Selection on bristle length has the ability to drive the evolution of male abdominal appendages in the sepsid fly *Themira biloba*. Herath B1, Dochtermann NA1, Johnson JI1, Leonard Z1, Bowsher JH1.

Many exaggerated and novel traits are strongly influenced by sexual selection. Although sexual selection is a powerful evolutionary force, underlying genetic interactions can constrain evolutionary outcomes. The relative strength of selection vs. constraint has been a matter of debate for the evolution of male abdominal appendages in sepsid flies. These abdominal appendages are involved in courtship and mating, but their function has not been directly tested. We performed mate choice experiments to determine whether sexual selection acts on abdominal appendages in the sepsid *Themira biloba*. We tested whether appendage bristle length influenced successful insemination by surgically trimming the bristles. Females paired with males that had shortened bristles laid only unfertilized eggs, indicating that long bristles are necessary for successful insemination. We also tested whether the evolution of bristle length was constrained by phenotypic correlations with other traits. Analyses of phenotypic covariation indicated that bristle length was highly correlated with other abdominal appendage traits, but was not correlated with abdominal sternite size. Thus, abdominal appendages are not exaggerated traits like many sexual ornaments, but vary independently from body size. At the same time, strong correlations between bristle length and appendage length suggest that selection on bristle length is likely to result in a correlated increase in appendage length. Bristle length is under sexual selection in *T. biloba* and has the potential to evolve independently from abdomen size.
Drosophila evolution:

Speciation, the process by which new biological species arise, involves the evolution of reproductive barriers, such as hybrid sterility or inviability between populations. However, identifying hybrid incompatibility genes remains a key obstacle in understanding the molecular basis of reproductive isolation. We devised a genomic screen, which identified a cell cycle-regulation gene as the cause of male inviability in hybrids resulting from a cross between Drosophila melanogaster and D. simulans. Ablation of the D. simulans allele of this gene is sufficient to rescue the adult viability of hybrid males. This dominantly acting cell cycle regulator causes mitotic arrest and, thereby, inviability of male hybrid larvae. Our genomic method provides a facile means to accelerate the identification of hybrid incompatibility genes in other model and nonmodel systems.

Hybrid male rescue (Hmr) on the D. melanogaster X-chromosome
Lethal hybrid rescue (Lhr) on the D. simulans second chromosome
Third factor: Suppressor of Killer-of-prune [Su(Kpn)]–glutathione-S-transferase–containing FLYWCH zinc finger protein (gfzf): DNA damage–induced G2-M cell cycle–checkpoint mechanism to block cell proliferation

Mutagenized 55,000 D. simulans males by feeding adults with ethyl methane sulfonate (EMS) and crossed these males to D. melanogaster females.
The decline in fitness with inbreeding: evidence for negative dominance-by-dominance epistasis in *Drosophila melanogaster*.

Sharp NP1,2, Agrawal AF1.

Tested for dominance-by-dominance epistasis in Drosophila melanogaster by examining viability at five inbreeding levels that were generated simultaneously, avoiding the bias against detecting non-linearity that has affected previous studies. We find an accelerating rate of fitness decline with inbreeding, indicating that dominance-by-dominance epistasis is negative on average, which should favour sex and recombination.

Increases in the evolutionary potential of upper thermal limits under warmer temperatures in two rainforest Drosophila species.

van Heerwaarden B1, Malmberg M1,2, Sgro CM1.

Comparative developmental analysis of Drosophila and Tribolium reveals conserved and diverged roles of abrupt in insect wing evolution.

Ravisankar P1, Lai YT1, Sambrani N1, Tomoyasu Y2.

RNAi screening for candidate genes has identified *abrupt* (*ab*) as a potential key player in elytron evolution. In this study, we performed a series of RNA interference (RNAi) experiments in both Tribolium and Drosophila to understand the contributions of *ab* to the evolution of beetle elytra. We found that (i) *ab* is essential for proper wing vein patterning both in Tribolium and Drosophila, (ii) *ab* has gained a novel function in determining the unique elytron shape in the beetle lineage.
During asymmetric division, fate determinants at the cell cortex segregate unequally into the two daughter cells. It has recently been shown that Sara (Smad anchor for receptor activation) signalling endosomes in the cytoplasm also segregate asymmetrically during asymmetric division. Biased dispatch of Sara endosomes mediates asymmetric Notch/Delta signalling during the asymmetric division of sensory organ precursors in Drosophila. In flies, this has been generalized to stem cells in the gut and the central nervous system, and, in zebrafish, to neural precursors of the spinal cord. However, the mechanism of asymmetric endosome segregation is not understood. Here we show that the plus-end kinesin motor Klp98A targets Sara endosomes to the central spindle, where they move bidirectionally on an antiparallel array of microtubules. The microtubule depolymerizing kinesin Klp10A and its antagonist Patronin generate central spindle asymmetry. This asymmetric spindle, in turn, polarizes endosome motility, ultimately causing asymmetric endosome dispatch into one daughter cell. We demonstrate this mechanism by inverting the polarity of the central spindle by polar targeting of Patronin using nanobodies (single-domain antibodies). This spindle inversion targets the endosomes to the wrong cell. Our data uncover the molecular and physical mechanism by which organelles localized away from the cellular cortex can be dispatched asymmetrically during asymmetric division.
Polarized endosome dynamics by spindle asymmetry during asymmetric cell division.
Asymmetry in genitalia does not increase the rate of their evolution.
Eberle J1, Walbaum W1, Warnock RC2, Fabrizi S3, Ahrens D4.

Left-right asymmetry is a frequently encountered phenomenon in the copulation organs of insects. While various causes have been proposed for genital asymmetry, we raise the question of whether asymmetry might facilitate, or even accelerate, morphological divergence of genitalia between species. We tested this hypothesis in the scarab chafer genus Schizonycha, which comprises species with symmetric as well as asymmetric male genitalia. Morphometric analyses were conducted in the context of their phylogeny, inferred from mitochondrial and nuclear ribosomal DNA sequence data (cox1, rrnL, and 28S) for a sample of 99 South African specimens, including 34 species and 5 outgroup taxa. Trees were reconstructed with maximum likelihood and Bayesian analysis. The extent of asymmetry and the variation of male copulation organs were analyzed with Generalized Procrustes analysis (GPA), by quantifying shape divergence of the parameres. We found a continuous transition in the degree of asymmetry among the investigated species. Ancestral state reconstruction revealed multiple origins and a high degree of evolutionary plasticity of paramere asymmetry in Schizonycha. However, no significant correlation between evolutionary rates of paramere shape divergence and the degree of paramere asymmetry was found, and so we conclude that asymmetric genitalia in Schizonycha do not increase the rate of genital shape divergence.
Asymmetry:
Left-right asymmetric cell intercalation drives directional collective cell movement in epithelial morphogenesis.
Sato K1, Hiraiwa T1, Maekawa E2, Isomura A2, Shibata T1, Kuranaga E2,3,4,5.

Morphogenetic epithelial movement occurs during embryogenesis and drives complex tissue formation. However, how epithelial cells coordinate their unidirectional movement while maintaining epithelial integrity is unclear. Here we propose a novel mechanism for collective epithelial cell movement based on Drosophila genitalia rotation, in which epithelial tissue rotates clockwise around the genitalia. We found that this cell movement occurs autonomously and requires myosin II. The moving cells exhibit repeated left-right-biased junction remodelling, while maintaining adhesion with their neighbours, in association with a polarized myosin II distribution. Reducing myosinID, known to cause counter-clockwise epithelial-tissue movement, reverses the myosin II distribution. Numerical simulations revealed that a left-right asymmetry in cell intercalation is sufficient to induce unidirectional cellular movement. The cellular movement direction is also associated with planar cell-shape chirality. These findings support a model in which left-right asymmetric cell intercalation within an epithelial sheet drives collective cellular movement in the same direction.
Drosophila development:

Development. 2015 Dec 23. pii: dev.129163. [Epub ahead of print]

Transcriptomes of lineage-specific Drosophila neuroblasts profiled via genetic targeting and robotic sorting.
Yang CP1, Fu CC2, Sugino K1, Liu Z1, Ren Q1, Liu LY1, Yao X1, Lee LP3, Lee T4.

A brain consists of numerous distinct neurons arising from a limited number of progenitors, called neuroblasts in Drosophila. Each neuroblast produces a specific neuronal lineage. To unravel the transcriptional networks that underlie the development of distinct neuroblast lineages, we marked and isolated lineage-specific neuroblasts for RNA sequencing. We labeled particular neuroblasts throughout neurogenesis by activating a conditional neuroblast driver in specific lineages using various intersection strategies. The targeted neuroblasts were efficiently recovered using a custom-built device for robotic single-cell picking. Transcriptome analysis of the mushroom body, antennal lobe, and type II neuroblasts compared to non-selective neuroblasts, neurons, and glia revealed a rich repertoire of transcription factors expressed among neuroblasts in diverse patterns. Besides transcription factors that are likely pan-neuroblast, there exist many transcription factors that are selectively enriched or repressed in certain neuroblasts. The unique combinations of transcription factors present in different neuroblasts may govern the diverse lineage-specific neuron fates.
WHAMY is a novel actin polymerase promoting myoblast fusion, macrophage cell motility and sensory organ development.

Brinkmann K1, Winterhoff M2, Önel SF3, Schultz J4, Faix J2, Bogdan S5.

Wiskott-Aldrich syndrome proteins (WASP) are nucleation promoting factors (NPF) that differentially control the Arp2/3 complex. In Drosophila, three different family members, SCAR/WAVE, WASP and WASH, have been analyzed so far. Here, we characterize WHAMY, the fourth Drosophila WASP family member. whamy originated from a wasp gene duplication and underwent a sub-neofunctionalization. Unlike WASP, WHAMY specifically interacts with activated Rac1 through its two CRIB domains that are sufficient for targeting WHAMY to lamellipodial and filopodial tips. Biochemical analyses showed that WHAMY promotes exceptionally fast actin filament elongation, while it does not activate the Arp2/3 complex. Loss- and gain-of function studies revealed an important function of WHAMY in membrane protrusions and cell migration in macrophages. Genetic data further imply synergistic functions between WHAMY and WASP during morphogenesis. Double mutants are late-embryonic lethal and show severe defects in myoblast fusion. Trans-heterozygous mutant animals show strongly increased defects in sensory cell fate specification. Thus, WHAMY is a novel actin polymerase with an initial partitioning of ancestral WASP functions in development and subsequent acquisition of a new function in cell motility during evolution.
Epigenetic information is widely appreciated for its role in gene regulation in eukaryotic organisms. However, epigenetic information can also influence genome evolution. Here, we investigate the effects of epigenetic information on gene sequence evolution in two disparate insects - the fly *Drosophila melanogaster*, which lacks substantial DNA methylation, and the ant *Camponotus floridanus*, which possesses a functional DNA methylation system. We found that DNA methylation was positively correlated with the synonymous substitution rate in *C. floridanus*, suggesting a key effect of DNA methylation on patterns of gene evolution. However, our data suggest the link between DNA methylation and elevated rates of synonymous substitution was, in large part, explained by the targeting of DNA methylation to genes with signatures of transcriptionally-active chromatin, rather than the mutational effect of DNA methylation itself. This result highlights the importance of chromatin structure as the primary epigenetic driver of genome evolution in insects. This phenomenon may be explained by an elevated mutation rate for genes residing in transcriptionally active chromatin, or by increased structural constraint on genes in inactive chromatin. Overall, our study highlights how different epigenetic systems contribute to variation in the rates of coding sequence evolution.
A CRISPR-Cas9 gene drive system targeting female reproduction in the malaria mosquito vector *Anopheles gambiae*.

Hammond A1, Galizi R1, Kyrou K1, Simoni A1, Siniscalchi C2, Katsanos D1, Gribble M1, Baker D3, Marois E4, Russell S3, Burt A1, Windbichler N1, Crisanti A1, Nolan T1.

They identified three genes (AGAP005958, AGAP011377 and AGAP007280) that confer a recessive female-sterility phenotype upon disruption, and inserted into each locus CRISPR-Cas9 gene drive constructs designed to target and edit each gene. For each targeted locus we observed a strong gene drive at the molecular level, with transmission rates to progeny of 91.4 to 99.6%.

CRISPR/Cas9-mediated mutagenesis of the white and Sex lethal loci in the invasive pest, *Drosophila suzukii*.

Li F1, Scott MJ2.

They used the CRISPR/Cas9 system to introduce site-specific mutations in the *D. suzukii white* (w) and *Sex lethal* (Sxl) genes. Hemizygous males with w mutations develop white eyes and the mutant genes are transmissible to the next generation. **Somatic mosaic females that carry mutations in the Sxl gene develop abnormal genitalia and reproductive tissue.** The *D. suzukii* Sxl gene could be an excellent target for a Cas9-mediated gene drive to suppress populations of this highly destructive pest.