GENETICS, Vol. 196, APRIL 2014, Copyright © 2014

The extracellular matrix protein artichoke is required for integrity of ciliated mechanosensory and chemosensory organs in *Drosophila* embryos, pp. 1091–1102

Marta Andrés, Enrique Turiégano, Martin C. Göpfert, Inmaculada Canal, and Laura Torroja

Sensory cilia are often surrounded by an extracellular matrix that is thought to be directly involved in mechanotransduction. This study identifies an evolutionarily conserved protein in *Drosophila*, Artichoke, that contributes to cilium assembly but is not produced by ciliated cells. In *Drosophila*, accessory cells secrete Artichoke into the extracellular matrix that surrounds the cilium. Loss of Artichoke impairs cilium organization, causing locomotion and chemosensation deficits. This represents the first experimental evidence that extracellular matrix is crucial not only for mechanotransduction but for chemosensation.

Discovery of supernumerary *B* chromosomes in *Drosophila melanogaster*, pp. 1007–1016

Elisabeth Bauerly, Stacie E. Hughes, Dana R. Vietti, Danny E. Miller, William McDowell, and R. Scott Hawley

Individuals in many eukaryotic species carry unnecessary extra (supernumerary, or "B") chromosomes. This study identifies a lab strain of *Drosophila melanogaster* that has acquired an average of 10 B chromosomes per fly in the space of a decade. These mitotically unstable B chromosomes appear to be derived from one of the standard chromosomes. The discovery of B chromosomes in a genetically tractable model provides a new tool for understanding how cells cope with extra chromosomes and how chromosomes evolve.

The fates of mutant lineages and the distribution of fitness effects of beneficial mutations in laboratory budding yeast populations, pp. 1217–1226

Evgeni M. Frenkel, Benjamin H. Good, and Michael M. Desai

The fate of a beneficial mutation depends on both luck and merit. Even when carried by many individuals, a beneficial mutation may be driven to extinction by competition from other, fitter mutations spreading through the population. The authors examine this process in lab-evolved yeast populations to understand which mutations can decisively overcome this competition and how often they occur. This study illuminates the fates of mutations in adapting microbial populations. Because these fates are shaped by competition from other mutations arising during the experiment, this data allows the authors to infer the distribution of fitness effects of competing beneficial mutations.

Fast and efficient estimation of individual ancestry coefficients, pp. 973–983

Eric Frichot, François Mathieu, Théo Trouillon, Guillaume Bouchard, and Olivier François

In population studies, genomic data is often used to infer the proportions of an individual genome that originate from multiple ancestral pools. Individual ancestry coefficients are commonly calculated using likelihood methods implemented in the computer programs STRUCTURE or ADMIXTURE. This article describes a fast and efficient alternative that has run-times 10-30 times shorter than ADMIXTURE as well as improved accuracy for inbred species.

Faster-X adaptive protein evolution in house mice, pp. 1131–1143

Athanasios Kousathanas, Daniel L. Halligan, and Peter D. Keightley

and

Disproportionate roles for the X chromosome and proteins in adaptive evolution, pp. 931–935

Bret A. Payseur

The X-chromosome plays an outsized role in speciation. One possible explanation is that recessive mutations are more exposed to selection if they are X-linked, leading to faster adaptive evolution. This is known as the "faster-X" hypothesis. The authors of this article found that X-linked genes indeed have a faster average rate of adaptive substitution than autosomal genes and this effect is more pronounced for male-specific genes. This could mean that new advantageous mutations are usually recessive, or that genes expressed during spermatogenesis evolve faster when on the X chromosome than when on autosomes due to genetic conflict. See also Commentary in this issue by Bret Payseur.

Enhancing the power to detect low-frequency variants in genome-wide screens, pp. 1293–1302

Chang-Yun Lin, Guan Xing, Hung-Chih Ku, Robert C. Elston, and Chao Xing

Conventional genome-wide association statistics lack the power to detect associations for low-frequency variants. Such statistics are analogous to one type of linkage disequilibrium measure, r^2. These investigators propose a test statistic analogous to a different type of linkage disequilibrium measure, D', and show the new test is more powerful than conventional methods.

Neandertal admixture in Eurasia confirmed by maximum likelihood analysis of three genomes, pp. 1241–1251

Konrad Lohse and Laurent A. F. Frantz

Neandertals and non-African modern humans share patterns of genetic variation that have been interpreted as a signature of interbreeding. However, this pattern could also be due to population sub-structure that pre-dated the Human-Neandertal divergence. To distinguish these two alternatives, the authors developed a likelihood method that uses the distribution of genealogical histories to estimate all relevant parameters from three genomes. The results reject ancestral population structure in Africa and strongly favor Neandertal admixture in Eurasia. This new method will be particularly useful for analyzing extinct or rare species with limited samples.

Loss of *Caenorhabditis elegans* BRCA1 promotes genome stability during replication in *smc-5* mutants, pp. 985–999

Stefanie Wolters, Maria A. Ermolaeva, Jeremy S. Bickel, Jaclyn M. Fingerhut, Jayshree Khanikar, Raymond C. Chan, and Björn Schumacher

Mutations in the BRCA1 gene lead to genome instability. This article shows that in *Caenorhabditis elegans*, loss of BRCA1 can also alleviate genome instability caused by defects in the structural maintenance of chromosomes complex SMC-5/6. These unexpected results suggest an explanation for the persistence of BRCA1 mutations in the human population, since loss of BRCA1 might be advantageous during replication stress.

This Month in the American Journal of Human Genetics

Antisense oligonucleotide-based therapy in human erythropoietic protoporphyria, Am. J. Hum. Genet. 94(4)

Vincent Oustric, Hana Manceau, Sarah Ducamp, Rima Soaid, Zoubida Karim, Caroline Schmitt, Arienne Mirmiran, Katell Peoc'h, Bernard Grandchamp, Carole Beaumont, Said Lyoumi, François Moreau- Gaudry, Véronique Guyonnet- Dupérat, Hubert de Verneuil, Joëlle Marie, Herve Puy, Jean-Charles Deybach, and Laurent Gouya

Many cases of erythropoietic protoporphyria (EPP) are caused by a rare variant in *FECH* that is inherited in trans with a common polymorphism. This polymorphism increases the use of a cryptic splice site leading to an mRNA isoform encoding a premature stop codon. Individuals affected by EPP have reduced FECH abundance and accumulate the protoporphyrin IX (PPIX). In this study, Oustric *et al.* targeted the cryptic splice site using an antisense oligonucleotide to prevent its use and demonstrate improved production of wild type *FECH* mRNA and reduced accumulation of PPIX in erythroblasts from EPP-affected individuals, suggesting that this might be a useful therapeutic approach for individuals with this combination of alleles.