

Diverse lineages of *Candida albicans* live on old oaks, pp. 277–288

Douda Bensasson, Jo Dicks, John M. Ludwig, Christopher J. Bond, Adam Elliston, Ian N. Roberts, and Stephen A. James

Most humans are inhabited by the yeast *Candida albicans* at some point. While largely harmless, it is the most common cause of yeast infections. Though previously unclear whether the yeast can live outside of warm-blooded animals, Bensasson *et al.* report that genetically diverse strains of *C. albicans* can live on oak trees for prolonged periods. The study of woodland *C. albicans* could lead to a better understanding of how virulent forms of yeast emerge.

Heterochromatin-enriched assemblies reveal the sequence and organization of the *Drosophila melanogaster* Y chromosome, pp. 333–348

Ching-Ho Chang and Amanda M. Larracuent

Heterochromatic repeat-rich regions are often missing from even the best genome assemblies. Chang and Larracuent designed a de novo assembly strategy to improve the *Drosophila melanogaster* assembly in heterochromatin, extending the reference assembly by 11.9 Mb, including 10.6 Mb from the Y chromosome. Their ~15 Mb Y chromosome assembly details the repeat landscape and gene organization. They observe high duplication rates both to and on the Y chromosome and high gene conversion rates in the *crystal-Stellate* family. This important genomic reference also provides insights into mutation patterns on an old *Drosophila* Y chromosome.

Reconstructing the history of polygenic scores using coalescent trees, pp. 235–262

Michael D. Edge and Graham Coop

As both GWAS and procedures for inferring gene genealogies progress, there will be major opportunities for learning about trait evolution using gene genealogies of trait-associated loci. Edge and Coop introduce statistical procedures for estimating the historical time course of a population-mean polygenic score and for testing that time course for evidence of natural selection.

Pervasive positive and negative feedback regulation of insulin-like signaling in *Caenorhabditis elegans*, pp. 349–362

Rebecca E. W. Kaplan, Colin S. Maxwell, Nicole Kurhanewicz Codd, and L. Ryan Baugh

The *Caenorhabditis elegans* genome encodes 40 insulin-like peptides, but the dynamics of insulin signaling both during development and in response to nutrient availability is not well understood. Kaplan and Maxwell *et al.* report that transcription of the majority of insulin genes is regulated by insulin signaling itself, suggesting extensive positive and negative feedback control. They show that the insulin peptide *daf-28* promotes its own expression through positive feedback and that *ins-6* cross-regulates *daf-28* expression via feedback. Their results suggest that feedback regulation produces an organismal FoxO-to-FoxO signaling network that supports homeostasis during fluctuations in nutrient availability.

Female sex development and reproductive duct formation depend on Wnt4a in zebrafish, pp. 219–233

Michelle E. Kossack, Samantha K. High, Rachel E. Hopton, Yi-lin Yan, John H. Postlethwait, and Bruce W. Draper

Wnt4 is a key regulator of ovary development in mammals, but its role in other vertebrates is unknown. Here, Kossack *et al.* show that zebrafish *wnt4a* is the ortholog of mammalian *Wnt4* and is expressed in zebrafish somatic gonad cells during the time sex

determination likely occurs. Through analysis of *wnt4a* mutants, they show that Wnt4a promotes female sex determination and the development of male and female reproductive ducts, concluding that Wnt4/Wnt4a is likely a conserved regulator of ovarian and reproductive duct development in all vertebrates.

Local PCA shows how the effect of population structure differs along the genome, pp. 289–304

Han Li and Peter Ralph

Principal component analysis (PCA) is often used to describe overall population structure—patterns of relatedness arising from past demographic history—among a set of genomes. Here, Li and Ralph describe how the patterns uncovered by PCA differ at a local scale along the genome. They find that these patterns can be driven strongly by chromosomal inversions (and possibly also regions of low recombination), with correlations suggesting that the strong remaining heterogeneity may be caused by the past action of linked selection.

Evolutionary quantitative genetics of genomic imprinting, pp. 75–88

Eleanor K. O'Brien and Jason B. Wolf

Genomic imprinting creates a difference in how maternal and paternal gene copies contribute to quantitative genetic variation and evolutionary change. To fully understand these impacts, O'Brien and Wolf develop a definitive extension to the classic quantitative genetics framework. They find that imprinting creates a difference in heritable variation through mothers and fathers that alters their contributions to evolution and contributes transitory variation—but does not directly contribute to evolution since it is erased each generation when imprints are reset. Recognizing the impacts of imprinting can improve our understanding of resemblance of relatives and evolutionary change.

The role of maternal HP1a in early *Drosophila* embryogenesis via regulation of maternal transcript production, pp. 201–217

Ah Rume Park, Na Liu, Nils Neuenkirchen, Qiaozhi Guo, and Haifan Lin

Heterochromatin protein1a (HP1a) is a highly conserved epigenetic factor that associates with heterochromatin in gene silencing. Though it can activate transcription, the function of HP1a in development has been under-investigated. Here, Park and Liu *et al.* report the role of maternal HP1a in producing transcripts that drive early *Drosophila* embryogenesis. Maternal HP1a upregulates genes involved in translation, mRNA splicing, and cell division but down-regulates genes involved in neurogenesis, organogenesis, and germline development, which all occur later in development.

This Month's Perspectives**Selective sweeps**

Wolfgang Stephan

The study of natural selection through linked neutral variation has become an important concept in evolutionary genetics, because the patterns of neutral variation can be used to infer where selective events (whose genomic location is usually unknown) have occurred along the genome. This may then lead to the identification and characterization of genes that are important in adaptation. This paper describes the models and inference methods that have been developed to detect selective sweeps and localize the targets of selection in the genome. Selective sweeps are caused by strong directional selection driving beneficial alleles to fixation.