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A PUF Hub drives self-renewal in *Caenorhabditis elegans* germline stem cells, pp. 147–161

Kimberly A. Haupt, Kimberley T. Law, Amy L. Enright, Charlotte R. Kanzler, Heaji Shin, Marvin Wickens, and Judith Kimble

The *Caenorhabditis elegans* network for germline stem cells (GSCs) was missing key intrinsic regulators of self-renewal. Haupt *et al*. report that these missing regulators are PUF RNA-binding proteins, PUF-3 and PUF-11, which together with FBF-1 and FBF-2, account for the effects of niche signaling on self-renewal. This discovery underscores the principle role of PUF RNA-binding proteins in stem cell maintenance and places them as central to a regulatory "PUF hub" that drives GSC self-renewal. This PUF hub, composed of four PUF proteins and two PUF partners, constitutes the intrinsic self-renewal node of the *C. elegans* GSC regulatory network.

The driver of extreme human-specific Olduvai repeat expansion remains highly active in the human genome, pp. 179–191

Ilea E. Heft, Yulia Mostovoy, Michal Levy-Sakin, Walfred Ma, Aaron J. Stevens, Steven Pastor, Jennifer McCaffrey, Dario Boffelli, David I. Martin, Ming Xiao, Martin A. Kennedy, Pui-Yan Kwok, and James M. Sikela

Olduvai (formerly DUF1220) protein domains have undergone the largest human-specific increase in copy number of any coding region in the genome (~300 copies of which 165 are human-specific) and have been implicated in human brain evolution. By analyzing the human genome reference sequence (hg38) and single molecule optical mapping data of 186 individuals, Heft *et al*. show that the genomic mechanism that allowed this dramatic evolutionary copy number increase is also responsible for the extreme Olduvai variability found among present day human populations. In other words, their findings indicate the same process that may have been a key contributor to the expansion of the human brain remains highly active in present human populations.

An activating mutation in ERK causes hyperplastic tumors in a *scribble* mutant tissue in *Drosophila*, pp. 109–120

Tatyana Kushnir, Shaked Bar-Cohen, Navit Mooshayef, Rotem Lange, Allan Bar-Sinai, Helit Rozen, Adi Salzberg, David Engelberg, and Ze'ev Paroush

Excessive RTK signaling, often caused by activating mutations in Ras, Raf and/or MEK, occurs in most human tumors. Intriguingly, confirmed cancer-driver mutations in the downstream effector kinase, ERK, have not been reported. To test if active ERK mutants can function as oncoproteins, Kushnir *et al*. introduced an activating mutation, originally identified in a yeast ERK, into the single *Drosophila* ERK. The authors find that this mutation renders ERK catalytically active independently of upstream signaling, and that its expression induces extensive over-proliferation and hyperplastic tumor formation *in vivo*. Thus, some human ERK1/2 mutations identified in patient-derived tumors may actually represent overlooked oncogenic, cancer-causing mutations.

Rapid and predictable evolution of admixed populations between two *Drosophila* species pairs, pp. 211–230

Daniel R. Matute, Aaron A. Comeault, Eric Earley, Antonio Serrato-Capuchina, David Peede, Anaïs Monroy-Eklund, Wen Huang, Corbin D. Jones, Trudy F. C. Mackay, and Jerry A. Coyne

In this article, Matute *et al*. report an experiment in which they generated eight interspecific admixed populations using two species pairs of *Drosophila*. They found that in both species pairs, and across all experimental replicates, all phenotypes (morphology, behavior, and fertility) rapidly regressed to those of the parental continental species, becoming indistinguishable from that species. Consistent with this observation, the genomes of the admixed populations also regressed to the continental species with only some traces of the island species. Their results show that the evolutionary outcome of hybridization can be highly repeatable and predictable at least in hybridizing species of *Drosophila*.