**E V O L U T I O N**

**Going Farther with Sex**

Plant mating systems are under pressure to produce the most and the best offspring (seeds) and to offer them the greatest opportunity to thrive in the absence of competition by dispersal. Self-pollination is one means by which plants can maximize their reproductive potential, but how it relates to dispersal is unclear. Plants that cannot self-fertilize have been linked to lower dispersal rates because it has been thought that if they were to disperse widely, the lack of a nearby pollen source would result in reproductive failure. Despite this theory, it has been noted that invasive species, those with fleshy fruits, and island colonizers—all of which typify plants with good dispersal systems—tend to have mating systems that separate the sexes or select against self-pollination.

Cheptou and Massol construct a metapopulation model that captures the evolution of dispersal and self-fertilization in environments defined by stochastic pollen limitation. Their calculations show that self-fertilization associates with no dispersal and that outcrossing goes with dispersal, contrary to some previous postulates. Based on these results, they explain the overrepresentation of dioecious plants (those with only male or female individuals) in flora, such as papaya, that have colonized islands. — LMZ


**P H Y S I O L O G Y**

**Growing Fat with Mom’s Help**

A mutation in the α subunit of the guanine nucleotide-binding protein Gα, which transduces signals from various hormone receptors, causes obesity and insulin resistance in the human disorder Albright hereditary osteodystrophy. These disease manifestations occur in individuals with mutations in the maternal allele of the gene encoding Gαs, GNAS, because genomic imprinting causes expression primarily from the maternal allele of GNAS in some tissues. Chen *et al.* provide evidence that the differential expression of GNAS in the brain (the paraventricular nucleus of the hypothalamus) accounts for the metabolic effects of the disease. They observed that mice with a disrupted maternal Gαs allele became obese as a result of reduced energy expenditure. These animals also became insulin-resistant and diabetic even before they became obese. Loss of Gαs function mimicked some of the effects seen with loss of the MC4R melanocortin receptor; melanocortins function through Gαs-coupled receptors to control energy expenditure and food intake. Specifically, effects of melanocortin on food intake operated independently of Gαs. On the other hand, the stimulation of energy expenditure by melanocortin was diminished in animals carrying the mutant maternal allele. — LBR

*Cell Metab.* 9, 548 (2009).

**P L A N T S C I E N C E**

**Abstaining from Sex**

When a desirable plant appears, featuring an unusual flower color or a valuable nutritional trait, it would be handy to be able to propagate identical versions of that individual, but apomixis (asexual reproduction through seeds) is uncommon in crop plants. d’Erfurth *et al.* have identified a gene in the sexual plant *Arabidopsis* that causes an omission of the second meiotic division (*OSD1*) during gametogenesis. Developing gametes that carry mutations in *OSD1* generate two diploid rather than four haploid cells. These gametes are functional and give rise after fertilization to plants with increased ploidy. Although aspects of recombination during the first meiotic division generated progeny not identical to the parent, by combining the *OSD1* mutation with two other mutations that alter the first meiotic division, the authors obtained gametes that carried the same genetic program as the parental plant, a promising start to engineering apomixis. — PJH


**C H E M I S T R Y**

**Nuclear Glow**

Traditional methods of detecting radioactivity rely on sampling the stream of photons and particles that are actually being radiated. The decay of unstable atomic nuclei sends forth a burst of fast-moving charges—at times accompanied by high-energy, very-short-wavelength gamma rays—and multiple sensing technologies are available to capture them. At the same time, the underlying nuclear transformation also leaves behind a slow heavy co-product, and it is this latter decay signature which Berezin *et al.* target for sensing. They present an indole-based dye that fluoresces in the near-infrared (760 nm peak emission wavelength) with an efficiency that varies depending on the identity of a coordinated metal ion. Copper (Cu) quenches the fluorescence, whereas zinc enhances it; nickel has little impact. Thus, decay of the unstable 64Cu isotope to a mixture of nickel and zinc can give rise to a significant shift in the fluorescence intensity of the chelating dye. The

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approximately 12-hour half-life of this nucleus facilitated experimentation; the nuclear decay process does not disrupt coordination of the product ions, nor does it appear to damage the dye scaffold, which retains its primary absorption features throughout. Energy and charge transfer are put forward as the most likely mechanisms for the Cu-induced fluorescence quenching. The authors envision multiple molecular imaging applications. — JSY


CANCER

Fat Feeds Tumors

Weight-loss campaigns emphasize the impact of obesity on the risk of heart disease and diabetes, but in reality the situation may be even grimmer. Emerging evidence suggests that obesity also increases the risk of developing common cancers such as breast and colorectal cancer and may be associated with a poorer prognosis if cancer occurs. The biological mechanisms by which obesity affects tumorigenesis are unclear, although research has centered on the concept that adipose tissue (fat) serves as a source of hormones, growth factors, and cytokines that promote tumor cell growth or invasiveness.

Zhang et al. provide evidence for an intriguing alternative in which adipose-derived cells rather than signaling molecules play a key role. Using mouse tumor models, they show that stromal and endothelial cells derived from white adipose tissue (WAT) are recruited by tumors to help build the blood vessels required for tumor expansion. Mobilization of these cells (green) into the circulation and their engraftment into the tumor stroma and vasculature (red) were associated with an increase in tumor growth and progression in the mice. Whether WAT-derived cells function similarly in human tumors remains to be explored. — PAK

Cancer Res. 69, 5259 (2009).

CHEMISTRY

Concentrating on Etching

The etching of nanometer-scale features on substrates is usually achieved with polymeric masks patterned by ultraviolet lithography, with resolution tied to the irradiation wavelength. Liu et al. report an alternative approach, whereby carbon nanotubes can act as catalysts for etching trenches in silicon dioxide 4 to 6 nm deep and about 60 nm in width. They first grew carbon nanotubes on silicon via chemical vapor deposition, with diameters of 1 to 4 nm. Immersion in basic solution led to etching rates that were about three times faster in the vicinity of the carbon nanotubes than elsewhere on the substrate, an effect which the authors attribute to the greater concentration of hydroxide groups that adsorb on the hydrophobic surface of the nanotubes in the electrical double layer. Atomic force microscopy revealed that most of the carbon nanotubes on the surface were released into solution after etching. — PDS


CANCER

Partners in Repression

Chromosomal translocations are structural abnormalities caused by the breaking and aberrant joining of regions from two or more chromosomes. If portions of two genes become fused, the fusion gene may encode a protein that combines two functions and produces a deleterious outcome. In some diseases, a single gene can be linked to one of several partners: Whereas the common gene dictates a basic similarity in disease etiology, the partner’s identity may define a distinctive mechanism of causation.

In patients with acute promyelocytic leukemia (APL), the retinoic acid receptor alpha (RARA) is expressed as a fusion protein with one of five different partners, such as PML or PLZF. Although most APL patients express RARA-PML and can be treated with all-trans retinoic acid, those carrying RARA-PLZF are resistant. Boukarabila et al. show that both fusion proteins inappropriately recruit the same Polycomb-repressive complex (PRC2) to mediate the silencing of retinoic acid–responsive genes. For RARA-PML, repression is reversed on treatment with retinoic acid; in contrast, RARA-PLZF also recruits PRC1, which contributes to tumorigenesis but is resistant to retinoic acid. Understanding the precise mechanisms of action of fusion proteins is important for the development of effective therapies. — HP

Genes Dev. 23, 1195 (2009).