GENETICS, Vol. 204, September 2016, Copyright © 2016

Bayesian inference of the evolution of a phenotype distribution on a phylogenetic tree, pp. 89–98

M. Azim Ansari and Xavier Didelot

Many traits are determined by a mixture of genetic and environmental factors. Ansari and Didelot model the evolution of the probability of a trait being observed, rather than the evolution of the trait itself. Reconstructing the history of this trait distribution reveals when and to what extent genetic factors affected the trait. A phylogenetic tree can therefore be divided into parts within which the trait probability is constant. The authors apply the method to real data from HIV immunology and bacterial ecology.

Remarkably long-tract gene conversion induced by fragile site instability in *Saccharomyces cerevisiae*, pp. 115–128

Shahana A. Chumki, Mikael K. Dunn, Thomas F. Coates, Jeanmarie D. Mishler, Ellen M. Younkin, and Anne M. Casper

Loss of heterozygosity (LOH) in human tumors is strongly correlated with break-prone chromosomal locations called common fragile sites. Gene conversion is one cause of LOH. Chumki *et al.* report that gene conversion is increased 48- to 62-fold by instability at the yeast fragile site FS2. The tract characteristics suggest a prominent mechanism for fragile site repair is break-induced replication that switches templates (dBIR). Thus, dBIR is likely to drive common fragile site-stimulated LOH in human tumors.

The genetic basis of natural variation in *Caenorhabditis elegans* telomere length, pp. 371–383

Daniel E. Cook, Stefan Zdraljevic, Robyn E. Tanny, Beomseok Seo, David D. Riccardi, Luke M. Noble, Matthew V. Rockman, Mark J. Alkema, Christian Braendle, Jan E. Kammenga, John Wang, Leonid Kruglyak, Marie-Anne Félix, Junho Lee, and Erik C. Andersen

Despite the importance of telomeres to genome stability, their length varies significantly between individuals. Cook *et al.* characterize natural variation in telomere lengths in *Caenorhabditis elegans* and map that variation to a polymorphism in a gene encoding a telomere-regulatory factor. They show that telomere length variation has little fitness consequence in the laboratory and find no evidence of selection at the regulatory locus in wild populations. These results suggest that telomere lengths beyond a basal level are not advantageous in wild populations.

Fast-flowering mini-maize: seed to seed in 60 days, pp. 35–42

Morgan E. McCaw, Jason G. Wallace, Patrice S. Albert, Edward S. Buckler, and James A. Birchler

McCaw *et al.* developed two *Zea mays* lines as a short-generation model for maize. Five generations per year of Fast-Flowering Mini-Maize (FFMM) can be achieved routinely with reduced greenhouse space, as opposed to two to three for traditional lines. Phenotypic kernel mutations have been introduced into one line to facilitate genetic analysis and demonstration of Mendelian principles in an educational setting. A whole genome resequence of one line, aligned to the B73 reference genome, is also reported.

Detecting heterogeneity in population structure across the genome in admixed populations, pp. 43–56

Caitlin McHugh, Lisa Brown, and Timothy A. Thornton

Ancestry patterns are often assumed to be similar across the genome. Previous studies have suggested that systematic ancestry differences in admixed populations may arise due to selection and/or sex-specific patterns of non-random mating. McHugh *et al.* propose the CAnD method for detecting heterogeneity in population structure genome-wide. Application of CAnD to HapMap Mexican American (MXL) samples provides strong evidence of heterogeneity in population structure (p=1e-05), largely driven by elevated Native American ancestry on the X chromosome as compared to the autosomes.

Histone deacetylases with antagonistic roles in *Saccharomyces cerevisiae* heterochromatin formation, pp. 177–190

Deborah M. Thurtle-Schmidt, Anne E. Dodson, and Jasper Rine

Saccharomyces cerevisiae silent mating loci have been a paradigm for studying heterochromatin formation, yet an interesting conundrum has plagued the Sir-silencing model. The only known role for Sir2 in silencing is to deacetylate H4K16, however preemptively blocking this acetylation does not restore silencing in Sir2 mutants. Thurtle-Schmidt *et al.* found that restoration of silencing depends on two deacetylases, Rpd3 and Hst3, independent of their known histone substrates, revealing unexpected and antagonistic roles for multiple deacetylases in Sir protein-based silencing.

Fine-scale human population structure in southern Africa reflects ecogeographic boundaries, pp. 303–314

Caitlin Uren, Minju Kim, Alicia R. Martin, Dean Bobo, Christopher R. Gignoux, Paul D. van Helden, Marlo Möller, Eileen G. Hoal, and Brenna M. Henn

The African KhoeSan populations have remained largely isolated until about 2,000 years ago. Though dozens of KhoeSan groups exist, very little is known about their population history. Uren *et al.* examine fine-scale population structure in southern Africa and find it does not always correspond to linguistic or subsistence categories as previously suggested, but rather reflects the role of geographic barriers and the ecology of the greater Kalahari Basin. The authors conclude that local adoption of pastoralism, at least by the Nama, appears to have been primarily a cultural process with limited genetic impact from eastern Africa.

Genetic architecture of domestication-related traits in maize, pp. 99–113

Shang Xue, Peter J. Bradbury, Terry Casstevens, and James B. Holland

Domestication radically transformed the morphology of maize compared to its wild ancestor, teosinte. Nevertheless, some of the traits presumed to be important under domestication retain variation in maize. Xue *et al.* tested if this variation is due to genes in regions known to differentiate maize and teosinte. They found no major effect loci remaining, but many small effect genes outside of the known domestication genome regions affect variation in domestication-related traits.

This Month in the American Journal of Human Genetics

The power of human protective modifiers: PLS3 and CORO1C unravel impaired endocytosis in spinal muscular atrophy and rescue SMA phenotype, Am. J. Hum. Genet. 99(3)

Seyyedmohsen Hosseinibarkooie, Miriam Peters, Laura Torres-Benito, Raphael H. Rastetter, Kristina Hupperich, Andrea Hoffmann, Natalia Mendoza-Ferreira, Anna Kaczmarek, Eva Janzen, Janine Milbradt, Tobias Lamkemeyer, Frank Rigo, C. Frank Bennett, Christoph Guschlbauer, Ansgar Büschges, Matthias Hammerschmidt, Markus Riessland, Min Jeong Kye, Christoph S. Clemen, and Brunhilde Wirth

Spinal muscular atrophy (SMA), a motor neuron disease typified by progressive loss of voluntary muscle function, is caused by homozygous mutations in *SMN1*. Previous work revealed upregulation of *PLS3* expression in asymptomatic individuals who harbor *SMN1* deletions. Now, Wirth and colleagues demonstrate that a combination of transgenic *PLS3* overexpression and administration of SMN-antisense oliognucleotides can reverse the expected course of disease in a mouse model of SMA. Further work revealed that a role for PLS3 and its binding partner, CORO1C, in endocytic trafficking. Together, the authors' work provides new insights into the basic cellular pathways that are disrupted by *SMN1* loss of function and offers hope for the development of novel SMA treatment strategies.