Estimating the number of ancestral lineages using a maximum-likelihood method based on rejection sampling, pp. 1741–1757

Michael G. B. Blum and Noah A. Rosenberg

Did our ancestors mate with Neanderthals? Using an algorithm for simulating genealogical trees conditional on a fixed number of lineages at a given time in the past, the authors estimate the number of ancestral human lineages at the time of potential admixture with Neanderthals. Making use of mitochondrial DNA data, they conclude that the portion of the modern human genome resulting from Neanderthal admixture is likely to be <5%.

Beneficial mutation–selection balance and the effect of linkage on positive selection, pp. 1759–1798

Michael M. Desai and Daniel S. Fisher

Multiple beneficial mutations segregating in a population can compete and interfere if they are linked. This "traffic" makes the dynamics of adaptation in large populations look very different from the simple picture of sequential selective sweeps. This article presents a framework for analyzing this accumulation of linked beneficial mutations and provides a new approach for studying the dynamics of adaptation in large asexual populations.

A complex genetic interaction between *Arabidopsis thaliana* TOC1 and CCA1/LHY in driving the circadian clock and in output regulation, pp. 1501–1510

Zhaojun Ding, Mark R. Doyle, Richard M. Amasino and Seth J. Davis

The CCA1, LHY, and TOC1 proteins are thought to make up the metronome of the *Arabidopsis thaliana* circadian clock, which drives the plant's rhythms, including seasonal control of flowering and photomorphogenesis. The authors dissect this oscillator and find that TOC1 regulates the floral transition through CCA1 and LHY, while CCA1 and LHY function upstream of TOC1 to regulate a photomorphogenic process.

A Markov chain Monte Carlo approach for joint inference of population structure and inbreeding rates from multilocus genotype data, pp. 1635–1651

Hong Gao, Scott Williamson and Carlos D. Bustamante

Excess homozygosity is a signature of nonrandom mating. It can result from selfing, inbreeding, assortative mating, and/or undetected population stratification. This article develops an extension of a popular Bayesian clustering approach to distinguish selfing/simultaneous inference of inbreeding rates and population-of-origin classification using multilocus genotypes.

The evolution of condition-dependent sex in the face of high costs, pp. 1713–1727

Lilach Hadany and Sarah P. Otto

Many organisms have sex only when growth conditions are unfavorable. Alleles that mediate this can invade an asexual population even when sex has a high cost. Such facultative sexual alleles spread in the population because they escape from low fitness genotypes and become associated with genotypes of higher fitness, providing a satisfying model for the evolution and maintenance of facultative sex.

Functional analysis of maize RAD51 in meiosis and double-strand break repair, pp. 1469–1482

Jin Li, Lisa C. Harper, Inna Golubovskaya, C. Rachel Wang, David Weber, Robert B. Meeley, John McElver, Ben Bowen, W. Zacheus Cande and Patrick S. Schnable

This article reports that RAD51 plays an important role in meiotic chromosome pairing and synapsis in maize. The absence of RAD51 results in defects in chromosome pairing and synapsis and increased exchange between nonhomologous chromosomes. Thus, RAD51 functions to increase the precision of homologous chromosome pairing.

A sensitized PiggyBac-based screen for regulators of border cell migration in Drosophila, pp. 1579–1590

Juliette Mathieu, Hsin-Ho Sung, Céline Pugieux, Jan Soetaert and Pernille Rorth

Migration of border cells during *Drosophila melanogaster* oogenesis is a good model for cell migration *in vivo*. The authors identify genes required for border cell migration. One of them, *puckered*, encodes a negative regulator of the Jun kinase (JNK) signaling pathway. It is unusual in that defects are observed only when signaling from the receptor tyrosine kinase PVR is reduced. Further results suggest that JNK may counteract PVR signaling by antagonizing the caspase inhibitor DIAP1.

Ure2p function is enhanced by its prion domain in *Saccharomyces cerevisiae*, pp. 1557–1565

Frank Shewmaker, Lori Mull, Toru Nakayashiki, Daniel C. Masison and Reed B. Wickner

The Ure2 protein of *Saccharomyces cerevisiae* regulates gene expression in response to nitrogen availability. It can also become a prion via its N-terminal asparagine-rich domain. The prion domain was thought to be unnecessary for Ure2 function, but the authors of this article show that Ure2p function is compromised by truncation of the prion domain, which may be involved in interaction with other components of the nitrogen regulation system.