HCV protein, red; nucleus, blue.

Hepatology **48**, 10.1002/hep.22464 (2008); *Autophagy* **4**, 830 (2008).

Science Signaling
AAAS

<< Restricted Redundancy

The serine-threonine protein phosphatase PP2A, which participates in signaling cascades induced by TGF-β family ligands, is a heterotrimer composed of catalytic, structural, and regulatory subunits. The B family of regulatory subunits comprises four members that differ in tissue specificity and subcellular localization but otherwise appear similar enough to be functionally redundant. Batut *et al.* report that Bα and Bδ have distinct functions in mediating signaling elicited by the ligands TGF-β, Activin, and Nodal. Bδ knockdown expanded anterior structures in *Xenopus* embryos, whereas Bα knockdown caused loss of anterior structures, suggesting that Bα potentiated and Bδ inhibited Nodal signaling. In *Xenopus* animal cap assays, Bα knockdown blocked Activin-induced axial elongation reduced phosphorylation of the TGF-β family effector Smad2 (pSmad2), and prevented nuclear accumulation of pSmad2. In contrast, Bδ knockdown enhanced elongation and increased the amount and nuclear accumulation of pSmad2, suggesting that Bα and Bδ affected Activin signaling oppositely. Knockdown analyses in keratinocytes and in *Xenopus* animal caps indicated that Bα inhibits lysosomal degradation of ALK4 and ALK5—type I receptors that transduce signaling from TGF-β ligands—and that Bδ inhibits ALK4 receptor clustering. Thus, both subunits appear to influence the threshold ligand concentration that can elicit signaling, but act on distinct processes to exert their opposing effects. — AMV

Development **135**, 2927 (2008).

CREDIT: SIR *ET AL.*, *HEPATOLOGY* **48**, 10.1002/HEP.22464 (2008)

Published by AAAS

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Science **321** (5893), 1134.
DOI: 10.1126/science.321.5893.1134a