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Comparative oligo-FISH mapping: an efficient and powerful methodology to reveal karyotypic and chromosomal evolution, pp. 513–523

Guilherme T. Braz, Li He, Hainan Zhao, Tao Zhang, Kassandra Semrau, Jean-Marie Rouillard, Giovana A. Torres, and Jiming Jiang

Development of a eukaryotic karyotype relies on identification of individual chromosomes in the species, which has been accomplished only in a limited number of plant and animal species. Here, Braz *et al*. report a novel chromosome identification system using potato as a model. A set of oligos selected from the potato genome generated 26 distinct fluorescence in situ hybridization (FISH) signals collectively used as a 'barcode' to uniquely label the 12 potato chromosomes. The barcode was then used to study karyotypic and chromosomal evolution of several species distantly related to potato.

A failsafe for sensing chromatid tension in mitosis with the histone H3 tail in *Saccharomyces cerevisiae*, pp. 565–578

Christopher J. Buehl, Xiexiong Deng, Jianjun Luo, Visarut Buranasudja, Tony Hazbun, and Min-Hao Kuo

The yeast protein Shugoshin 1 (Sgo1p) senses tension between sister chromatids during mitotic chromosome alignment. A regulatory region on histone H3—the tensor sensing motif—is responsible for retaining Sgo1p at the pericentromere and is negatively regulated by the acetyltransferase Gcn5p. Here, Buehl *et al*. interrogate the mechanism of this regulation, revealing a novel pathway for the mitotic recruitment of Sgo1p and demonstrating a novel role for the tail of histone H3 in mitotic regulation.

Tissue-specific gene inactivation in *Xenopus laevis*: knockout of lhx1 in the kidney with CRISPR/Cas9, pp. 673–686

Bridget D. DeLay, Mark E. Corkins, Hannah L. Hanania, Matthew Salanga, Jian Min Deng, Norihiro Sudou, Masanori Taira, Marko Horb, and Rachel K. Miller

Xenopus laevis is a classic developmental model, but its allotetraploid genome has limited our ability to perform genetic manipulations. The advance of CRISPR/Cas9 gene editing makes genetic knockouts more feasible, however. Here, DeLay *et al*. demonstrate that CRISPR efficiently knocks out both homeologs in F0 generation embryos using a single sgRNA and that editing can be targeted to a tissue of interest. These findings establish *Xenopus* as an efficient, cost-effective model for studying the genetics of developmental processes, making this aquatic tetrapod amenable to high throughput gene and drug screens.

Keeping pace with the Red Queen: identifying the genetic basis of susceptibility to infectious disease, pp. 779–789

Ailene MacPherson, Sarah P. Otto, and Scott L. Nuismer

The results of genome-wide association studies are known to be affected by epistasis and gene-by-environment interactions. Using a statistical model, MacPherson, Otto, and Nuismer explore the effect of between species gene-by-gene interactions on the ability to detect genes underlying susceptibility to infectious disease. They propose that a more complex study design, which incorporates both host and pathogen genetics, is required to uncover the basis of disease susceptibility. Using previously published data on *Daphnia magna* susceptibility to *Pasteuria ramosa*, they illustrate that comparisons between genome-wide association studies with and without gene-by-gene interactions can inform us about the genetic architecture of susceptibility.

Detection of epistasis for flowering time using Bayesian multilocus estimation in a barley MAGIC population, pp. 525–536

Boby Mathew, Jens Léon, Wiebke Sannemann, and Mikko J. Sillanpää

Flowering time is a well-known complex trait in crops and is influenced by many interacting genes. In this study, Mathew *et al*. identify two-way and three-way epistasis for the flowering time trait in a Multi-Parent Advanced Generation Inter-Cross (MAGIC) barley population by using dimension reduction combined with the Bayesian multilocus model. Many of the identified genomic regions have been previously reported for the flowering time trait in barley and closely related species, thus illustrating the potential of genome-wide mapping of higher-dimensional epistasis in MAGIC populations.

Relaxed selection during a recent human expansion, pp. 763–777

Stephan Peischl, I. Dupanloup, A. Foucal, M. Jomphe, V. Bruat, J.-C. Grenier, A. Gouy, K. J. Gilbert, E. Gbeha, L. Bosshard, E. Hip-Ki, M. Agbessi, A. Hodgkinson, H. Vézina, P. Awadalla, and Laurent Excoffier

Peischl *et al*. explore the way evolutionary forces shape genetic variability in expanding human populations. Over a few generations of separate evolution, front individuals present fewer but more deleterious mutations than core individuals. Furthermore, harmful mutations are segregating at higher frequencies in front individuals, leading to an increased risk for genetic diseases in recently colonized areas. These results provide evidence for differential strength of natural selection in humans and show that range expansions are affecting the evolution of deleterious variants in modern human populations.

A common pathway of root growth control and response to CLE peptides through two receptor kinases in *Arabidopsis*, pp. 687–704

Adriana Racolta, Michael D. Nodine, Kelli Davies, Cameron Lee, Scott Rowe, Yulemi Velazco, Rachel Wellington, and Frans E. Tax

Racolta *et al*. show two different impacts of CLE peptide treatment of *Arabidopsis* roots. In all genotypes tested, they find increased proliferative activity in the stem cell niche and a delay in differentiation of daughter cells. In genotypes that are sensitive to the application of CLE peptide, there is a reduction of cell divisions within the longitudinal cell files of differentiating cells, resulting in short roots. They provide genetic evidence that the receptor kinases RPK1 and TOAD2/RPK2 function together to control the root responses to CLE peptides.

Compensatory internalization of Pma1 in V-ATPase mutants in *Saccharomyces cerevisiae* requires calcium- and glucose-sensitive phosphatases, pp. 655–672

Swetha Devi Velivela and Patricia Marie Kane

Loss of V-ATPase activity in organelles triggers compensatory endocytic downregulation of the plasma membrane proton pump Pma1. Here, Velivela and Kane report that mutations in calcineurin or glucose-sensitive phosphatase Glc7-Reg1 exhibit negative synthetic genetic interactions with *vma* mutations, compromising Pma1 endocytosis. Loss of V-ATPase activity triggers internalization of approximately half of surface Pma1, but a comparable reduction in Pma1 expression in a *pma1-007* mutant neither compensates for loss of V-ATPase activity nor limits further Pma1 endocytosis, suggesting that loss of V-ATPase activity may transmit cytosolic calcium, glucose, or pH signals, but that residual Pma1 levels are not sensed.