Summary

The authors present a review of models relating to the long term maintenance of recombination suppression on sex chromosomes, and complement this with simulations exploring the 'constraint hypothesis' whereby recombination remains restricted due to a low rate of reversion. This paper is very clearly written, and provides a compelling argument for the limitations of the constraint scenario alone to drive long-term stability of recombination suppression on sex chromosomes. They also nicely summarise recent competing models by clearly delineating the possible short term causes of initial recombination arrest, and three main proposed mechanisms for the long-term maintenance of that initial arrest. Simulation results show that even rare reversions can be sufficient to remove non-recombining sex chromosomes from the population, given strong selection against Y chromosomes that accumulate a greater deleterious load. While the argument is in general clearly laid out and compelling, we have minor comments the authors should consider addressing.

General comments

1) A key goal of this paper is to evaluate what factors can drive the long-term maintenance of recombination suppression on the sex chromosomes despite Y degeneration. Furthermore, a major result of the simulations is that the constraint scenario is unlikely to be the explanation for the long-term suppression of recombination. However, it would be worth clarifying the timescales required to be considered 'long term'. Clearly mammalian sex chromosomes are ancient, but there is growing evidence that this may be the exception. Given that the simulation results show inversions that can persist for a million generations (and this clearly depends on the exact parameters that haven't been fully explored) the constraint scenario actually presents a quite interesting possible scenario that might well be consistent with what is observed in many taxa. In particular, many sex-linked regions may have relatively young origins, with heterogeneity across the chromosome in the timing of recombination suppression that might reflect variation in the accumulation of genetic load/the distribution of fitness effects, differences in rates and/or the precision of reversion, and other factors. In other words, a flip side to the authors' results is that this transient recombination suppression they show in their simulations might be a very important source for the recurrent turnover of sex linkage and degeneration seen in a growing number of taxa. This point would be useful to spell out more clearly- i.e. the mechanisms described in Jay et al could drive recurrent gains/losses of sex-linked regions over evolutionary time, a phenomenon that may in fact be quite common.

2) Further empirical support for the authors' arguments about the plausibility of reversion of inversions can be found in the recent literature. For example, Giner-Delgado et al. 2019 (doi: 10.1038/s41467-019-12173-x.) and Prubsky et al 2020 (https://doi.org/10.1038/s41588-020-0646-x) provide evidence for inversion hotspots whereby the same inversion polymorphisms have arisen multiple times

independently, suggesting the rate of back-mutation may be higher than often assumed. This is worth adding in the discussion (around line 353).

3) A recent preprint from Olito and Charlesworth (https://doi.org/10.1101/2023.11.27.568803) suggests that the conclusion from Jay et al that Y-linked inversions have a fixation advantage needs to be reconsidered, given the approach used to estimating fixation probabilities. Although the focus here is on the long-term maintenance of recombination suppression, this issue is probably worth mentioning in a revised version.

Minor comments

44: Typo. “shot” -> short