ISSUE HIGHLIGHTS

Transmission dynamics of heritable silencing induced by double-stranded RNA in Caenorhabditis elegans, pp. 1275–1288
Rosa M. Alcazar, Ruyeling Lin and Andrew Z. Fire

Gene silencing can persist for generations. In Caenorhabditis elegans the effects of RNAi usually recede after the F1 generation, but there are examples of it persisting to the F2 generation and beyond. The authors use a pedigree-based assay to track RNAi-induced gene silencing, revealing that in early generations it is transmitted independently of the original targeted locus, like a diffusible epigenetic element.

A genomewide survey argues that every zygotic gene product is dispensable for the initiation of somatic homolog pairing in Drosophila, pp. 1329–1342
Jack R. Bateman and C-ting Wu

In 1908, Nettie Maria Stevens made a remarkable discovery: maternal and paternal homologous chromosomes are intimately paired in virtually all cells in flies. A century later, it is clear that chromosomes physically interact in diverse cell types throughout the living world, but it remains unclear how homologous chromosomes find each other and align. The authors systematically survey the genome of Drosophila melanogaster for genes essential for chromosome pairing during embryogenesis. Remarkably, no zygotic gene product is required. Thus, the answer to the century-old riddle of how homologous chromosomes pair remains shrouded in the maternal genome.

Convergent evolution in the genetic basis of Müllerian mimicry in Heliconius butterflies, pp. 1567–1577

The mimetic butterflies Heliconius melpomene and H. erato display colorful wing patterns to warn off predators. Their phenotypes have converged in local populations, yet differ dramatically between geographic regions. These authors show that red wing patterns are controlled by homologous chromosomal loci in both species. Subtle phenotypic differences in wing patterns between the two species imply that while they evolved independently by somewhat different developmental routes, they are regulated by the same genetic switch locus.

Components of the RNAi machinery that mediate long-distance chromosomal associations are dispensable for meiotic and early somatic homolog pairing in Drosophila melanogaster, pp. 1355–1365
Justin P. Blumenstiel, Roxana Fu, William E. Theurkauf and R. Scott Hawley

The manner by which homologous chromosomes pair with one another in meiosis is poorly understood. One possibility: the mechanism that mediates dispersed chromosomal interactions is the same one that mediates homolog pairing. Indeed, recent studies have indicated that the RNAi machinery mediates several long-distance chromosomal interactions. While the authors did identify a novel function for in meiotic progression, these investigators found that the RNAi machinery is not necessary for homolog pairing in Drosophila, although it did identify a novel function for it in meiotic progression.

Use of a Drosophila model to identify genes regulating Plasmodium growth in the mosquito, pp. 1671–1678
Stephanie M. Brandt, Giovanna Jaramillo-Gutierrez, Sanjeev Kumar, Carolina Barillas-Mury and David S. Schneider

Mosquitoes carry infectious diseases that kill millions of people each year. The mosquito is not a passive host; it actively fights these infections. Because genetic analysis of mosquitoes is difficult, these authors use the fruit fly to identify candidate genes involved in pathogen growth in mosquitoes.

Selective sweep at a quantitative trait locus in the presence of background genetic variation, pp. 1645–1660
Luís-Miguel Chevin and F одéric Hospital

What can we learn about the genetics of adaptation from molecular signatures of selection? How does selection on polygenic traits translate into selection on genes? The authors investigate the dynamics of selection on a gene affecting a quantitative trait with background genetic variance. Background variance can strongly influence the temporal trajectory of a beneficial mutation, depending on the form of selection. Under similar strengths of selection, molecular signatures of selection are stronger for traits with lower background genetic variance, and not selected for an optimum. Phenotypic traits exhibiting strong signatures of selection are thus a biased subset of all adaptive traits.

Waiting for two mutations: With applications to regulatory sequence evolution and the limits of Darwinian evolution, pp. 1501–1509
Rick Durrett and Deena Schmidt

This article shows that the rapid turnover of transcription factor binding sites in Drosophila can be explained by a population genetics result concerning the waiting time to observe two prespecified mutations. The authors also expose flaws in intelligent design advocate Michael Behe’s arguments about the limits of Darwinian evolution.

Quantitative trait loci mapping and the genetic basis of heterosis in maize and rice, pp. 1707–1724
Antonio Augusto Franco Garcia, Shengchu Wang, Albrecht E. Meichinger and Zhao-Bang Zeng

Heterosis, particularly in maize and rice, is largely responsible for the Green Revolution. By employing multiple interval mapping on two data sets, one for maize and one for rice, this article reveals that the genetic basis for heterosis in yield is quite different for maize and for rice. In maize, the evidence points to dominant quantitative trait loci as the main cause for the heterosis. In rice, epistasis appears to be the main reason. This distinction seems to be related to open or self pollination of the respective species.

A simple formula for obtaining markedly improved mutation rate estimates, pp. 1773–1778
Philip Gerrish

The formula derived in this article provides a significant improvement over the standard method for estimating mutation rates (the fluctuation test), with superior statistical methods of estimation. The amount of extra lab work required is negligible, yet the improvement in accuracy of the estimates is remarkable.

A novel septin-associated protein, Syp1p, is required for normal cell cycle-dependent septin cytoskeleton dynamics in yeast, pp. 1445–1457
Wenjie Qiu, Suat Peng Neo, Xianwen Yu and Mingjie Cai

The authors identify another regulator of septin dynamics in yeast. Syp1p colocalizes with septins and associates with several septin subunits. Loss of Syp1p delays septin assembly early in the cell cycle and its disassembly at the end of cell division; overexpression of Syp1p accelerates septin disassembly. This brings us closer to understanding the mechanism of yeast cell division.

Identification of selective sweeps in closely related populations of the house mouse based on microsatellite scans, pp. 1537–1545
Meike Teschke, Odette Mukabayire, Thomas Wiehe and Diether Tautz

Genome scans of DNA sequence polymorphisms can provide insights into the pattern and prevalence of positive selection under natural conditions. Microsatellite markers offer the potential to focus on very recent events. The authors assess this concept in a large-scale study of natural populations of the house mouse. The average frequency of selective sweeps in the populations was higher than 1 in 100 generations, exceeding previous estimates and implying that natural selection acts frequently in natural populations.

The DNA end-binding protein Ku regulates silencing at the internal HML and HIM loci in Saccharomyces cerevisiae, pp. 1407–1418
Catherine L. Vandre, Rohinton T. Kamakaka and David H. Rivier

The DNA end-binding protein Ku regulates several diverse processes associated with DNA ends. This article describes a study of one Ku-dependent process—silencing of genes adjacent to telomeres—and makes a surprising discovery: Ku contributes not only to telomeric silencing, but also to silencing at internal silent loci not near DNA ends. Recruitment of Ku to these internal sites is dependent on the Sir4 silencing protein. Hence, Ku is endowed with two modes of DNA binding: direct binding to DNA ends and recruitment to internal regions via protein–protein interactions.

Role of recombination in the long-term retention of transposable elements in rRNA gene loci, pp. 1617–1626
Xian Zhang, Michael T. Eckbush and Thomas H. Eckbush

In most insects, the tRNA-specific retrotransposons, R1 and R2, inactivate a large fraction of the tRNA units. The simulation model of the tRNA locus described in this article offers the remarkable suggestion that high rates of retrotransposition are likely to be accommodated by minor adjustments in the sister chromatid exchange rate, rather than by direct selection on the number of functional tRNA units. This model explains why most eukaryotes encode two to three times more tRNA units than are needed for the synthesis of tRNA and why R1 and R2 elements can be so remarkably successful.