

Research Approach: Maize as a model of demography and selection in complex genomes

Maize as a model for complex genomes

Genome size variation in plants, humans. Differences in large complex genomes – Fraser (Fraser, 2013) vs. (Pyhäjärvi et al., 2013) or (Hancock et al., 2011)

Notes

(Peischl and Excoffier, 2015) predict more u-shaped SFS in expanded pops, also more homozygous deleterious. this is seen in humans. Li's results in maize

(Fu et al., 2014) “Indeed, a substantial amount of the higher density of deleterious alleles in EA individuals in the simulated data is attributable to weakly deleterious mutations ($|s| \approx 10^{-4}$)”

(Balick et al., 2013) show weighted sum of the SFS can be used to differentiate recessive vs. not at deleterious sites.

(Lohmueller, 2014) “Under a model where a mutation's effect on a trait is correlated with its effect on fitness, rare variants explain a greater portion of the additive genetic variance of the trait in a population that has recently expanded than in a population that did not recently expand. Further, when using a single-marker test, for a given false-positive rate and sample size, recent population growth decreases the expected number of significant associations with the trait relative to the number detected in a population that did not expand. However, in a model where there is no correlation between a mutation's effect on fitness and the effect on the trait, common variants account for much of the additive genetic variance, regardless of demography.”

(Tennessen et al., 2012) Shows vast majority of functionally important alleles rare, attributes to explosive population growth and weak purifying selection

(Hufford et al., 2013) (Hufford et al., 2012)

References

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