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A novel statistical model to estimate host genetic effects affecting disease transmission, pp. 871–884

Osvaldo Anacleto, Luis Alberto Garcia-Cortés, Debby Lipschutz-Powell, John A. Woolliams, and Andrea B. Doeschl-Wilson

This article provides insight into how host genetic diversity affects disease spread, and offers novel means for disease control in livestock and predicting and controlling disease outbreaks in human populations. The authors introduce a novel statistical model for estimating genetic risks associated with the traits of host susceptibility to infection and heritable variation in disease transmission to susceptible individuals. They show that the model accurately estimates susceptibility and infectivity genetic parameters from incomplete time-to-infection data.

Inferring bottlenecks from genome-wide samples of short sequence blocks, pp. 1157–1169

Lynsey Bunnefeld, Laurent A. F. Frantz, and Konrad Lohse

These authors developed a novel way to facilitate likelihood-based population genomic inference by summarizing blocks of sequence data from small samples of individuals. They used a population bottleneck model to demonstrate the power of their method to detect past demographic events. The method is particularly useful for inference on non-model organisms, because long-range linkage and phase information is not required. It will also be applicable to a wide range of other models of population history.

Stimulation of chromosomal rearrangements by ribonucleotides, pp. 951–961

Hailey N. Conover, Scott A. Lujan, Mary J. Chapman, Deborah A. Cornelio, Rabab Sharif, Jessica S. Williams, Alan B. Clark, Francheska Camilo, Thomas A. Kunkel, and Juan Lucas Argueso

and

Elevated genome-wide instability in yeast mutants lacking RNase H activity, pp. 963–975

Karen O'Connell, Sue Jinks-Robertson, and Thomas D. Petes

RNA association with DNA can lead to genome instability. Conover *et al*. show that the recombinogenic effect of ribonucleotide incorporation during DNA replication is primarily caused by ribonucleotides incorporated by DNA polymerase ε, with only minor contributions from DNA polymerases α or δ. Their results extend previous studies demonstrating that the mutagenicity of ribonucleotides is asymmetric between the nascent leading and lagging DNA strands. O'Connell *et al*. present data that suggests that RNA:DNA hybrids (R-loops) formed during transcription are a more significant source of genetic instability than misincorporated ribonucleotides. It's no wonder that organisms have several RNAases for clearing their chromosomes of RNA.

Extent of QTL reuse during repeated phenotypic divergence of sympatric threespine stickleback, pp. 1189–1200

Gina L. Conte, Matthew E. Arnegard, Jacob Best, Yingguang Frank Chan, Felicity C. Jones, David M. Kingsley, Dolph Schluter, and Catherine L. Peichel

Independent populations repeatedly evolve similar traits when adapting to similar environments. To what extent is the genetic basis of repeated evolution repeatable? Conte *et al.* studied this question using two pairs of threespine stickleback species that have independently adapted to similar environments by evolving many similar traits. They found that nearly half the genomic regions associated with the evolution of similar traits are the same in both species pairs, suggesting that repeated genetic evolution is pervasive. Might further understanding of why evolution is repeatable render it highly predictable?

The genomic impacts of drift and selection for hybrid performance in maize, pp. 1201–1211

Justin P. Gerke, Jode W. Edwards, Katherine E. Guill, Jeffrey Ross-Ibarra, and Michael D. McMullen

Maize is naturally outcrossing, but modern breeding utilizes inbred lines in controlled crosses to produce hybrids. To understand the genomic impacts of hybrid breeding, Gerke *et al.* genotyped a pair of long-term experimental populations selected for hybrid performance. Their data suggest most of the observed loss of diversity is due to genetic drift. They also observed signals of selection in numerous regions of the genome. Their results point to simple complementation as the predominant mechanism of hybrid vigor.

Suppression of meiotic recombination by CENP-B homologs in *Schizosaccharomyces pombe*, pp. 897–904

Peter Johansen and Hugh P. Cam

Because homologous recombination in meiosis can lead to deleterious genome arrangements when it occurs at transposable elements, organisms have evolved mechanisms to suppress it. This article describes a pathway involving transposase-derived CENP-B proteins that recruit factors including Set1, NHEJ, and Condensin to suppress homologous recombination at Tf2 LTR retrotransposons in the fission yeast *Schizosaccharomyces pombe*.

Disruption of endocytosis with the dynamin mutant *shibire^{ts1}* suppresses seizures in *Drosophila*, pp. 1087–1102

Jason R. Kroll, Karen G. Wong, Faria M. Siddiqui, and Mark A. Tanouye

Many epileptics are unable to control their seizures with current anti-epileptic drugs. Using *Drosophila* as a model for seizure disorders, Kroll *et al.* find that the *shibire^{ts1}* mutation affecting Dynamin, which causes defects in synaptic vesicle recycling and endocytosis, is a powerful seizure suppressor. Interfering with synaptic vesicle regulation through Dynamin and various Rab GTPases also suppresses gain-of-function mutations in a gene homologous to human *SCN1A*, the most widely mutated gene in human epilepsy disorders. These results could point the way to the development of new classes of anti-epileptic drugs.

Asymmetric Wnt pathway signaling facilitates stem cell-like divisions via the non-receptor tyrosine kinase FRK-1 in *Caenorhabditis elegans*, pp. 1047–1060

Danielle Mila, Adriana Calderon, Austin T. Baldwin, Kelsey M. Moore, McLane Watson, Bryan T. Phillips, and Aaron P. Putzke

When a cell divides asymmetrically how is the development of the two daughter cells differently controlled? Mila *et al*. present a novel mechanism by which a non-receptor tyrosine kinase regulates asymmetric Wnt pathway signaling during stem cell divisions in *Caenorhabditis elegans*. Their findings reveal the complex nature of Wnt signaling in individual cells to achieve two daughter cells with different identities.

This Month in the American Journal of Human Genetics

Privacy risks from genomic data-sharing beacons, Am. J. Hum. Gent. 97(4)

Suyash S. Shringarpure and Carlos D. Bustamante

In an effort to promote the sharing of genomic data, the human genetics community has recently established a series of beacons. These web-based tools are able to provide information regarding the presence of particular alleles in a given cohort. While such beacons are safe from re-identification efforts that rely on allele frequency data, many beacons contain genomic data for a particular disease or disorder, suggesting that beacons could be a source of privacy leaks for both genetic and phenotypic data. In light of these concerns, Shringarpure and Bustamante use available tools to test whether re-identification is possible. Their results show, for example, that just 1,000 queries—a task easily completed by a single computer—can identify a member of the Personal Genome Project. Having uncovered this vulnerability, the authors propose a series of actions that can be taken to better protect the privacy of individuals who choose to participate in genomic studies.