GENETICS, Vol. 194, MAY 2013, Copyright © 2013

An organelle gatekeeper function for *Caenorhabditis elegans* UNC-16 (JIP3) at the axon initial segment, pp. 143–161

S. L. Edwards, S.-c. Yu, C. M. Hoover, B. C. Phillips, J. E. Richmond, and K. G. Miller

Nerve cell bodies have a vastly different organelle composition than axons. This article (see accompanying commentary by Zheng and Nonet, pp. 35–37) provides insight into the basis of this difference. The authors report the discovery of a previously unrecognized organelle gatekeeper function, mediated by UNC-16 (JIP3 in humans), that acts at the axon initial segment to restrict the flow of Golgi and endosomal organelles into the synaptic region of axons.

Novel proteins required for meiotic silencing by unpaired DNA and siRNA generation in *Neurospora crassa*, pp. 91–100

T. M. Hammond, H. Xiao, E. C. Boone, L. M. Decker, S. A. Lee, T. D. Perdue, P. J. Pukkila, and P. K. T. Shiu

and

Identification of small RNAs associated with meiotic silencing by unpaired DNA, pp. 279–284

T. M. Hammond, W. G. Spollen, L. M. Decker, S. M. Blake, G. K. Springer, and P. K. T. Shiu

Genes unpaired during meiosis are silenced in *Neurospora* by a mechanism known as meiotic silencing by unpaired DNA (MSUD). Two articles in this issue of *GENETICS* report the identification of novel players in this process, including small RNAs and the first nuclear MSUD protein. This protein is not required for meiosis, providing the first indication that MSUD is not necessarily coupled to sexual development.

Intragenomic conflict between the two major knob repeats of maize, pp. 81–89

L. B. Kanizay, P. S. Albert, J. A. Birchler, and R. K. Dawe

Large genomes are often replete with tandem repeats. Why do they exist? Here the authors investigate the distribution of tandem repeats in maize heterochromatic domains called knobs. The data suggest an intragenomic conflict whereby one family of repeats suppresses proliferation of the other. Similar competition may underlie the formation and maintenance of many tandem repeat arrays.

Nonrandom distribution of interhomolog recombination events induced by breakage of a dicentric chromosome in *Saccharomyces cerevisiae*, pp. 69–80

W. Song, M. Gawel, M. Dominska, P. W. Greenwell, E. Hazkani-Covo, K. Bloom, and T. D. Petes

This article presents the first high-resolution mapping of the positions of chromosome breaks that result from the bridge-fusion-breakage cycles of dicentric chromosomes. Sites of recombination between a dicentric chromosome and its normal homolog revealed the locations of breaks in the dicentric chromosome, which were distributed in a quasi-random fashion between the two centromeres.

Systems genetics of environmental response in the mature wheat embryo, pp. 265–277

J. D. Munkvold, D. Laudencia-Chingcuanco, and M. E. Sorrells

This article illustrates the utility of network approaches for understanding gene expression by environment interaction, even in organisms with highly complex genomes. A unique Weighted Gene Co-Expression Network Analysis approach was used to compare gene expression networks in mature wheat embryos from two distinct growing environments across a segregating population. This approach identified environmentally conserved and unique co-expression modules and their genetic control.

The innate immune response transcription factor Relish is necessary for neurodegeneration in a *Drosophila* Model of ataxia-telangiectasia, pp. 133–142

A. J. Petersen, R. J. Katzenberger, and D. A. Wassarman

Neurodegeneration is a hallmark of the human disease ataxia-telangiectasia (A-T), which is caused by mutation of the *A-T mutated* (*ATM*) gene. These investigators found that activation of an NF-κB-mediated innate immune response in glial cells causes neurodegeneration in *Drosophila ATM* mutants. Their finding that Relish, one of the three NF-κB proteins in flies, is necessary for neurodegeneration suggests that neurodegeneration in human A-T is caused by activation of a specific NF-κB protein in glial cells.

Identifying loci under selection against gene flow in isolation-with-migration models, pp. 211–233

V. M. Sousa, M. Carneiro, N. Ferrand, and J. Hey

When diverging populations hybridize, regions of the genome near genes associated with population differences or hybrid incompatibilities are prevented from moving from one population to the other. Thus some genes move more freely than others between diverging populations. This article describes a method for estimating rates of gene flow at multiple loci and for identifying loci with reduced gene flow. The authors apply the method to two subspecies of rabbits to show how gene flow can be restricted.

Higher levels of Neanderthal ancestry in East Asians than in Europeans, pp. 199–209

J. D. Wall, M. A. Yang, F. Jay, S. K. Kim, E. Y. Durand, L. S. Stevison, C. Gignoux, A. Woerner, M. F. Hammer, and M. Slatkin

Neanderthals occupied most of Europe and parts of Western Asia from 30–300 thousand years ago, and coexisted with modern humans during part of that time. These authors found that modern day East Asians contain on average more Neanderthal DNA than do modern day Europeans. They also show that the East African Maasai contain some Neanderthal ancestry, probably due to recent back-migration of modern humans into Africa.

Genetic dissection of a major anthocyanin QTL contributing to pollinator-mediated reproductive isolation between sister species of *Mimulus*, pp. 255–263

Y.-W. Yuan, J. M. Sagawa, R. C. Young, B. J. Christensen, and H. D. Bradshaw Jr.

What underlies prezygotic barriers that lead to reproductive isolation, and ultimately, to speciation? The bumblebee-pollinated *Mimulus lewisii* and hummingbird-pollinated *M. cardinalis* are a classic example of pollinator-mediated prezygotic reproductive isolation. These investigators identified an anthocyanin pigment regulatory gene causing a flower color difference that contributes to pollinator preference for each species. This gene is likely to play a role in flower color variation in many other plants.

The nuclear Argonaute NRDE-3 contributes to transitive RNAi in *Caenorhabditis elegans*, pp. 117–131

J. J. Zhuang, S. A. Banse, and C. P. Hunter

RNA interference (RNAi) was initially perceived as a cytoplasmic gene silencing process, but genes important for RNAi in *C. elegans* were found to operate in the nucleus. This article describes unexpected features of one such gene underlying a **n**uclear **R**NAi **d**efective (*nrde*) mutant, which emphasizes the importance of nuclear events during RNAi silencing, especially for transitive RNAi—a mechanism central to RNAi amplification.

This Month's Perspectives

Charles Darwin's mitochondria, pp. 21–25

J. Hayman

Scholars have long speculated over the nature of Charles Darwin's debilitating illness, with most diagnoses stressing psychogenic roots. In this Perspectives, John Hayman proposes that Darwin suffered from a mitochondrial genetic disease, MELAS syndrome, caused by a maternally inherited, relatively common A3243G point mutation in the mitochondrial chromosome. Here the hypothesis is examined by a review of Darwin's symptoms, in comparison to MELAS syndrome, and by an examination of related illnesses in his maternal family.

This Month in the American Journal of Human Genetics

Sherlock: detecting gene-disease associations by matching patterns of expression QTL and GWAS, Am. J. Hum. Genet. 92(5)

X. He, C. K. Fuller, Y. Song, Q. Meng, B. Zhang, X. Yang, and H. Li

With DNA sequence variants that alter gene expression, GWAS studies are largely limited to cis-acting variant-gene interactions. He *et al*. circumvent this limitation with a new method—Sherlock—that utilizes eQTL data to identify variants that contribute to altered gene expression. Using Sherlock, the authors identified cis- and trans-acting variants that contribute to Crohn's disease and Type 2 Diabetes.