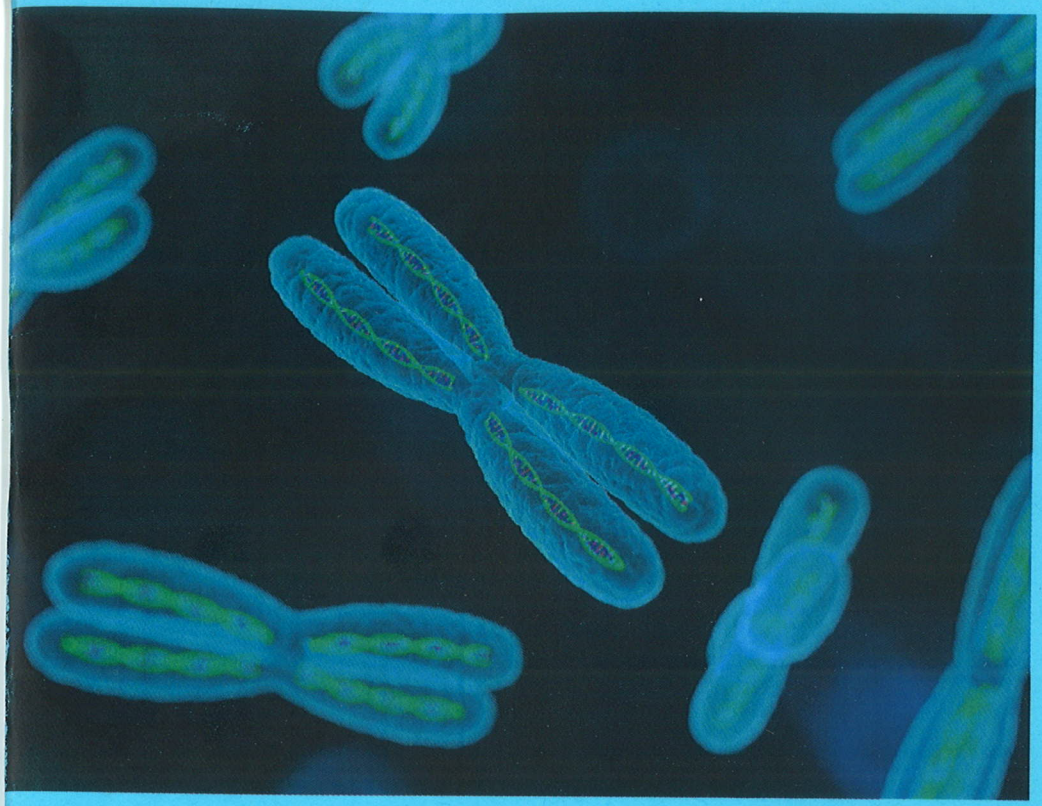


MPGI FALL SYMPOSIUM

Genomics of Conflict

CARGILL 105

AUGUST 24, 2017

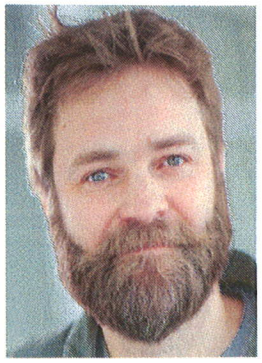
Schedule of Events

8:30 - 9:15	Breakfast
9:15 - 9:30	**Yaniv Brandvain, PMB** "A tour of genomic conflicts"
9:30 - 10:00	**Nels Elde, University of Utah** "Evolutionary innovations from biological collisions between microbes and their hosts"
10:00 - 10:30	**Sarah Anderson, PMB** "Analysis of transposon expression insights into TE-genome conflicts in maize"
10:30 - 11:00	Coffee
11:00 - 11:30	**Erin Kelleher, University of Houston** "Host Tolerance of invading transposable elements"
11:30 - 12:00	**Amanda Larracuente, University of Rochester** "Meiotic drive and sattelite DNA in Drosophila melanogaster"
12:00 - 1:00	Lunch
1:00 - 1:30	**Jennifer Mandel, University of Memphis** "Plant organellar genomes: heteroplasmy and its evolutionary consequences"
1:30 - 2:00	**Will Driscoll, EEB, BTI** "Social conflict during the experimental evolution of multicellularity"
2:00 - 2:15	Coffee
2:15 - 2:30	**Will Harcombe, EEB, BTI** "Systems biology and eco-evolutionary feedbacks in microbial communities"
2:30 - 3:30	**Harmit Malik, Fred Hutchinson Cancer Research Center** "Genetic conflicts shape meiosis and species"
3:30 - 4:30	Reception

Yaniv Brandvain, PMB

"A tour of genomic conflicts"

Succeeding in the social world is a balancing act. On the one hand the opportunity to work together with other genes, individuals, species provides the opportunity to do things differently and more efficiently. On the other hand, such interactions lead parties open to exploitation and deceit. We explore the genomics of conflict across plants, animals and microbes, and explore how such conflicts (and their resolution) can shape genome structure, gene expression, and basic features of life.



Nels Elde, University of Utah

Evolutionary innovations from biological collisions between microbes and their hosts

Biological collisions, for example virus infection of hosts, influence the fate of populations. These associations whether transient or seemingly permanent can provoke genetic conflicts of recurring, counteracting adaptations crucial for cell functions at the interfaces. I will discuss our work probing variation in primates and other mammals, which is revealing the far-reaching impact of selfish genetic elements on the origins and regulation of immune functions.

Sarah Anderson, PMB

"Analysis of transposon expression provides insights into TE-genome conflicts in maize"

Transposable elements (TEs) comprise a large portion of many eukaryotic genomes and are unique in their ability to replicate and move within genomes. The size of the maize genome is largely inflated by TE insertions, with the majority of these sequences derived from LTR retrotransposons, which require transcription for movement. RNA-directed DNA Methylation (RdDM), one mechanism plants have evolved to silence transposons to prevent movement, also relies on transcription to produce small RNAs which target DNA methylation machinery to TE sequences. Despite the abundance of RNA-seq datasets published in maize, TE expression has been under-assessed due to challenges associated with annotating, mapping, and assigning reads to the repetitive sequences common in TEs. We have utilized the new structural annotation of maize TEs in combination with a novel read assignment approach that accounts for these repetitive sequences to assess TE expression on a per-family basis across development and in mutants with subtle defects in DNA methylation. We find substantial variation in the proportion of the transcriptome derived from TEs across development, and in the specific families with varied expression across development and in mutants. Furthermore, similarities between TE families expressed in RdDM mutants and in nutritive endosperm support a role for interactions between RdDM and certain TE families during seed development.

Erin Kelleher, University of Houston

Host tolerance of invading transposable elements

Transposable elements (TEs) are obligate genetic parasites that propagate in host genomes by replicating in germline nuclei, thereby ensuring representation in offspring genomes. This selfish replication not only produces deleterious insertions: in extreme cases, TE mobilization induces germline genome instability that prohibits the development of viable gametes. To avoid these fitness costs, animal and plant genomes are known to repress germline TE activity through small-RNA-mediated silencing. However, it remains unknown whether host genomes can evolve tolerance of TEs, by desensitizing gametogenesis to TE activity. In part, this absence of research on tolerance reflects a lack of opportunity, as small-RNA- mediated repression masks variation in tolerance and evolves rapidly after a new TE invades.

We use the recent invasion of the Drosophila melanogaster genome by P- element DNA transposons in order to study tolerance of TE activity in the absence of repression. A GWAS on a panel of 1600 recombinant inbred lines that lack small-RNA- mediated silencing of P-elements, uncovered two major QTL that determine the tolerance of oogenesis to P-element mobilization. The first QTL harbors a well-studied regulator of oogenesis, suggesting that differences in the timing of gamete development could confer robustness to TE activity. By contrast, the second locus spans a centromere, and is explained by differential abundance of the pericentromeric satellite repeat Responder. Repetitive DNA could contribute to host tolerance of TE activity by determining sensitivity to DNA damage. Our results reveal that individuals differ in their response to germline TEs, and that these differences may reflect a range of developmental and genomic factors.

Amanda Larracuente,
University of Rochester

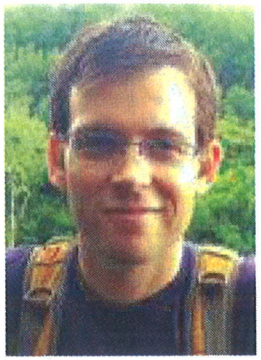
Meiotic drive and satellite DNA in Drosophila melanogaster

Conflicts arise within genomes when genetic elements are selfish and fail to play by the rules. Meiotic drivers are selfish genetic elements found across a wide variety of taxa that cheat meiosis to bias their transmission to the next generation. We are interested in the evolution of drive systems, their mechanisms of cheating meiosis, and impacts on genome evolution. Our model system is an autosomal male meiotic driver found on the 2nd chromosome of Drosophila melanogaster called Segregation Distorter (SD). Males heterozygous for SD and sensitive wild type chromosomes transmit SD to >95% of their progeny, whereas female heterozygotes transmit SD fairly, to 50% of their progeny. SD is a sperm killer that targets large blocks of tandem satellite repeats (called Responder) for destruction through an unknown mechanism. We will discuss the impact of SD on the regulation and evolution of the Responder satellite, and how this may lend insight into mechanisms of meiotic drive.

Jennifer Mandel,
University of Memphis

Plant organellar genomes: heteroplasmy and its evolutionary consequences

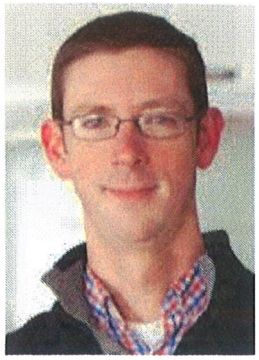
The occurrence of within individual organellar genomic variation, i.e., heteroplasmy, has been described and studied since at least the 1970s. However, recent studies have revealed that heteroplasmy is more common and widespread than once thought. In particular, advances in sequencing technologies now allow for the study of entire organellar genomes within individuals as compared to one or a few genes. The occurrence of within individual genetic variation raises profound questions relating to evolutionary processes: can individuals (as collective populations of organellar genomes) evolve? How sexual are organellar genomes? Does heteroplasmy influence cytonuclear interactions? Is there genetic conflict among genomes within individuals? I will provide an overview of our recent work in this area using the natural plant system, wild carrot, for both plastid and mitochondrial genomes including population genetic and cytonuclear linkage disequilibrium consequences of heteroplasmy. Finally, I will demonstrate a prototype of a software that enables visualization of heteroplasmy candidates at the genome-wide level.



Will Driscoll, BTI, EEB

Social conflict during the experimental evolution of multicellularity

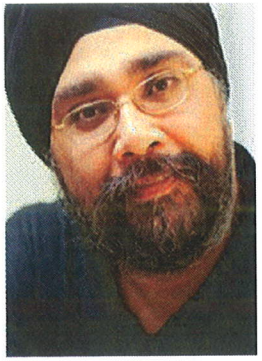
Multicellular life requires extensive inter-cellular cooperation, yet competition within multicellular groups favors 'selfish' lineages that forego cooperation. This tradeoff gives rise to a conflict between the interests of individual cells and multicellular collectives, which is widely viewed as a barrier to the evolution of multicellular life. Current models implicate selection to minimize such conflicts to explain pervasive features of modern multicellular organisms, including development from a single-celled propagule. However, direct tests of such hypotheses are impossible due to the ancient origins of modern multicellular lineages such as plants and animals. We employ experimental evolution to study the transition to multicellularity from a single-celled ancestor in the yeast Kluyveromyces lactis. We find evidence that contrasting selection at the levels of individual cells and multicellular clusters maintains both selfish unicellular and cooperative multicellular genotypes for > 300 generations in all ten replicate populations. Interestingly, complex life cycles involving phenotypic switching between unicellular and multicellular life stages evolved independently in 2/10 experimental populations, providing an opportunity to directly test hypotheses for the origin and evolution of this pervasive feature of multicellular organisms.



Will Harcombe, BTI, EEB

Systems biology and eco-evolutionary feedbacks in microbial communities

Conflict and cooperation between bacterial species drive the composition and function of microbial communities. Stability of these emergent properties will be influenced by the degree to which species' interactions are robust to genetic perturbations. We use genome-scale metabolic modeling to computationally analyze the impact of genetic changes when Escherichia coli and Salmonella enterica compete, or cooperate. We systematically knocked out in silico each reaction in the metabolic network of E. coli to construct all 2,583 mutant stoichiometric models. Then, using a recently developed multi-scale computational framework, we simulated the growth of each mutant E. coli in monoculture and in the presence of S. enterica. The type of interaction between the species was set by modulating the initial metabolites present in the environment. We found that both the species ratios and community productivity are most robust to genetic perturbation when the two species cooperate. Additionally, the number of mutations that have a substantial effect is lower when the species cooperate than when they are competing. These results highlight the utility of connecting metabolic mechanisms and studies of ecological stability. Cooperation and conflict alter the connection between genetic changes and properties that emerge at higher levels of biological organization.



Harmit Singh Malik
Fred Hutchinson Cancer Research Center

"Genetic conflicts shape meiosis and species"

Selfishness pervades biology. Nowhere is the impact of selfish genes felt more strongly than during or in the aftermath of meiosis in many organisms. Using case studies in fission yeast and Drosophila, I will present some of our latest and ongoing work on how selfish elements shape fundamental aspects of meiosis, and may even provide the initial schism to reproductively isolate species.