Main take away to the ‘paradox’

**Pattern of sexual dimorphism in the chromatin organization**

Females have longer axis and shorter DNA loops, a consequence of this is that there are different normalized measures of IFD (interference). The organization with longer loops results in stronger interference (males) because more of the DNA is folded up into the loops.

Due to this pattern – I propose that chromatin organization with longer DNA loops (shorter axis) results in stronger interference.

**The paradox is…**

High rec males clearly have stronger interference compared to the low rec strains,

According to the chromatin organization prediction (above), the high rec males should also have shorter axis and longer DNA loops.

But the ooposite is seen from the DSB measures and single bivalent SC length measures. High rec males seem to have longer SC (shorted DNA loops).

1. High rec males have significantly more DSBs – but same ratio of DSB to CO, suggesting the chromatin organization is driving this patterm
2. High rec males have slightly longer SC lengths (total SC) – this is mostly driven by lower KAZ measures of total SC. In the reduced SC lengths, the differences are not significant (but they are in the expected direction).

This is likely due to the noise in measuring indiviual SC lengths (noise from indiviual chromosomes and within nuc correlation in chromatin compaction).

**Paradox, data that doesn’t wuite fit expectations for the male groups:**

**1. difference in the normalized IFD and the raw IFD (the raw IFD should be closer to each other than the normalzied)**

**2. The SC length – interference prediction, (the short axis strains should have stronger interference)**

-the driving patterns

-IFD (the small / short IFDs in the low strains)

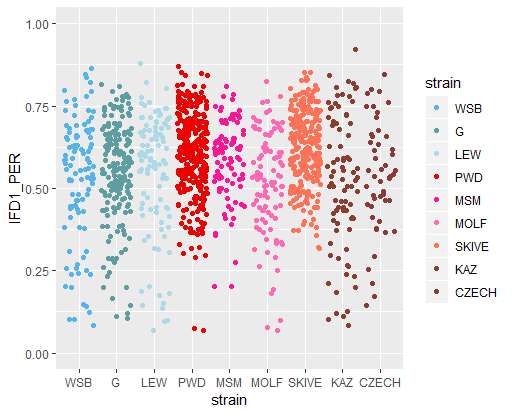
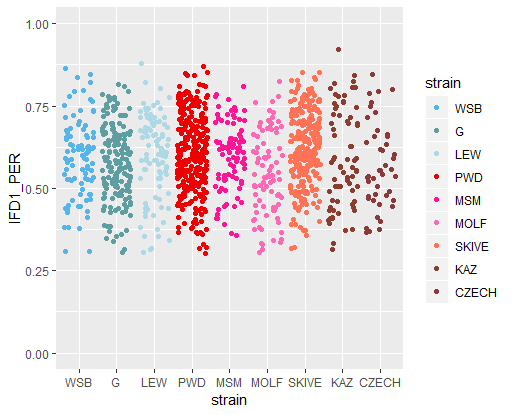
-SC length (KAZ has lower (total SC) compared to the other Musc)

How would this affect the patterns I’m observing?

(also, wouldn’t expect the difference in the raw IFD)

When I look closer – (at the IFDs of long bivalents

\*\* I noted that the primary driver of longer normalized IFDs is the lack of short IFDs in the high rec mice,



Data from removing short ifd observations in the ---

