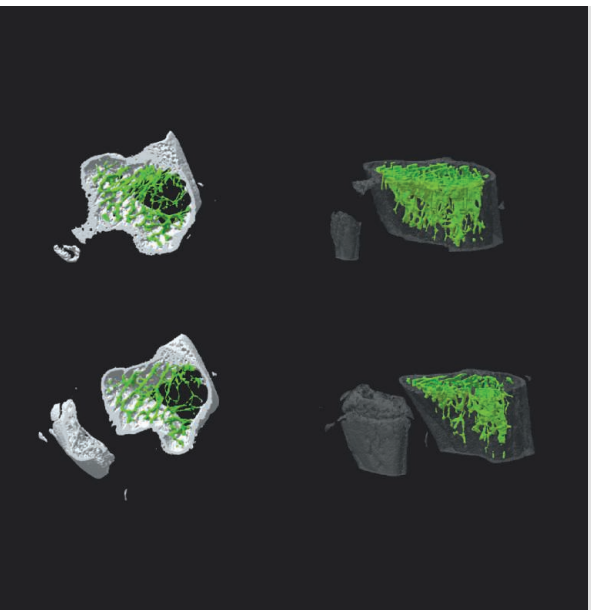

BIOMEDICINE

Splicing Therapy Comes of Age

The aging process is not fun, but when it begins decades ahead of schedule, it's tragic. Children with Hutchinson-Gilford progeria syndrome (HGPS) begin to show features of aging before reaching their teens and usually do not survive to their 20s. HGPS is caused by a mutation in the nuclear envelope protein lamin A, which results in the production of a truncated protein, called progerin. Progerin becomes aberrantly tethered to the cell nucleus, thereby disrupting vital nuclear structure and function. To date, most experimental therapies for HGPS have focused on preventing progerin from reaching the nucleus. Two related reports instead now focus on correcting the splicing defect, so that progerin is not produced in the first place. Lopez-Mejia *et al.* show that splicing factor SRSF1 is a critical participant in the pathogenic splicing mechanism and that its depletion reduces progerin production and corrects the aberrant nuclear phenotype in a cell culture model of HGPS. Osorio *et al.* show that mice carrying the precise genetic mutation seen in HGPS patients develop the main clinical features of the disorder. Many of the features are ameliorated after systemic delivery of an antisense oligonucleotide that prevents the pathogenic splicing event. — PAK

Hum. Mol. Genet. **20**, 10.1093/hmg/ddr385 (2011); *Sci. Transl. Med.* **3**, 106ra107 (2011).

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CHEMISTRY

Solidly Aromatic

Chemists have long been intrigued by the unusual stability conferred by the pi-bonding arrangements in benzene and related unsaturated carbon rings and heterocycles. The phenomenon, termed aromaticity, involves delocalization of a specific number of contiguous electrons (even non–multiples of four) in a plane. In recent years, researchers have increasingly sought to coax heavier congeners of carbon, such as silicon and germanium, into aromatic motifs. Because these elements tend to favor nonplanar bonding geometries, bulky substituents must be appended to push molecular frameworks toward planarity. Kuhn *et al.* explore a preexisting compound with a complementary means of imposing planarity: the constraints of a crystal lattice. Specifically, they probe a Zintl phase of the crystalline lithium silicide $Li_{12}Si_7$. The lattice contains alternating layers of planar five-membered silicon rings and compensating lithium ions, and previous theoretical analyses have predicted aromatic stabilization in these rings. A hallmark of aromaticity is the impact of magnetically induced ring currents on the chemical shifts of nearby nuclei (pushed upfield near the ring's interior; downfield on its periphery) in nuclear magnetic resonance (NMR) spectra. The authors therefore probed samples with solid-state Li NMR spectroscopy, and indeed observed a telltale upfield shift associated with the Li ions centered between the rings. — JSY

Angew. Chem. Int. Ed. **50**, 10.1002/anie.201105081 (2011).

GEOPHYSICS

Parting the Red Sea

The opening and closing of ocean basins has had a dramatic influence on climate and biological evolution over Earth's history. These processes occur roughly every 500 million years, but how they're initiated—particularly in the case of continental rifting to form the seafloor of new ocean basins—remains enigmatic. To understand the beginning stages of continental rifting, Ligi *et al.* performed geophysical surveys and collected seafloor samples from aboard the R/V *Urania* in the Red Sea, which rests atop one of the most prominent modern continental rift zones. The Red Sea Rift is still in the process of rifting, so it includes a span of oceanic crust formed over the past two million years. The data suggest that new seafloor forms initially as an intense pulse of convection-driven mantle upwelling breaks through the continental crust, rapidly declining as convection of the mantle weakens with a widening rift. This process is pulling Africa apart from Arabia to form a new ocean, just as it may have contributed in the past to the breakup of supercontinents. — NW

Geology **39**, 1019 (2011).

REPRODUCTIVE BIOLOGY

Preventing Early Delivery

Preterm birth is a major cause of infant morbidity and mortality, but its causes are largely unknown. Understanding the causative events and identifying early signs are of interest in order to help develop effective therapies to prevent preterm labor and birth. Inflammatory processes function during labor, with leukocytes infiltrating the myometrium (middle layer of the uterine wall) and cervix. Hamilton *et al.* examined inflammatory events in the decidua, or endometrium of the pregnant uterus, which is at the maternal-fetal interface. They found that the number of macrophages was increased in women experiencing full-term or preterm labor. Other immune cell populations were elevated in women in preterm labor relative to full-term labor. A rat model of preterm labor revealed that macrophages infiltrated the rat decidua before labor onset, and decidual infiltration was greater and occurred earlier than myometrial infiltration. Decidual inflammation may therefore be an early event in labor and a possible target for therapeutic intervention. — BAP

Biol. Reprod. **85**, 0.1095/biolreprod.111.095505 (2011).