**intro**

We performed hypothesis testing of models from the literature and adapted here fit the main results from this study. We choose to examine three modifier models and two functional and cell physiology models (table X). It is a bit challenging to test all of these models together since they’ve been built with difference parameters and built to describe different patterns of recombination variation. The modifier models were built to explain heterochiasmy variation between sexes and the COM functional model was built to describe the interference pattern and difference in the recombination landscape between the sexes. In cases where the models can’t be extended to predicting results, we list an NA.

**Reversed heterochiasmy**

The common direction of heterochiasmy is female biased, however male biased heterochiasmy has (significant number of observations of male biased heterochiasmy (cite hetC papers). Our results are novel, because the pattern is indicative of rapid male specific evolution.

**models**

The prediction from two of the modifier models, **haploid selection and SACE** are that males will generally evolve lower recombination rates overall compared to females. The **two locus modifier model** does parameterize cases of male biased heterochiasmy depending on the stage of action for the driver and the linkage status (see table 1 Brandvain and Coop). There are no prediction for reversal of heterochiasmy direction under the **COM** (pairing model, but it should be noted that this model wasn’t designed to incorporate this type of data.

Under the **spindle based model** faster male specific evolution of the genome wide recombination rate is primarily driven by differences in the strength of the SAC.

SAC (Lane Kauppi, )

 leading to different evolutionary trajectories for the genome wide recombination rate.

The SAC ‘read’s that all tetrads have tension -- (each set of centromeres have sinlge and opposite pole connection (synthellic?) and that the sister cohesion connecting the homologs -- sets up the tension -- opposes the pull from each of the poles.

Efficient normal SAC -- works by pausing entry into anaphase or triggers apoptosis -- if a unpaired / tension-lacking tetrad is detected

(sperm SAC is finer tuned (can detect 1-2 tetrads lacking tension -- ), while the female SAC  (has a higher threshold for being triggered (4-5?)

(relaxed selection - / inefficient SAC -- in females -- fits with higher rate of achiasmata bivalents (there might be alternative mechanisms facilitating achiasmate segregation)

 Mechanisms for sex differences in the strength of the SAC are the cell volume (diffusion of signal molecules) and the centrosome being present at the spindle have been proposed in the literature.

(testing / proving the mechanism for the new optima is for the high recombining group in males is out of the scope of this paper, but we propose some hypothesis are suggested below.

**Greater between cell variance**

Our results of greater between cell variation in number of crossovers have previously been observed in human and mouse (Gruhn, Kong, Zwel -- Cole).

While the **modifier models** parametrize sexual dimorphism in the strength of selection which can shape the variance in a trait these types of models make predictions for variance between individuals but not within individuals where the genetics are constant.

In the **spindle based selection model**, --

the source of between cell variance is attributed to difference strength of checkpoints. (Sexual dimorphism in the stringency of the spindle assembly checkpoint is a well-established result in the literature. Most empirical results come from mammals yet, some of the hypotheses for the mechanism of inefficient SAC in oocytes are attributed to acentrosomal spindles (cite) and cell volume (cite), which are conserved sexual dimorphic features of gametogenesis (cite).

(leaky SAC would lead to more between cell variance for crossover number) – across oocytes

The COM model doesn’t make predictions for between cell variation.

**TYPICAL landscape**

Our results on the broad scale pattern of crossover placement are generally supported by the literature. As reviewed in (Sardel Kirkpatrick), sexual dirmorohism in the broad scale landscape is a highly conserved trait.  However there are exceptions, (marsupial, bird?)

**the popgen models**

The two locus modifier model and SACE model make predictions for (sexual dimorphism ) in the broad scale recombination landscape, for diminishing the effect of drive systems and maintaining larger chromosome blocks respectively.

**functional models**

The two mechanical models also both predict sexual dimorphism in the recombination landscape for distinct reasons. The chromosome pairing based model (COM), predicts the sex differences in the positioning of crossovers is due to a combination of the length of the axis and intensity of the RPMs.  The spindle based model proposed that sexual dimorphism in the recombination landscape is driven by difference strength in the spindle assembly checkpoint (Lane Kauppi). The relaxed selection imposed by an ineffective SAC results in a uniform distribution of positions (while maintaining positive interference). Whereas,  in males, the directional selection could cause decreased variance in the recombination landscape via telomeric positioning of crossovers. Under the telomeric bias recombination landscape, entry into anaphase may be more synchronizes (and faster) given that the reductional separation of homologs is dependent on a minimal amount of sister cohesion connecting the homologs (Figure X).

**Sexual dimorphism in chromatin structure**

The results of sexual dimorphism in chromatin structure is supported by the literature, especially for mammals (Lynn, Gruhn, ).

**models**

Very few models make predictions for how chromatin organization relates to variation in recombination rates / the recombination landscape.  (the chromatin organization is how chromosomes with the same number of base pairs) are transformed into bottle brush structures with a central axis and most of the genetic material arranged as loops emanating outwards (figure).

Only the COM model states that chromatin structure will be longer in oocytes due to greater cell volume.  This prediction / model might fit broader pattern (such as in At where pollen is the larger cell and has longer axis length) (cahoon libuda).

Predictions for this trait are  inconclusive (due to lack of cytology measures from both sexes. We argue that the chromatin structure (and axis length) should be considered as an integral parameter when studying recombination landscape.  The sex differences in chromatin strucutre are closely connected to sex differences in the interference strength and our results mirror those in Petkov et al. (prediction, shorter-loop will have stronger interference).

**Evolved traits**, **Evolution of interference (hardest section)**

(interference is a fundamental aspect of the recombination landscape)

Some basics of the constraints bounds of interference are known, yet there are still many unknowns regarding the relationship of genome wide recombination rate and interference strength.  The non-random spacing of crossovers along the 2D length of chromosomes is the first level of quantification of crossover interference (cite gamma COI papers). Logically a negative correlation is expected; increasing the number of crossovers across chromosomes would most logically be done by  more densely spacing crossovers along chromosomes and decreasing interference strength. This pattern has empirical support from the most species (Otto and Payseur) and fits well with the fundamental relationship between the SC area or axis length, the physical upper limit for the number of crossovers.

Our results support an opposite pattern, a positive correlation between interference strength and genome wide recombination rates; we find support that interference strength has evolved in the two groups of male strains. There is a small number of positive correlations between genome wide recombination rate and interference strength in the literature. The within sex comparison of two breeds of cattle with different genome wide recombintion rates (Ma et al), between lab and wild mice of Peromyscus leucopus, and in a previous house mouse cross (Dumont,  preliminary data (HVR unpublished).

Theoretical models haven’t really considered the evolution of interference strength, neither the haploid selection or two locus modifier model can not be applied to evolution of interference strength. While the SACE modifier model does not explicitly model evolution of interference strength we note that a logical outcome of the main prediction of maintaining larger chromosome blocks in males, would be a landscape with stronger interference strength. The COM model predicts that interference and the recombination landscape arises from known oscillatory movements during prophase, it lacks evolutionary based predictions. We propose the spindle based selection model would support the evolution of interference strength in the positive direction via modulation of the amount of sister cohesion connecting homologs (figure).

Models from Goldstein et al (review in Otto and Payseur) suggest that if this pattern is widespread interference evolves whenever increased recombination rates evolve. Perhaps a distinguishing feature of models which come to this finding is that the number of crossovers is kept constant. The space across multiple loci (veller?) or between multiple crossovers increases in a positive manner with genome wide recombination rates. Given that the empirical range of crossovers per chromosome is quite small (1-3  (Otto Payseur 2019, Stapley et al 207) and the obligate crossover rule, the assumption of constraining the number of crossovers per chromosome fits well with empirical data.

Biological exceptions to interference:  experimentally increasing crossover number through mutants or fusion chromosomes  (Celegans, (plants, fungi with negative interference.

Fungi with negative interference (crossovers are spaced more closely together  in S.pombe and nidulans?. The formation of the DSBs for these crossovers is different from the usual ‘tethered loop’ model, (cite), so it is likely that interference (might be fundamentally different.

**REVIEW (spindle model the best)**

**Spindle model** is the most parsimonious

--also notable that  some of the reversals -- marsupial -- have strenge chromsome chains during MI (cite)