Sexual selection can partly explain low frequencies of Segregation Distorter alleles

Supplementary material

Thomas A Keaney, Therésa M Jones and Luke Holman

Click **here** to view the HTML report, which serves as online supplementary material for the associated manuscript (http://dx.doi.org/10.1098/rspb.2021.1190), published in *Proceedings of the Royal Society B*. The report is split into three parts. The first provides the supplementary methods, the second documents our empirical analysis (contains raw data) and the third provides an in-depth explanation for how we coded our population genetic model. Together, the report includes all supplementary figures and tables, the R script required to produce the analysis, figures and tables, and the raw data.

In an attempt to future proof the availability of our supplementary material, we also include the supplementary methods, Table S1-3 and Figures S1-8 in this document. Additionally, our raw data is deposited in the Dryad database **here**.

Pilot experiment: confirming that *SD* exhibits segregation distortion

In a pilot experiment, we measured the strength of segregation distortion produced by each of our experimental treatment lines. We crossed females from each of the three *SD/+* lines and the *+/+* line to males homozygous for the *bw* mutation (Figure S1); like *SD*, *bw* is located on chromosome 2, so this cross yielded *SD/bw* or *+/bw* progeny. We then mated 20 *SD/bw* (or *+/bw*) males from each of the four crosses to *bw/bw* females, and recorded the eye colour (red or brown) of the resulting female offspring to determine the proportion of offspring fertilised by *SD-* (or +) and *bw*-bearing sperm. Male progeny were not counted because some of them in the reciprocal cross (see below) expressed a white-eye phenotype (due to male hemizygosity and an X-linked mutation of *white*), preventing us from determining which copy of chromosome 2 they inherited.

SD alleles are commonly associated with viability costs, which might cause underestimation of the strength of segregation distortion. To correct for any such viability costs, we also performed the reciprocal cross (*SD/+* females × *bw/bw* males) and calculated the proportion of offspring inheriting the *SD* bearing chromosome as above. Because *SD* does not affect segregation in females, a shortage of adult offspring carrying *SD* (relative to the 50% Mendelian expectation) indicates reduced survival of *SD* progeny to adulthood (relative to bw progeny). We calculated the viability-corrected estimate of segregation distortion, k_c, using the formula in Temin (1991, Genetics).

To analyse our results, we fit a binomial model, in which red-eye daughters (i.e. the progeny that inherited the *SD* or *+* allele from their *SD/bw* or *+/bw* father) were treated as 'successes' and the brown-eye daughters as 'failures'. We included the sex of the experimental individual and the variant of *SD* (or control) as fixed effects (with the control as the reference level), as well as the interaction between these variables. We also included pair ID as a random effect.

Table S1: recipe for food medium used in our experiment. The provided quantities make ~ 1 litre of food.

Ingredients	Quantity
Soy flour	20 g
Cornmeal	73 g
Yeast	35 g
Dextrose	75 g
Agar	6 g
Water	1000 mL
Tegosept	17 mL
Acid mix (4 mL orthophosphoric acid, 41 mL propionic acid, 55 mL water to make 100 mL)	14 mL

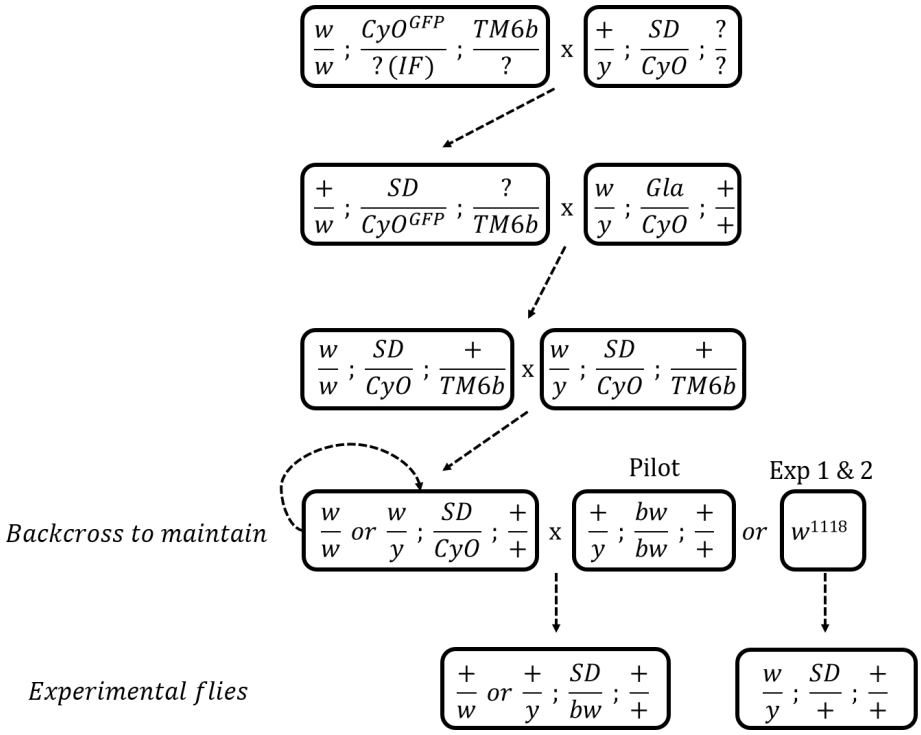

Figure S1. Crossing scheme used to standardise the genetic background across the *SD-5/+*, *SD-72/+*, *SD-Mad/+* and *SD+/+* lines. The *SD+/+* line was created in identical fashion except that we substituted the *SD* bearing chromosome with chromosome 2 from the w^{1118} isogenic line. Note that at step four there are three possible options 1) the leftmost genotype can be backcrossed to maintain it in the laboratory, 2) the leftmost genotype can be crossed to a *bw* stock to produce the experimental flies used in Experiment 1

or 3) the leftmost genotype can be crossed to w^{1118} to create the experimental flies used in Experiments 2 and 3.

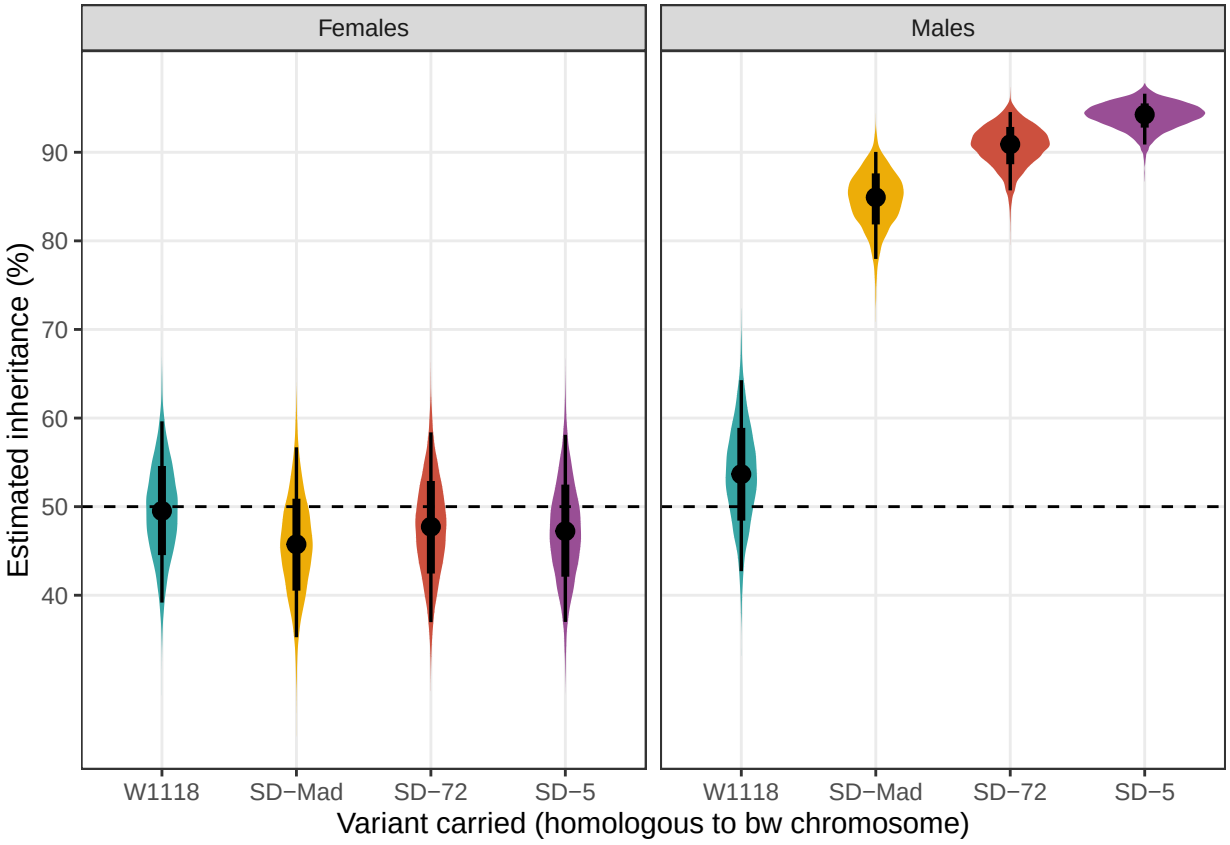

Figure S2: The estimated percentage of female offspring that inherited a *SD* allele from a heterozygous parent, split by the sex of the parent. Black points indicate the estimated mean with associated 66 and 95% uncertainty intervals, while coloured area shows the posterior distribution. The dotted line indicates 50% inheritance; the expectation in the absence of segregation distortion.

Table S2: the viability corrected inheritance (following Temin, 1991) of each *SD* variant and the control allele from a *SD/+* (or *+/+*) male.

Variant carried	k corrected for viability costs	Q2.5%	Q97.5%
SD-5	0.944	0.818	0.985
SD-72	0.909	0.724	0.975
SD-Mad	0.868	0.634	0.963
W1118	0.542	0.397	0.682

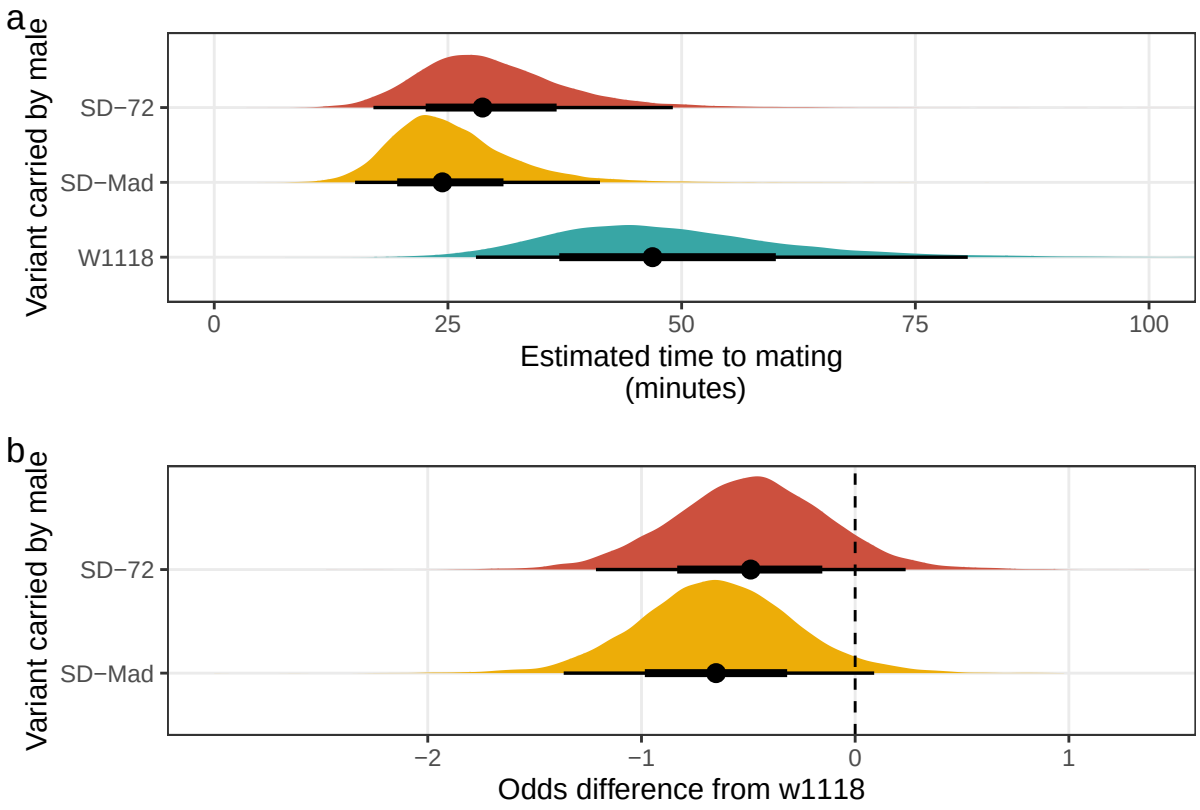

Figure S3: the mean time required for an *SD/+* male to start mating with a LH_m female in Experiment 1, and how this compares to w^{1118} control males. Panel **a** shows the estimated mating latency for *SD/+* and control males. Panel **b** shows effect sizes on the odds scale for the *SD* variants. Negative values indicate that *SD/+* males mated faster than w^{1118} control males. Black points indicate the estimated mean with associated 66 and 95% uncertainty intervals, while coloured area shows the posterior distribution.

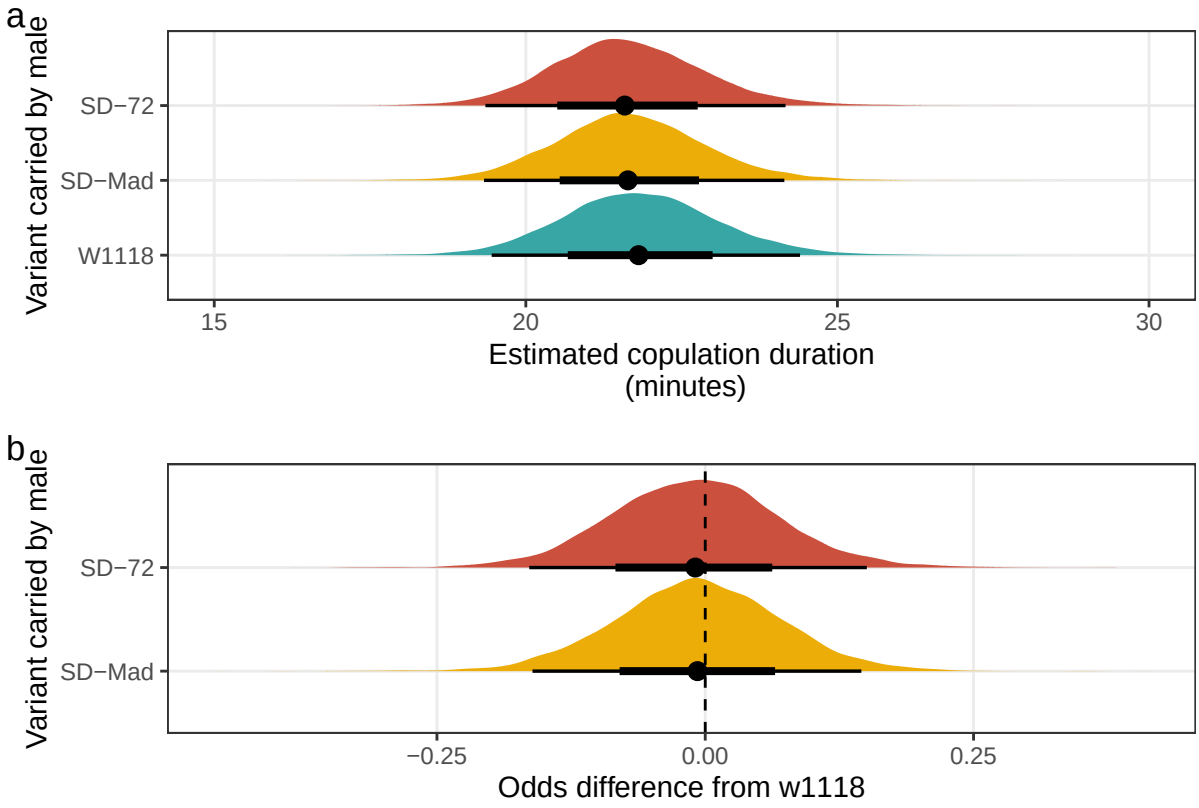

Figure S4: no difference in the duration of mating between a $SD/+$ male and a LH_m female in Experiment 1, compared to w^{1118} control males. Panel **a** shows the estimated copulation duration for $SD/+$ and control males. Panel **b** shows effect sizes on the odds scale for the SD variants. Positive values indicate that $SD/+$ males mated for longer than w^{1118} control males. Black points indicate the estimated mean with associated 66 and 95% uncertainty intervals, while coloured area shows the posterior distribution.

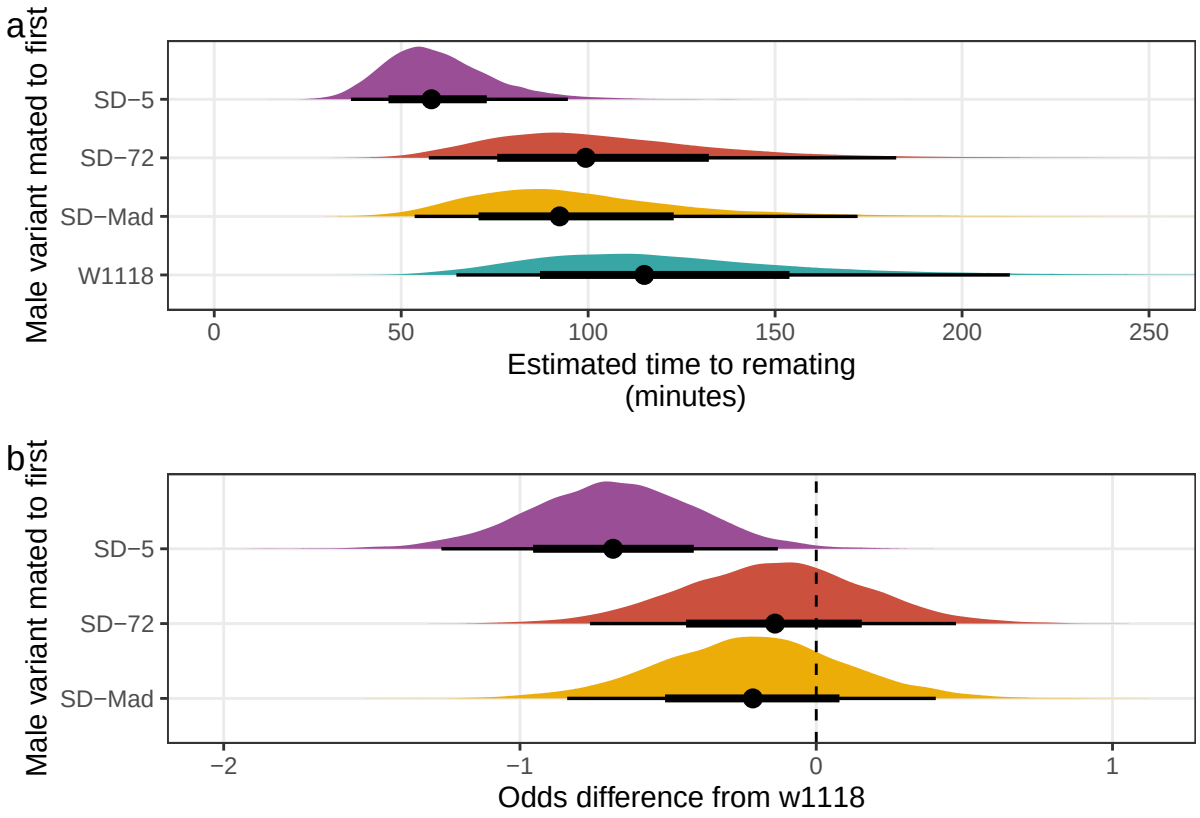

Figure S5: the effect that *SD/+* males had on female remating latency in Experiment 2, and how this compares to w^{1118} control males. Panel **a** shows the estimated remating latency for females exposed to ${LH_m}^{UBI}$ males over a three-hour period, that had mated with a *SD/+* or control male four days earlier. Panel **b** shows effect sizes on the odds scale for the *SD* variants. Negative values indicate that the mates of *SD/+* males remated faster than the mates of w^{1118} control males. Black points indicate the estimated mean with associated 66 and 95% uncertainty intervals, while coloured area shows the posterior distribution.

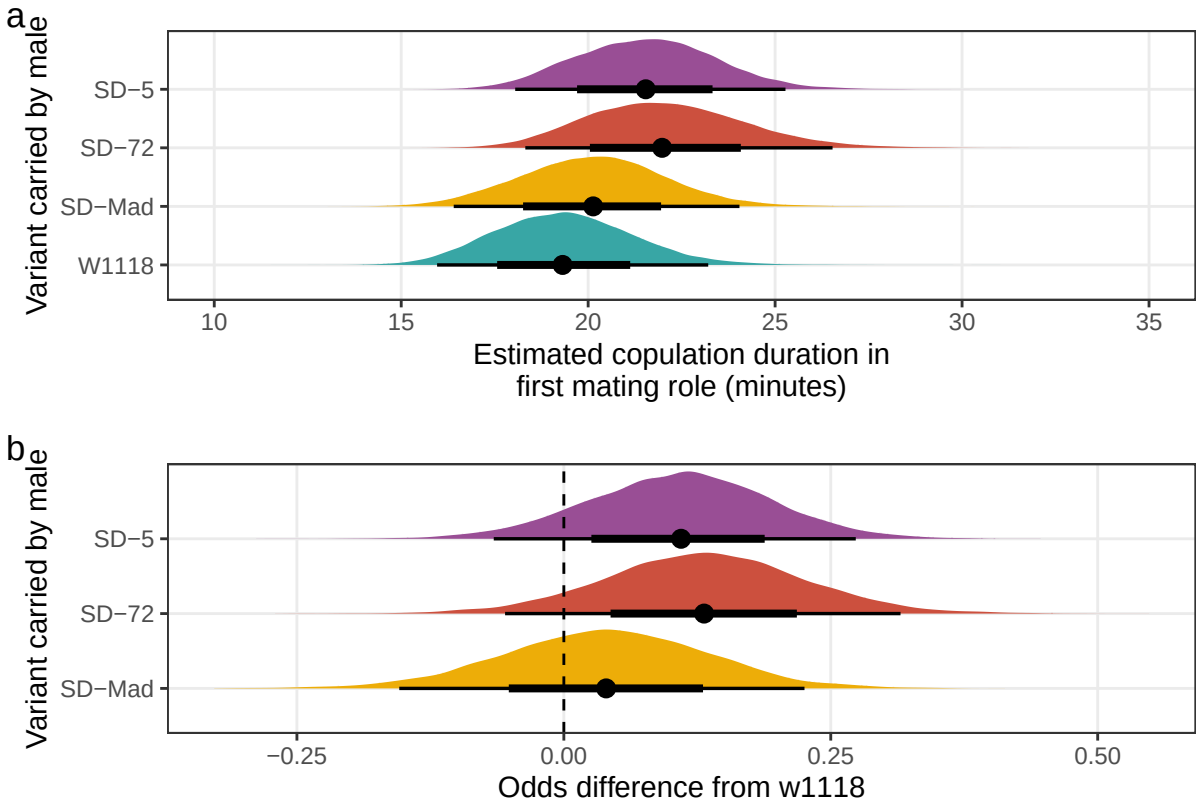

Figure S6: no difference in the duration of mating between a *SD/+* male and a LH_m female in Experiment 2, compared to w^{1118} control males, when the *SD/+* (or control) male mated first. Panel **a** shows the estimated copulation duration for *SD/+* and control males. Panel **b** shows effect sizes on the odds scale for the *SD*-variants. Positive values indicate that *SD/+* males mated for longer than w^{1118} control males. Black points indicate the estimated mean with associated 66 and 95% uncertainty intervals, while coloured area shows the posterior distribution.

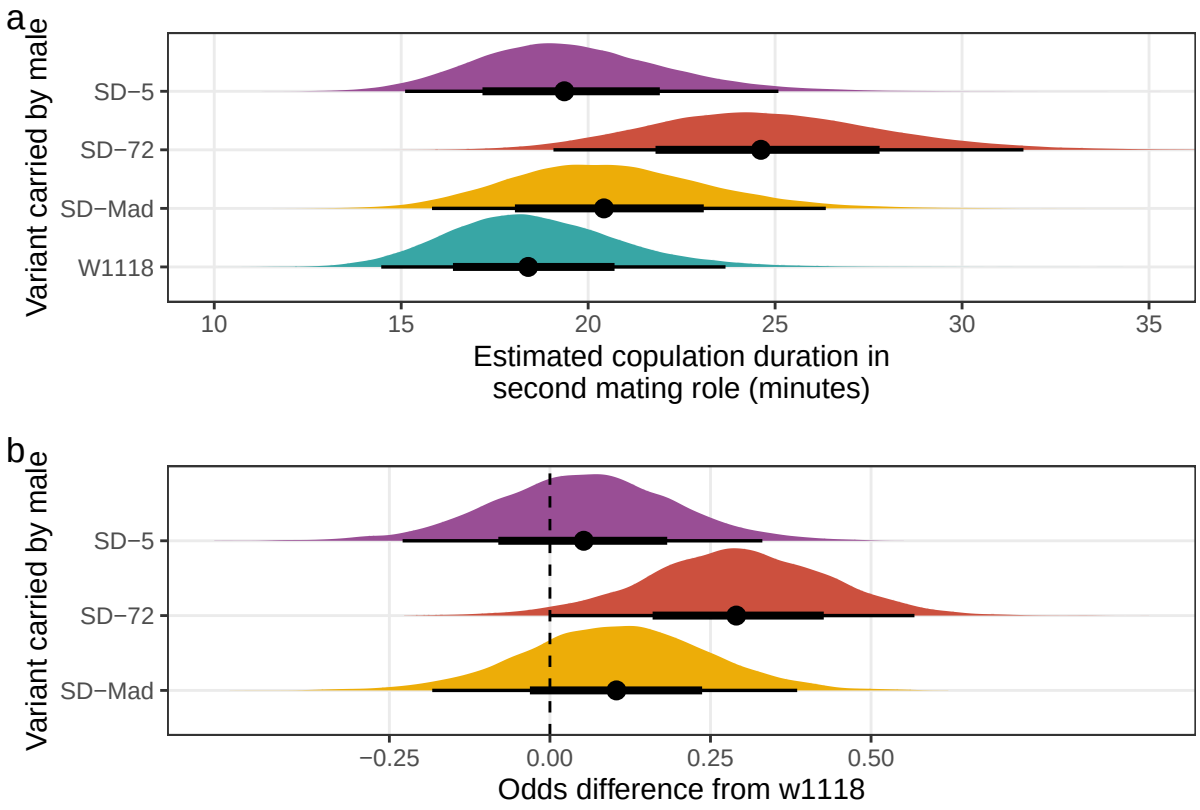

Figure S7: mating duration between a *SD/+* male and a LH_m female pair in Experiment 2, compared to w^{1118} control males, when the *SD/+* (or control) male mated second. Panel **a** shows the estimated copulation duration for *SD/+* and control males. Panel **b** shows effect sizes on the odds scale for the *SD* variants. Positive values indicate that *SD/+* males mated for longer than w^{1118} control males. Black points indicate the estimated mean with associated 66 and 95% uncertainty intervals, while coloured area shows the posterior distribution.

Checking that our simulation codes are consistent with previous analytical models

We can find *SD* allele frequencies for varying levels of segregation distortion if the only fitness cost associated with *SD* is homozygote lethality. This represents an upper bound for the frequency of *SD* alleles in our fully parameterised simulation.

Simplifying the simulation is also a useful exercise for comparing our model with previous models exploring the effect of homozygote lethality on segregation distorter frequencies. We find that our model reproduces the frequencies found using these previous models.

Bruck (1957) showed that homozygous lethal segregation distorting alleles (assuming no other fitness costs) reach an equilibrium frequency in adults at $q = \frac{1}{2} - \frac{\sqrt{k(1-k)}}{2k}$, where k is the strength of segregation distortion. We plug the three k values we observed in our pilot experiment into this equation and contrast the results with our simplified simulation results in Table S3 below. The code used to produce this table can be found **here**.

Table S3: *SD* equilibrium frequencies calculated assuming varying levels of segregation distortion (k) and no fitness cost other than homozygote lethality associated with *SD* alleles. The table shows the equivalence between using our simulation code and the difference equation presented in Bruck (1957).

k	Simulation SD equilibrium freq	Bruck SD equilibrium freq
0.5	0.001	0
0.944	0.378	0.378
0.909	0.342	0.342
0.868	0.305	0.305

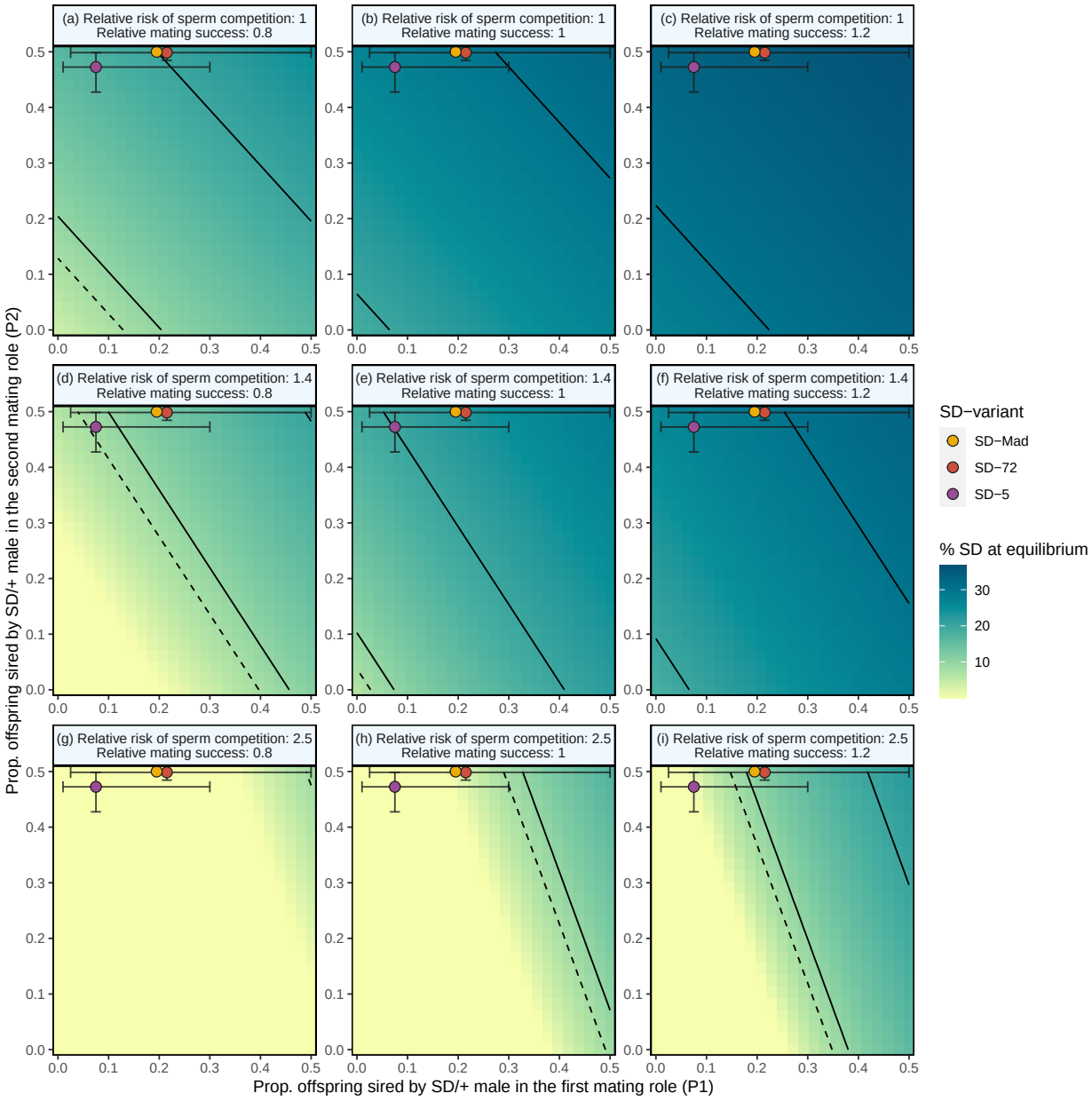

Figure S8: Predicted equilibrium frequency of the *SD* allele, calculated from the population genetic model. The plot depicts the interaction between the P1 (x-axis) and P2 (y-axis) costs suffered by *SD/+* males in their effects on the equilibrium frequency of *SD* (shown by the colour scale and 10% contour lines). The dashed line shows an equilibrium frequency of 8%, the upper estimate for *SD* alleles in natural populations. The plot is split into nine panels, with varying levels of *SD/+* male mating success across the rows and increasing likelihoods of a female remating after first mating to an *SD/+* male, $p_{SD/+}$, down the columns. The three points (with associated 95% credible intervals) in each panel show where males carrying each *SD* variant fall in the figure's parameter space. In the parameter space presented here, $k = 0.944$ (our highest estimated value), $P1_{normal} = 0.5$, *SD* homozygotes are non-viable and *SD* heterozygotes suffer no fitness costs outside of mating success and sperm competition.