Intro themes, major points

-mlh1 patterns (cell wide)

-(chromosome pairs) – bivalent patterns

-what is the patterns of variation in cell av counts across house mouse species complex? (what role does evo divergence and sex play?)

-what’s the pattern of variation in the single bivalent patterns (which leads to gwRR)? (which hold information on CO interference , chromatin compaction and …. )

**General mlh1 pattern / description (cells level) and Estimates of gwRR from (House mouse species complex)**

(-gwRR +1 for the PAR in males)

-number of cells, mice, strains, subsp

-quality control metrics? – measures of error, **repeatability**

-comparison to previously reported measures; males (PWD/PWK, WSB, G?), female (WSB) (Beth’s data)

-novel measures (most female measures ), new Musc inbred strains, Molossinus, (outgroups?)

- running a lm across all the male gwRR – results in the PWD, MSM, and SKIVE having significant effects. So males of these 3 strains will be designated ‘high rec’ group

*- Two male gwRR have rapidly evolved, 30% (MSM is novel) and these rates are sex specific (not in the females of the strain). SKIVE male is intermediate gwRR – the female rate is also elevate (low rate of heterochiasmy)*

-Overall pattern of variation for this scale / branch lengths – (greatest divergence for short evo divergence)

-degree of hetC is pretty low

(-reversal of hetc, uncommon between closely related species)

- g female different in gwrr (not in ratios / proportions)

**Analysis using a Mixed model framework for examining pattern of heterochiasmy**

-Logic for the Model choices (multiple models ), sex effect, subsp, interaction, and random effects

Also ran post hoc fixed effect models

-gwRR / mouse averages for full data set – and a higher quality dataset (to make sure the overall patterns were similar)

-lack of support for uniform/ consistent divergence in the trait – lack of support for neutral evolution

-Mixed model 2 (now 1), sex and interaction effects with subsp were significant () AND the random strain effect is significant. Variance due to strain effect is not 0.

1. *Predictions for ‘uniform’ patterns across all Mus musculus strains are not met; (ie. different subspecies didn’t diverge )*
2. *Sex is a significant effect, but not in a uniform manner. (significant as a interaction)*

Post hoc tests and models match the qualitative patterns; most divergent categories; G females, PWD males and MSM males.

-sex specific polymorphism in Musc –(X Mol, only 2 strains) (range of male levels – do I have more solid support that musc females don’t mirror the males…)

**Within Mouse variance for MLH1 counts**

-Brief background on within mouse variance in CO (cite RWang, KVeller)

*-all strains / data sets show that females have more within mouse variance for MLH1 counts per cell.*

**(weak-ish supporting evidence for) CO precursors correlate / indicate correlation with gwRR (in males)**

-subset of male samples (juvinille for DMC1 staining)

-L cells have more DMC1 than Z

-the higher Musc strain (PWD ) – has more than WSB

**gwRR variation translates to the chromosome level**

-(range is 1-3) – like many species (Stapley)

-higher rec strains/categories – have more 2CO bivalents

- rapid evolved males (MSM and PWD) also have different bivalent class proportions (1:1 1CO:2CO vs 2:1 1CO : 2CO )

-Do SKIVE have intermediate proportion? (apply chi sq test or something)

-*this is motivation to investigate more traits at the chromosome level*

**Bivalent Level Results**

-review motivation / driving questions

1. sex specific patterns

2. (focus on male specific variation in gwRR) (dom < low.musc , low.mol < PWD MSM

Make predictions with the following bivalent level metrics;

1. SC Length,
2. normalized 1CO position
3. IFD / interference

1**.Sex specific patterns and predictions (background / intro)**

-outline the predictions and the relevant background

-SC length and 1CO / telomere bias are supported in the lit

- sex specific comparisons of IFD / interference in the physical scale are very rare – only De Boer et al 2006 (1 classical lab strain) --- but genetic map defined interference is well established

chromosome level aspects of Heterochiasmy

* SC length and the normalized 1CO position are conserved chrm level traits of heterochiasmy in house mouse
* Some IFD patterns are Sexually dimorphic, --
* (which lines of evidence support – unreg vs reg patterns in male and female bivalent patterns

**Q1. SC Length differences**

Dom – no differences (not met)

-for almost all models, sex is the only significant effect. \_\_ except in comparison of the Musc strains—where the SKIVE strain effect is also significant – because both male and female bivalents are shorter compared to PWD and KAZ.

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**Q1. normalized 1CO position**

-Norm F1 has significant sex effect (haven’t run the )

**Q1. IFD / interference**

Run on ‘long biv’ dataset

-there are sex differences in the IFDs (which strain has biggest ones)

**Q2. SC Length differences**

-when all chrm classes are pooled, the general pattern is that the higher rec strains have longer bivalent SC lengths.

Glm( SC ~ strain )

-all strains are significantly different ()

- The mean.pooled.SC lengths can significantly predict if the mouse is high or low group.

When bivalents are subsetted by chrm class the patterns are a bit nuanced – but fit an overall pattern of SC lengths having more clustered / less overlapping ranges in high rec strains. Higher rec 1COs are shorter than lower rec 1COs.

*Shaper clustering of SC lengths across chromosome classes in the high Rec males*

**Q2. normalized 1CO position**

-high rec strains have more central normalized F1 pos

**Q2. IFD / interference**

-High Rec strains have long IFD