Maladaptation is common in nature – most telling by most species to have ever lived being extinct. Maladaptation can be caused by a variety of factors, but the end result is the same – populations stabilise around some distance away from a ‘phenotypic optimum’ – here populations are trapped by the competing effects of drift, mutation, and selection, and the various effects of the underlying traits’ effects on genetic architecture (including additive effect size variation, linkage, pleiotropy). Such behavior is usually modelled through the quantitative genetics lens of stabilizing selection on polygenic traits, however the focus is on adaptation towards an optimum rather than maladaptation at some point away from it. Following the adaptive walk towards an optimum, these models have expectations on how much genetic variance can be maintained, explaining how the population may be able to adapt to another environmental shift later on. Particularly prevalent are continuum of alleles models, where QTLs have many possible alleles, with distributions of effects that differ depending on the model’s underlying assumptions: whether mutation is strong versus selection (Gaussian) or weak (HoC). Whilst these models attempt to explain the dynamics of populations following adaptation, and the patterns of variation they can maintain, they hold little in the way of expectations of correlations between traits, and the effects of genetic architectures on variation. Yet neither Gaussian nor HoC models can predict observed variation in natural populations. Perhaps this is because these models behave differently under maladaptation than the adaptive framework they both assume to exist: it surely cannot be that most populations arrive exactly at an optimum. It has become clear that analytical approaches towards these complex adaptive scenarios may not be possible, owing to the many fine cogs churning our maladapted biosphere. To solve this problem, computational approaches have shown promise. Such computational models may be able to tease apart why both Gaussian and HoC models cannot explain natural variation by explaining the relative nature of adaptation versus maladaptation, how genetic architecture influences these models when populations are maladapted versus when they have reached the optimum, and how the underlying machinery (i.e. the allelic effects) behind these differences in trait variance, covariance, and adaptation is influenced by these models (and genetic architectures) under maladaptation versus adaptation.

Figure 1: Intro: Describe the differences between our models – HoC vs Gaussian (conceptual diagram with the differences between them)

Table 1: Methods: Parameter ranges for each of my variables, sources on why they were chosen etc.

Figure 2: Characterise the models in general – are we at m/s/d equilibrium? Are the Gaussian and HoC models behaving as expected in terms of variance? 2A: Distance/time 2B: Variance/time 2C: Cov/time (? maybe supplementary)

Figure 3: We know we are at equilbrium, so what model is more likely to get you there (HoC, Gaussian)? – Box plot of distance vs model type (HoC Gaussian Null)

Table 2: Statistically describe what model will take you to the optimum more often: Contingency table and chi square/Fisher exact of model type vs P(o) = 0 and P(o) = 1. Alternative is to compare mean probabilities with a regression analysis and put the regression table here.

Figure 4, 5, 6: We know that these models influence the ability to reach the optimum, but how do genetic architecture parameters influence these models? Is there a robustness against these effects with high/lower mutation/selection? Fig 4: Distance 5: Variance 6: Covariance – all boxplots of the dist/var/cov with the specific models and parameter, mean dot and conf interval also in there. Separated to two levels for those that reached the optimum versus those that didn’t.

Fig. 4,5,6) A – additive effect size, B – recombination rate, C – pleiotropy rate, D – mutational correlations

Tables 3,4,5,6,7,8: Regression analyses for the comparisons of means between each parameter and model type (the stuff outlined in figs 4,5,6)

Fig 7 – ultimately, underlying these differences between models with genetic architecture is the distribution of allelic effects: allelic fx of additive effect size vs model for models that reached the optimum vs those that didn’t (others parameters didn’t have as large an effect, so they will be in supplementary material)