Supplementary Material (Appendix A) for: Sexually antagonistic coevolution between the sex chromosomes of *Drosophila melanogaster*. *PNAS*

Katrine K. Lund-Hansen[a,b], Colin Olito[a], Edward H. Morrow[b,c], & Jessica K. Abbott[a]

[a] Department of Biology, Section for Evolutionary Ecology, Lund University, Lund 223 62, Sweden.

[b] School of Life Sciences, University of Sussex, Brighton BN1 9QG, United Kingdom.

[c] Department for Environmental and Life Sciences, University of Karlstad, 651 88, Sweden,

Corresponding author e-mail: katrine.lund-hansen@biol.lu.se

Materials and Methods

Drosophila **husbandry and fly stocks**. All fly stocks were kept on a two-week regime with non-overlapping generations. On day 12 after oviposition, 16 pairs of adult flies were randomly selected from the pool of adults and placed in a food-vial with 6 mg yeast. The pairs were left in the vials for two days, and then flipped (transferred without CO_2) into fresh vials without yeast. The females were allowed to oviposit for 18 hours, after which all adult flies were discarded. The number of eggs was reduced to 150-200 per vial to maintain constant larval density.

For the creation of the novel populations we used clone-generator (CG) females with a double-X-chromosome (DX), a Y-chromosome, and two translocations of chromosome II and III (C(1)DX, *y*, *f*; T(2;3) *rdgC st in ri* p^P bw^D).

Construction of *novel X* and *Y* populations. The five wild-type stocks were crossed in a round robin-scheme where one stock was crossed to two others. Pairings were performed reciprocally, generating four novel populations for each wild-type population (*wt*), giving a total of ten *novel X* (X chromosome from a different population than the rest of the genome) and ten *novel Y* (Y chromosome from a different population than the rest of the genome) populations. The crosses were synchronised such that male fitness could be assayed simultaneously from all 20 novel populations.

To illustrate the principle of the entire crossing scheme we will employ the use of two hypothetical wild-type populations A and B:

Novel Y - Cross 1: CG females were crossed to A males (Fig. S1.1). This cross was performed with 672 breeding adults (♀32:16♂ per vial) in 14 vials and flipped once without reducing egg numbers. Cross 2: Heterozygote females (DX-T(2;3)/A)

were crossed to *wt* A males a second time to produce the DX-A females required for crosses 3 and 4 in the *novel X* crossing scheme (see below; Fig. S1.2). This cross was performed with 640 breeding adults (♀16:16♂ per vial) in 20 vials and flipped twice without reducing egg numbers. Cross 3: Heterozygote females (DX-T(2;3)/A) were crossed to B males (Fig. S1.3), with 320 breeding adults (♀16:16♂ per vial) in ten vials and flipped twice without reducing egg numbers. Cross 4: B females were crossed to heterozygote males (T(2;3)/B) bearing a Y chromosome from A (Fig. S1.4) with 224 breeding adults in seven vials without reducing egg numbers. This creates *novel Y* males, for use in subsequent assays, where Y chromosomes from A have been placed in a B genetic background (Fig. S1.5).

Novel X - Cross I: CG females were crossed to B males (Fig. S1.I), with 672 breeding adults (♀32:16♂ per vial) in 14 vials and flipped once without reducing egg numbers. Cross II: Heterozygote females (DX-T(2;3)/B) were crossed to B males (Fig. S1.II), with 640 breeding adults (♀16:16♂ per vial) in 20 vials and flipped twice without reducing egg numbers. Cross III: DX-A females (from cross 2 in *novel Y*) were crossed to heterozygote males (T(2;3)/B) (Fig. S1.III), with 320 breeding adults (♀16:16♂ per vial) in ten vials and flipped twice without reducing egg numbers. Cross IV: DX-A females (from cross 2 in novel Y) were crossed to heterozygote males (T(2;3)/A) bearing a X chromosome from B (Fig. S1.IV). This cross was performed with 224 breeding adults (♀16:16♂ per vial) in seven vials without reducing egg numbers. This creates *novel X* males, for use in subsequent assays, where X chromosomes from population B have been placed into a population A genetic background Fig. S1.V).

In both crossing schemes it should be noted that since there is no balancer for the small dot chromosome IV, we could not control how it was inherited. The dot

chromosome only makes up 1% of the total genome and should thus have a limited effect (1). Since the dot chromosome is almost non-recombining (2), at the end of the *novel Y* crosses the probability of the dot chromosome being from the corresponding autosome population is 0.4. For the *novel X* crosses the probability is 0.5, and any effects of chromosome IV origin would be averaged out across the multiple individuals assayed.

Construction of novel XY populations. To estimate the effect of changing the autosomal background when both sex chromosomes were transferred together, we crossed four wild-type populations (*Dahomey*, *Innisfail*, LH_M, and *Odder*) to create novel populations where X and Y chromosomes from one population are placed into a genetic background of the two largest autosomes from a second population. As with the *novel X*/*Y* experiment, crosses were synchronized so that male fitness could be assayed simultaneously for all novel populations. Again, we illustrate the crossing scheme using two hypothetical populations A and B.

Novel XY - Cross 1: DX-A females were crossed to A males with a Y chromosome from B (Fig. S3.1), with 224 breeding adults (♀16:16♂ per vial) in 7 vials. Cross 2: DX-A females with a Y chromosome from B were crossed to A males with a X chromosome from B (Fig. S3.2), with 448 breeding adults (♀16:16♂ per vial) in 14 vials. This creates males with both sex chromosomes from B but autosomes from A (Fig. S3.3). The probability of the dot chromosomes being from the same population background as autosome II and III was in this case 0.7.

Evolution experiment protocol. The three treatments (*novel X*, *novel Y*, and *wt*) were kept in an adult breeding population of 160 pairs (♀ 16:16 ♂ per vial) in 10 vials,

with a minimum of 1,500 offspring. There were four populations of each treatment, with each population kept in two replicates.

Novel X – Males carrying a novel X chromosome were crossed at each generation to females who carried a double-bound X-chromosome (DX) and a Y chromosome (Fig. S11, *Novel X*). Because only half of the offspring of DX females survive to adulthood, the number of eggs were reduced to ~300 for these populations. The other half is inviable because they receive the wrong complement of sex chromosomes, either two Y chromosomes or three X chromosomes.

Novel Y – At each generation males carrying a novel Y chromosome were cross to X/X females (Fig. S11, *Novel Y*).

Wt population – The four *wt* populations were kept at the same population size as the novel treatments.

Selected populations. For all the following phenotypic assays we chose to assay novel populations with a ΔFitness significantly different from zero (Table S5). For the evolution experiment we selected four *novel X* populations (*Innisfail*–L_X, *Innisfail*–O_X, *Odder*–I_X, and *Odder*–D_X), four *novel Y* populations (*Innisfail*–L_Y, *Innisfail*–O_Y, *Odder*–I_Y, and *Odder*–D_Y), and their four corresponding *wt* populations (*Dahomey*, *Innisfail*, LH_M, and *Odder*).

Male thorax size. Body size was estimated using measurements of thorax length. On day 12 after oviposition, 50 males from the selected populations were placed in 95 % ethanol. Air-dried males were measured using a Nikon SMZ800 dissecting microscope at 63x magnification fitted with an eyepiece graticule. To ensure

repeatability of the measurements, one person measured all the flies and we presumed that any bias would be equalised over the populations.

Sperm competition assay. Sperm offence (P2) was determined by the share of paternity achieved by the last male mated to a female. Five LH_M-*bw* virgin females were combined for an hour with ten LH_M-*bw* males. The females were the left on their own for 48 hours where after they were combined for 24 hours with ten target males from the selected populations. The females were then isolated in test tubes for 18 hours and the tubes were left under standard LH_M conditions for 12 days. The adult offspring of the five females were counted and scored for eye-colour to assess paternity. P2 was assayed at two different time points: generation 6 (two blocks, seven experimental replicates per selected population in each) and generation 30 (one block with seven experimental replicates per selected population).

Female longevity with male harassment. Five LH_M virgin females were collected on day of eclosion and crossed with five target males from the selected genotypes in a yeasted vial on day 12 after oviposition. Mortality of the females was scored daily, six days a week until 95% of them were dead. The flies were flipped into new food-vials with yeast every third day. At every flip, new males and LH_M-*bw* females were added to the vials to replace any dead flies and keep the sex ratio equal. The flies were maintained under standard LH_M conditions until death. This assay was done with ten experimental replicates per population.

Male effect on female fecundity. Male effect on female fecundity was measured as the number of eggs laid by females during an 18 hour period, which corresponds to

the period normally available to females from the base population (3). Five LH_M virgin females were collected on the day of eclosion and combined with five males from the selected populations on day 12 after oviposition. After two days the five females were isolated into single test-tubes for 18 hours to lay eggs and were then discarded, while the test-tubes were frozen so the eggs could be counted at a later date. The fecundity for each female was averaged across the number of eggs laid by the five females. This assay was performed in nine experimental replicates per population.

Offspring egg-to-adult viability assay. Offspring egg-to-adult viability was estimated as the proportion of 100 eggs that developed into live adults within 12 days. 20 pairs of flies from each of the selected populations were placed in bottles with a Ribena-agar medium plate overnight to oviposit (Ribena is a blackcurrant-flavoured juice drink). 100 eggs were counted and transferred to a fresh food-vial, and left under standard LH_M conditions. After 12 days all offspring and pupae were counted and sexed for estimation of sex ratio. Egg-to-adult viability was assayed at two different time points: generation 4 (two blocks, five experimental replicates per selected population in each) and generation 39 (four blocks with six experimental replicates per selected population per replicated population in one block).

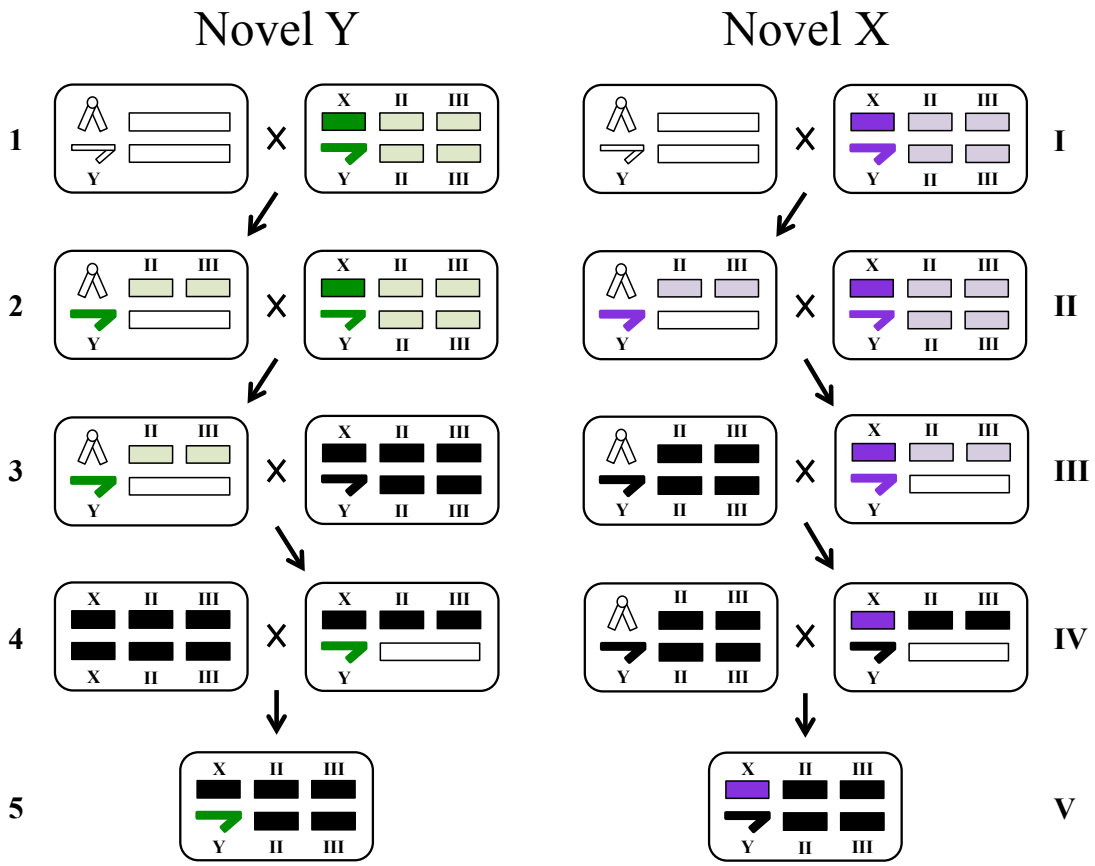

Fig. S1: Protocol for the crosses to create novel populations. Autosomes (II and III) and chromosome X are depicted as rectangles, and the autosome translocation (2:3) is depicted as the elongated white rectangle. The double-X-chromosome is depicted as ♒ and the chromosome Y as a half arrow. Only offspring used for the cross in the next generation is shown. For additional details see *Construction of novel X and Y populations*.

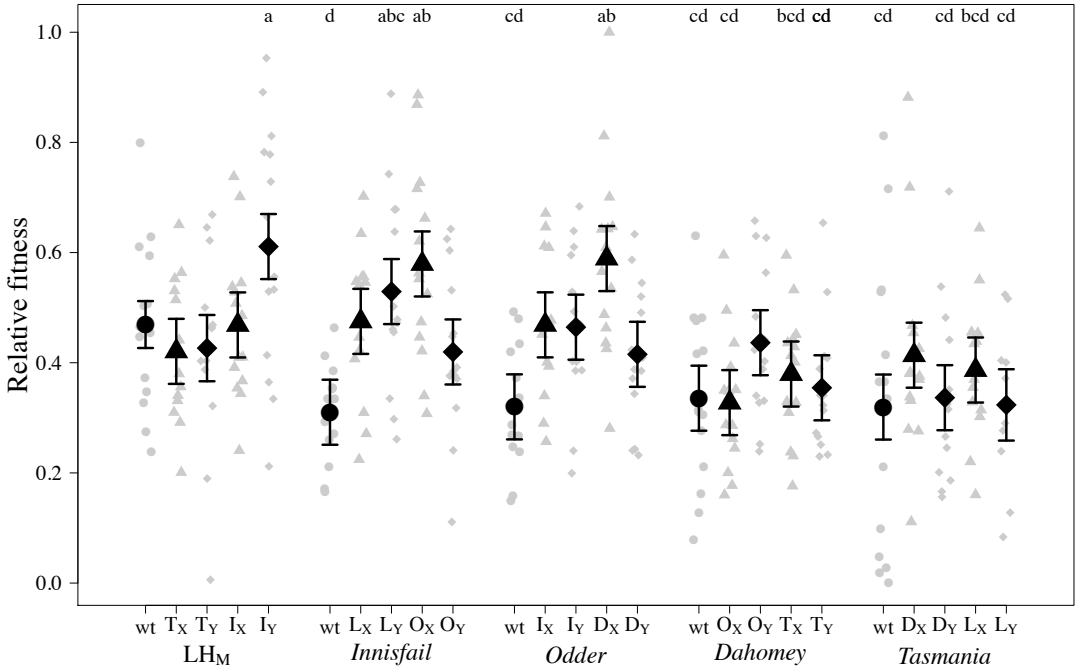

Fig. S2: Male reproductive fitness assays at generation 0. Mean (±SE) of fitted values from the linear model. The raw data is plotted as grey points. *wt:* black circle, *novel X:* triangle**,** *novel Y:* diamonds. To make the plot more readable only populations that are significant different from each other are denoted with letters (Tukey HSD; $P < 0.05$).

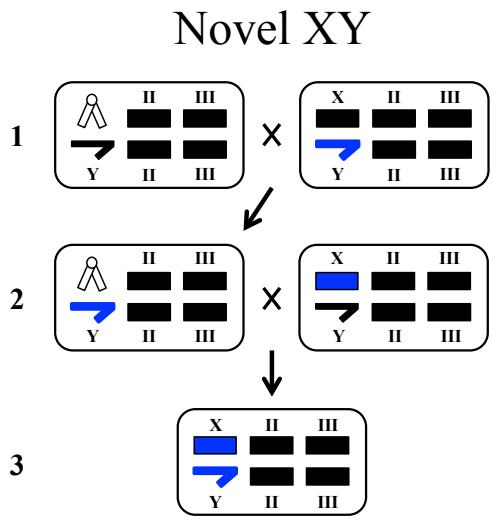

Fig. S3: Protocol for the crosses to create *novel XY* populations. Autosomes (II and III) and chromosome X are depicted as rectangles, the double-X-chromosome is depicted as ⩚, and the chromosome Y as a half arrow. Only offspring used for the cross in the next generation is shown. For additional details see *Construction of novel XY populations*.

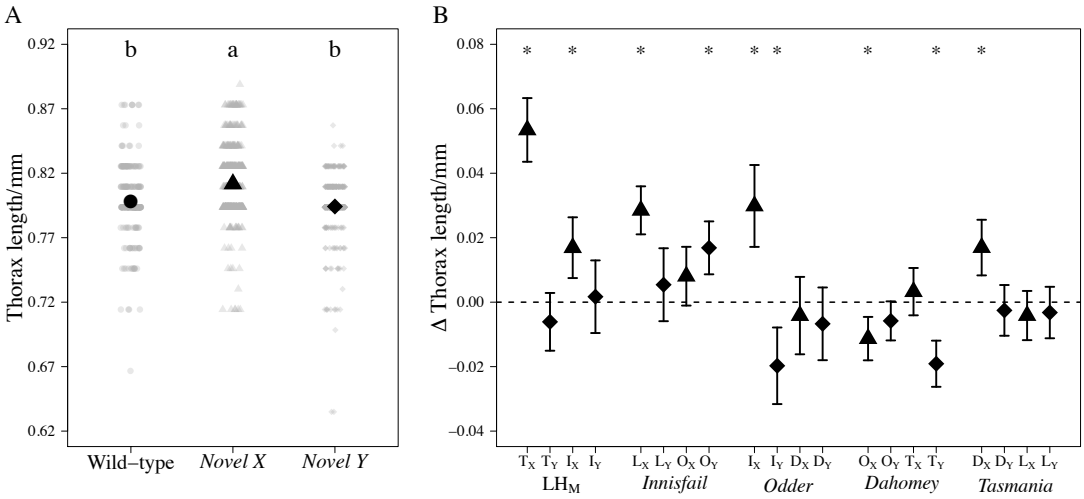

Fig. S4: Thorax Size. (A) Comparison between the three treatments. *Novel X* males were significant larger than the two other treatments. Mean (±SE) of fitted values from the linear models. *wt:* black circle, *novel X:* triangle**,** *novel Y:* diamonds**.** The raw data is plotted as grey points. Letters indicates significance (Tukey HSD; $P < 0.05$). (B) Change in thorax size between the *wt* and the novel populations (Δ = novel population - wild-type) with bars indicating bootstrap 95% confidence. *Novel X:* triangle *and novel Y:* diamonds**.** Asterisks indicate significance difference.

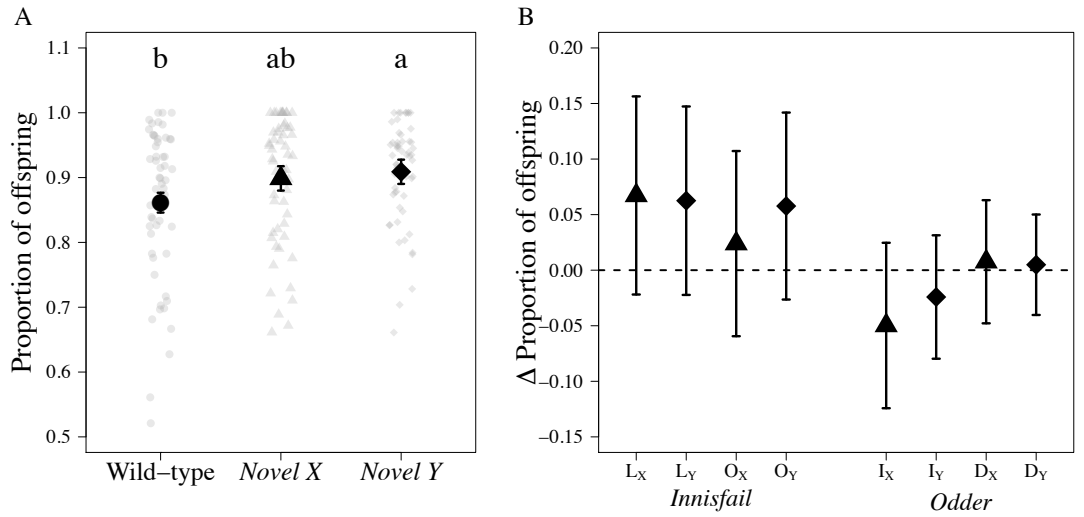

Fig. S5: Sperm competition. (A) Comparison between the three treatments. *Novel Y* males were significantly better at replacing sperm than the other two treatments. Mean (±SE) of fitted values from the linear models. *wt:* black circle, *novel X:* triangle**,** *novel Y:* diamonds**.** The raw data is plotted as grey points. Letters indicate significance (Tukey HSD; $P < 0.05$). (B) Change in proportion of offspring between

the *wt* and the novel populations (Δ = novel population - wild-type) with bars indicating bootstrap 95% confidence. *Novel X:* triangle *and novel Y:* diamonds**.**

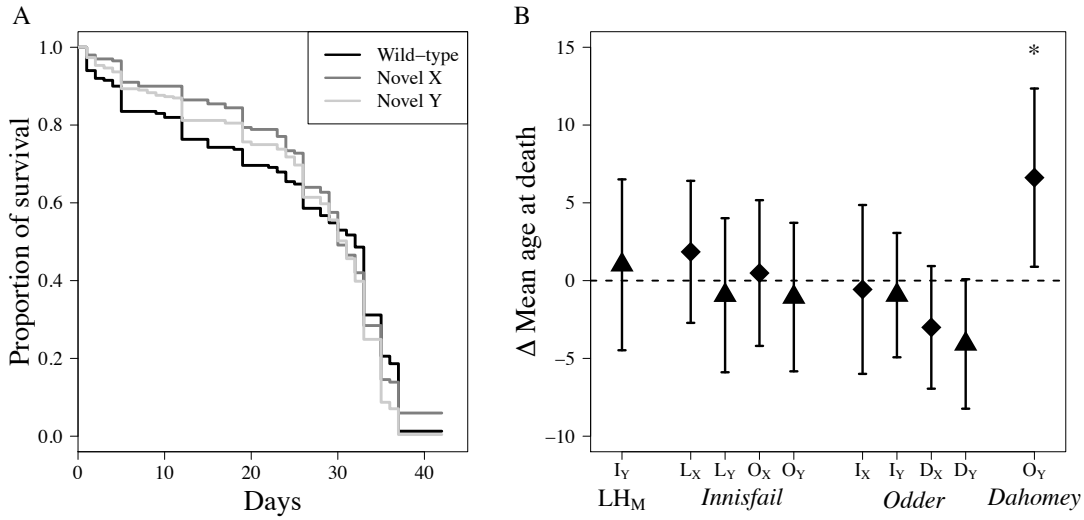

Fig. S6: Male effect on female longevity. (A) Comparison between the three treatments. There was no significant difference in female lifespan when continually exposed to males from the treatments. *wt:* black line, *novel X:* grey line**,** *novel Y:* light grey line**.** (B) Change in mean age at death between the *wt* and the novel populations (Δ = novel population - wild-type) with bars indicating bootstrap 95% confidence. *Novel X:* triangle *and novel Y:* diamonds**.** Asterisks indicate significance difference.

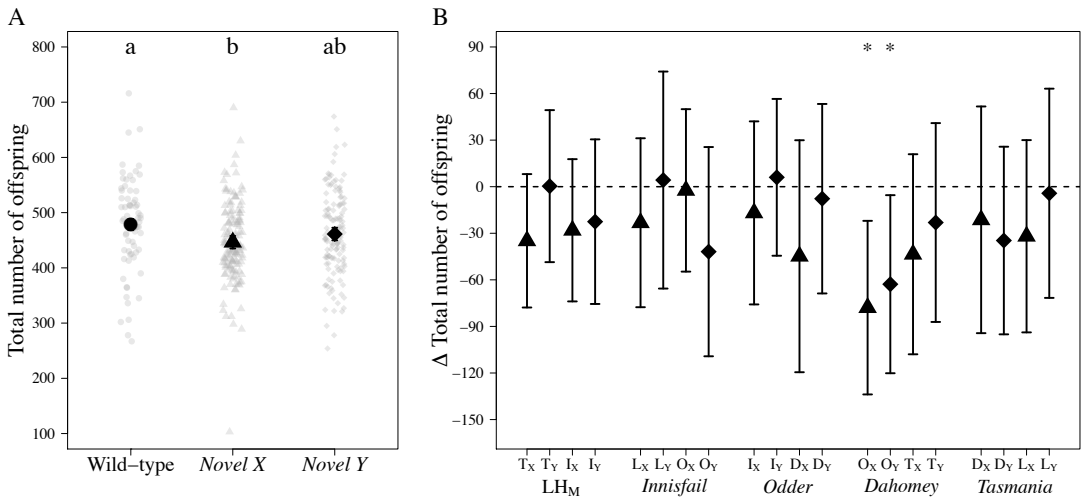

Fig. S7: Total number of offspring. (A) The total number of offspring produced by LH_M-*bw* females in the male reproductive fitness assay. There was a significant lower number of offspring sired by *novel X* males. Mean (±SE) of fitted values from the linear models. *wt:* black circle, *novel X:* triangle**,** *novel Y:* diamonds**.** The raw data is

plotted as grey points. Letters indicate significance (Tukey HSD; $P < 0.05$). (B) Change in total number of offspring between the *wt* and the novel populations (Δ = novel population - wild-type) with bars indicating bootstrap 95% confidence. *Novel X:* triangle *and novel Y:* diamonds**.** Asterisks indicate significance difference.

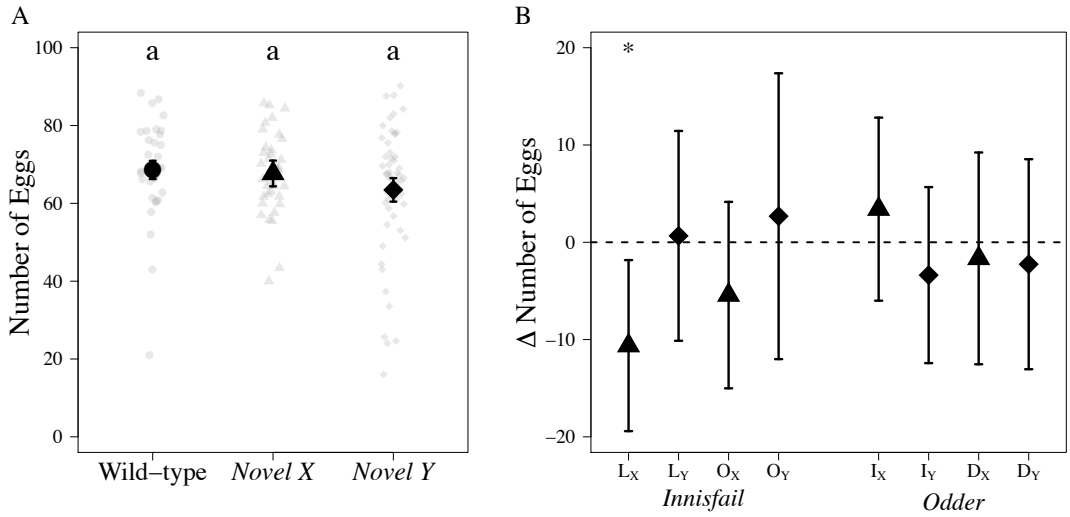

Fig. S8: Male effect on female fecundity. (A) There was no effect of males from the novel treatments on female fecundity. Mean (±SE) of fitted values from the linear models. *wt:* black circle, *novel X:* triangle**,** *novel Y:* diamonds**.** The raw data is plotted as grey points. Letters indicate significance (Tukey HSD; $P < 0.05$). (B) Change in number of eggs between the *wt* and the novel populations (Δ = novel population - wild-type) with bars indicating bootstrap 95% confidence. *Novel X:* triangle *and novel Y:* diamonds**.** Asterisks indicate significance difference.

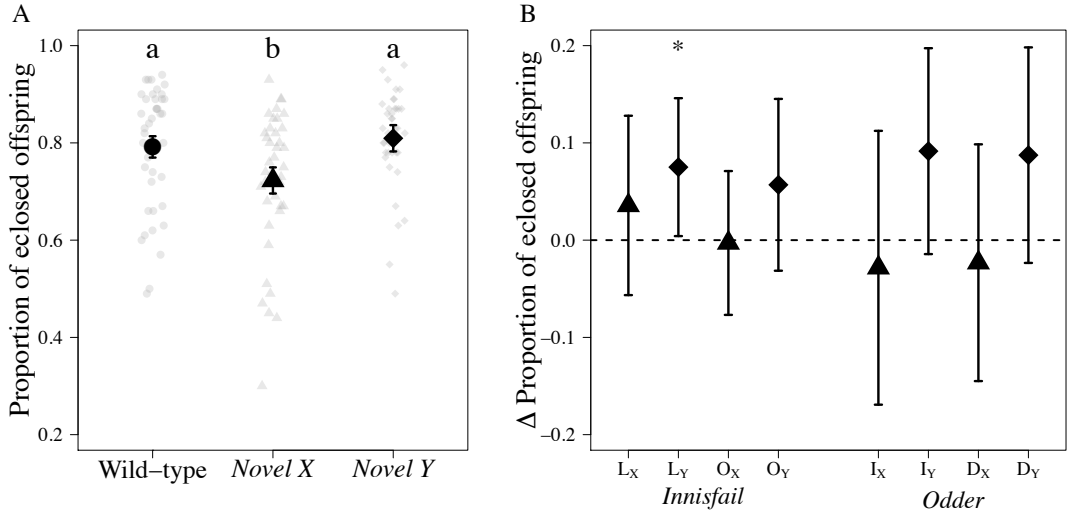

Fig. S9: Egg-to-adult offspring viability. (A) The offspring of *novel X* males had a significant lower survival than the two other treatments. Mean (±SE) of fitted values from the linear models. *wt:* black circle, *novel X:* triangle**,** *novel Y:* diamonds**.** The raw data is plotted as grey points. Letters indicates significance (Tukey HSD; $P < 0.05$). (B) Change in proportion of eclosed offspring between the *wt* and the novel populations (Δ = novel population - wild-type) with bars indicating bootstrap 95% confidence. *Novel X:* triangle *and novel Y:* diamonds**.** Asterisks indicate significance difference.

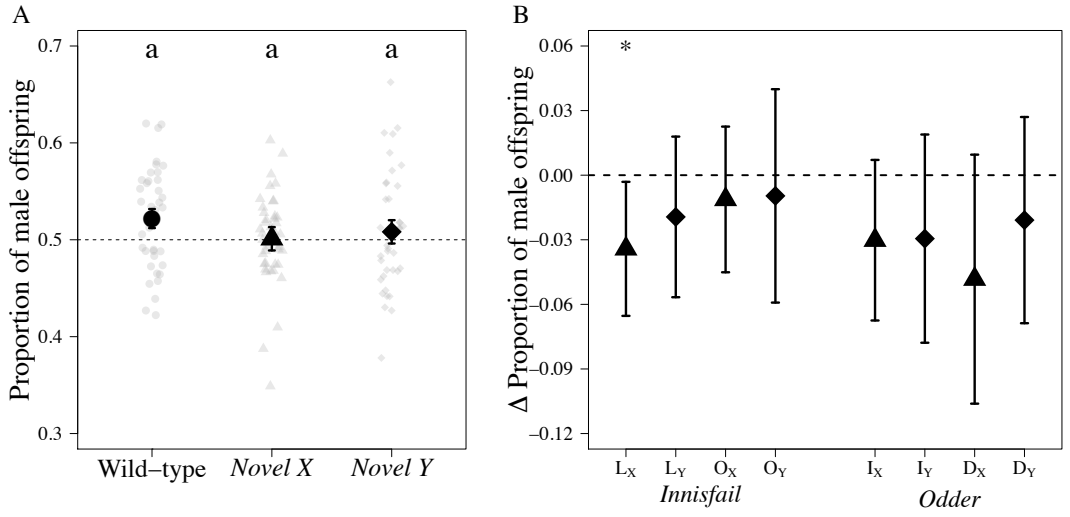

Fig. S10: Sex ratio. (A) There was no significant difference in the proportion of male offspring produced by *wt* and novel treatment males. Mean (±SE) of fitted values from the linear models. *wt:* black circle, *novel X:* triangle**,** *novel Y:* diamonds**.** The raw data is plotted as grey points. Letters indicates significance (Tukey HSD; $P < 0.05$). (B) Change in proportion of male offspring between the *wt* and the novel

populations (Δ = novel population - wild-type) with bars indicating bootstrap 95% confidence. *Novel X:* triangle *and novel Y:* diamonds**.** Asterisks indicate significance difference.

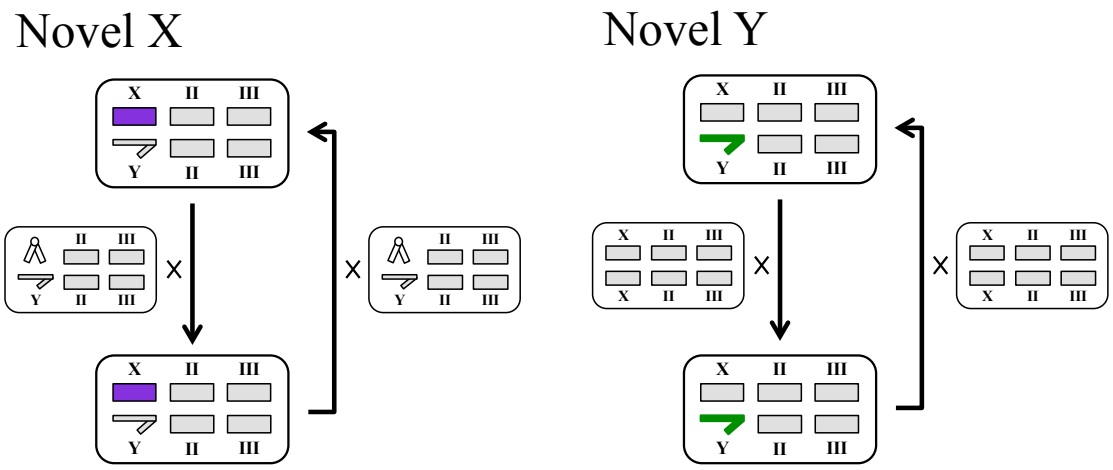

Fig. S11: The experimental setup for the evolution experiments for the novel populations. Autosomes (II and III) and chromosome X are depicted as rectangles, the double-X-chromosome is depicted as ⩓, and the chromosome Y as a half arrow. Only offspring used for the cross in the next generation is shown. For additional details see *Evolution experiment protocol*.

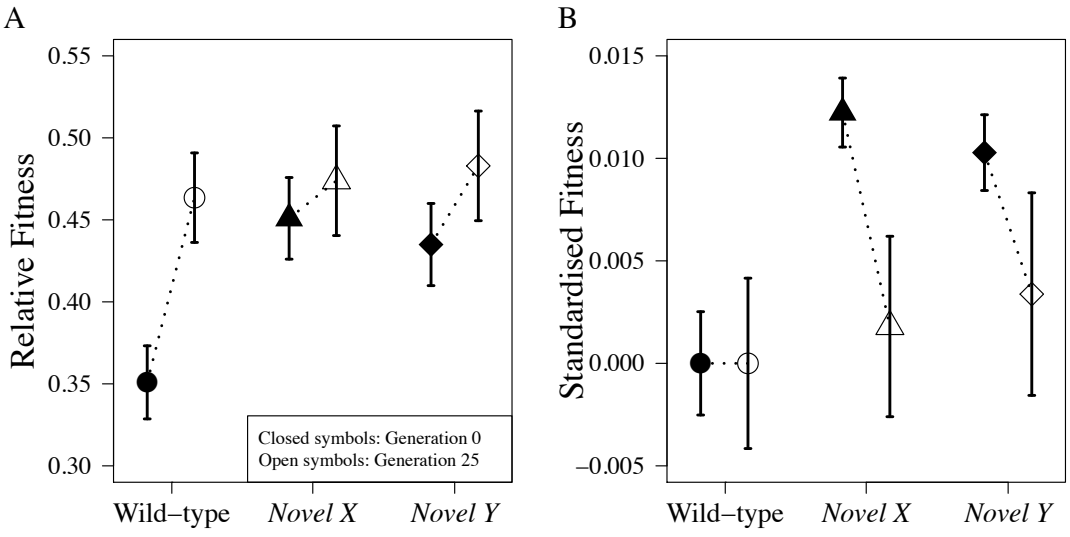

Fig. S12: Male reproductive fitness assays at generation 0 and generation 25. *wt* (circle), *novel X* (triangle), and *novel Y* (diamonds). Closed symbols: generation 0 and open symbols: generation 25. Mean (±SE). (A) Relative fitness. (B) Standardised fitness relative to the wild-type.

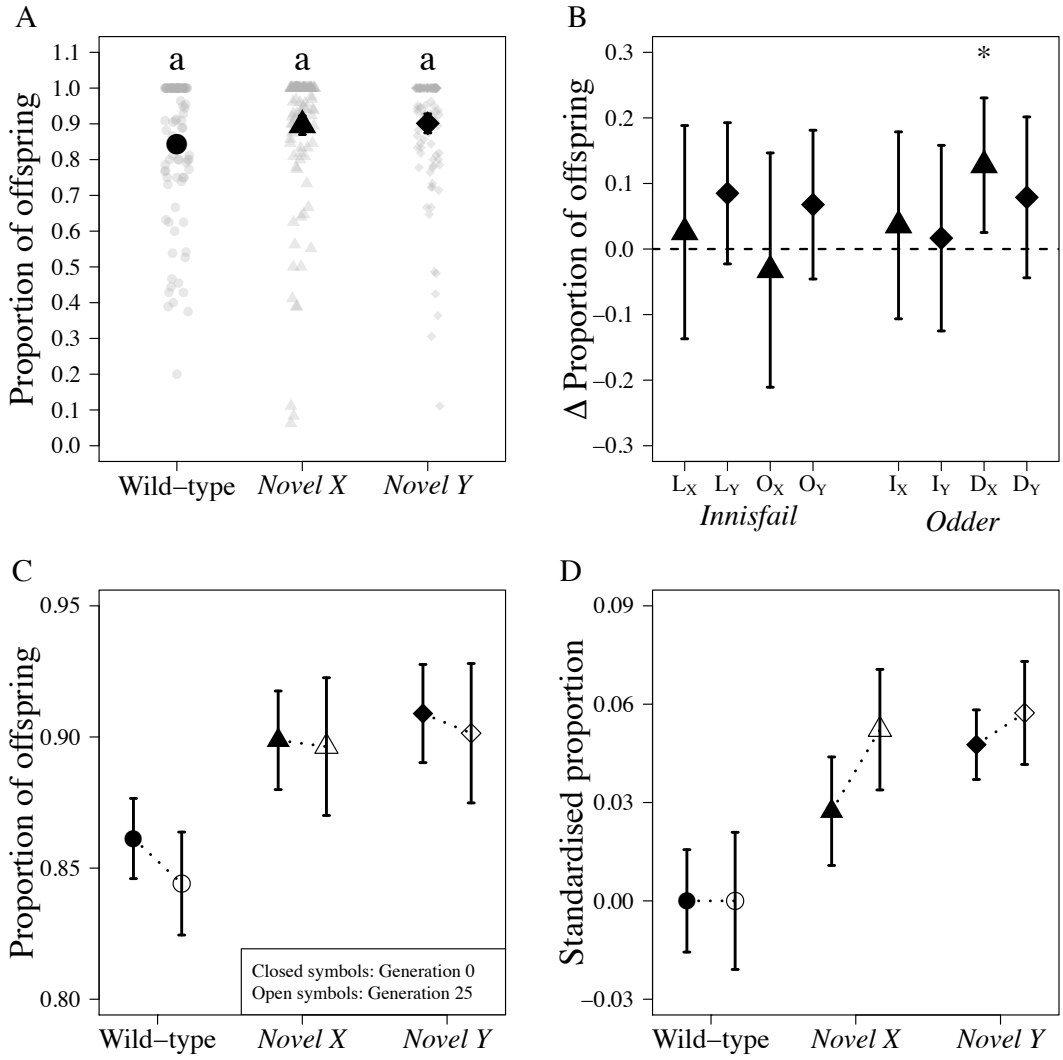

Fig. S13: Sperm competition at the end of the evolution experiment. **(**A) After 32 generations there was no longer a significant effect of *novel Y* males ability to replace sperm compared to the other two treatments. Mean (±SE) of fitted values from the linear models. *wt:* black circle, *novel X:* triangle**,** *novel Y:* diamonds**.** The raw data is plotted as grey points. Letters indicate significance (Tukey HSD; $P < 0.05$). (B) Change in proportion of offspring between the *wt* and the novel populations (Δ = novel population - wild-type) with bars indicating bootstrap 95% confidence. *Novel X:* triangle *and novel Y:* diamonds**.** Asterisks indicate significance difference. (C) Sperm competition at generation 0 and generation 25. (D) Standardised proportion of offspring relative to the wild-type. Mean (±SE). *wt* (circle), *novel X* (triangle), and *novel Y* (diamonds). Closed symbols: generation 0 and open symbols: generation 25.

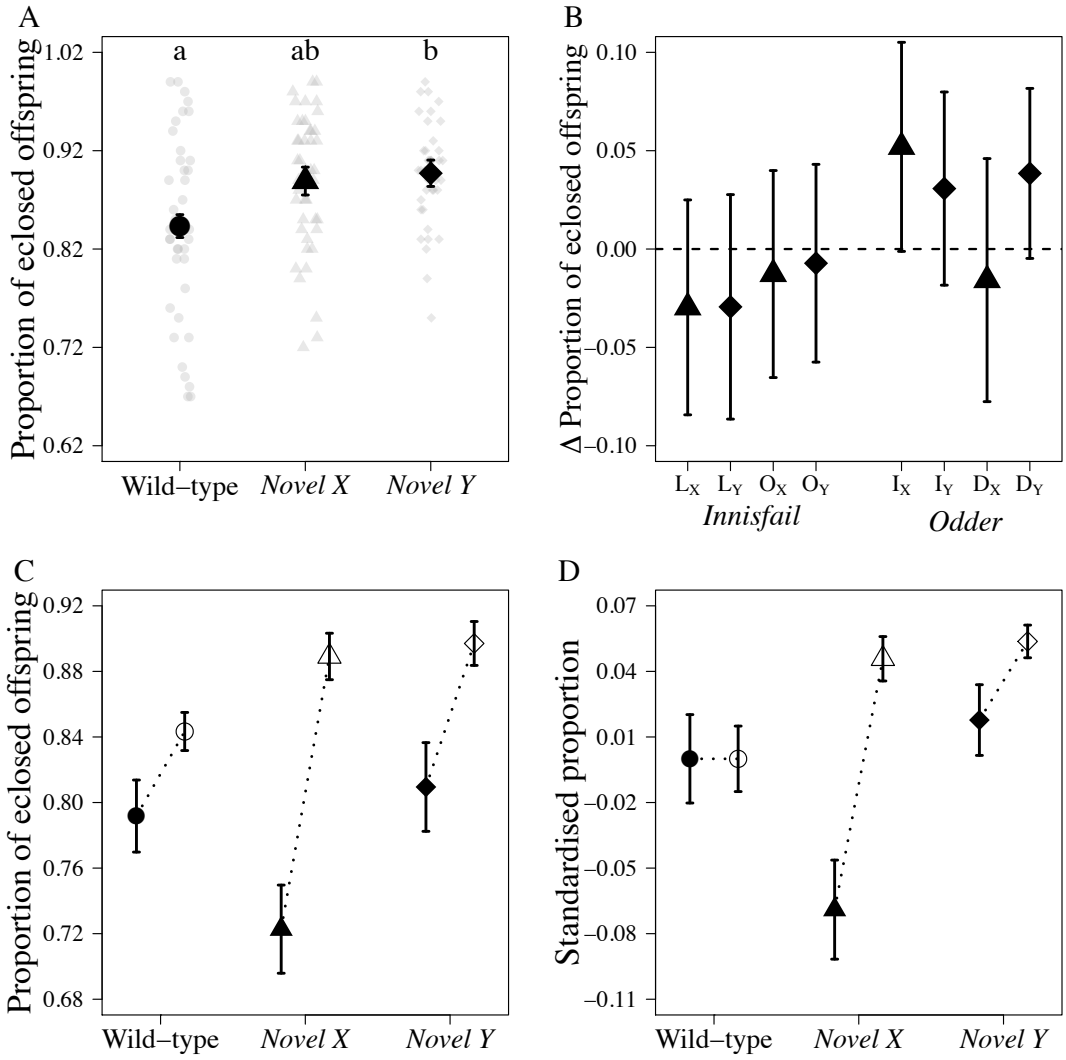

Fig. S14: Egg-to-adult offspring viability at the end of the evolution experiment. **(**A) After 39 generations there was no longer a significant effect of *novel X* males on offspring survival. Mean (±SE) of fitted values from the linear models. *wt:* black circle, *novel X:* triangle**,** *novel Y:* diamonds**.** The raw data is plotted as grey points. Letters indicate significance (Tukey HSD; $P < 0.05$). (B) Change in proportion of eclosed offspring between the *wt* and the novel populations (Δ = novel population - wild-type) with bars indicating bootstrap 95% confidence. *Novel X:* triangle *and novel Y:* diamonds**.** Asterisks indicate significance difference. (C) Egg-to-adult offspring viability at generation 0 and generation 25. (D) Standardised proportion of eclosed offspring relative to the wild-type. Mean (±SE). *wt* (circle), *novel X* (triangle), and *novel Y* (diamonds). Closed symbols: generation 0 and open symbols: generation 25.

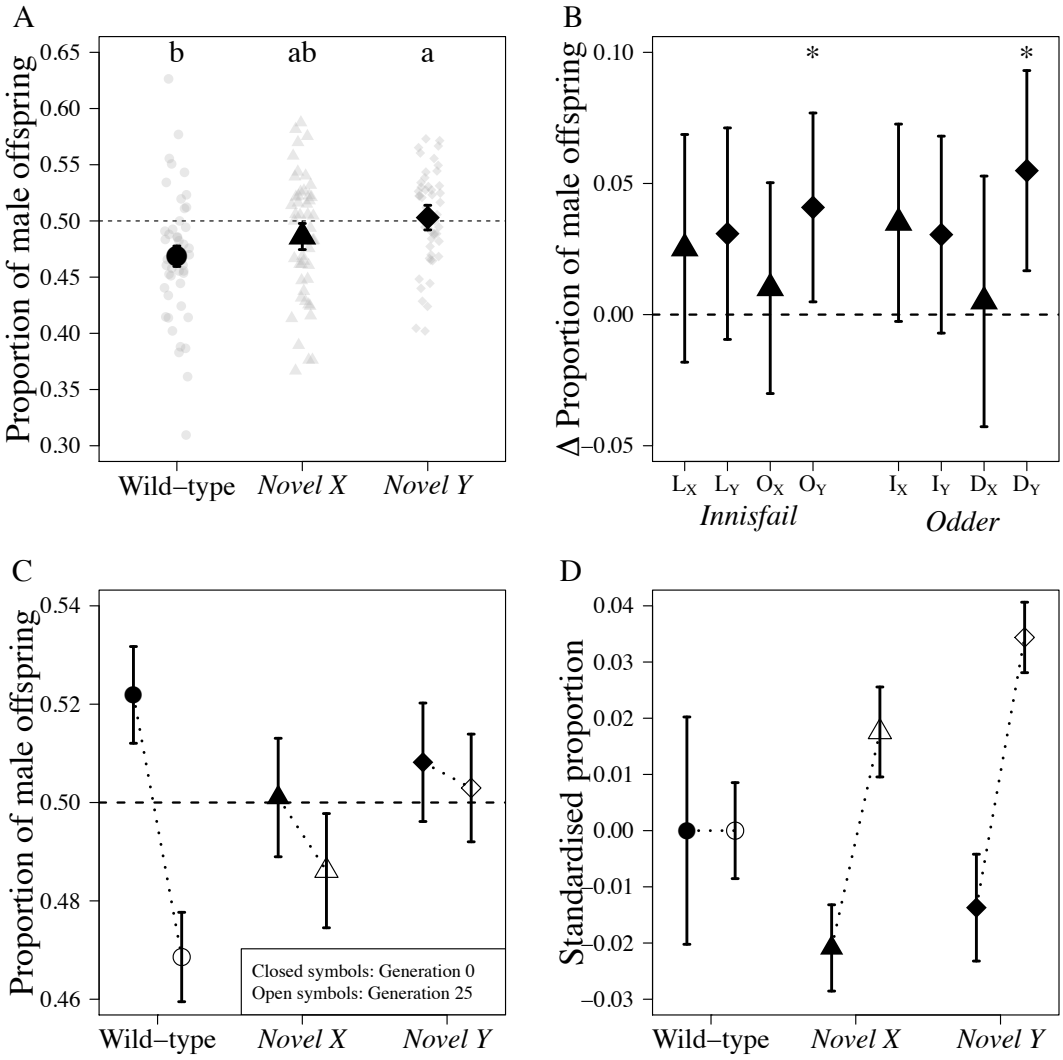

Fig. S15: Sex ratio at the end of the evolution experiment. **(**A) After 39 generations *novel Y* males produce significantly more male offspring than the *wt* treatment. Mean (±SE) of fitted values from the linear models. *wt:* black circle, *novel X:* triangle**,** *novel Y:* diamonds**.** The raw data is plotted as grey points. Letters indicate significance (Tukey HSD; $P < 0.05$). (B) Change in proportion of male offspring between the *wt* and the novel populations (Δ = novel population - wild-type) with bars indicating bootstrap 95% confidence. *Novel X:* triangle *and novel Y:* diamonds**.** Asterisks indicate significance difference. C) Sex ratio at generation 0 and generation 25. (D) Standardised proportion of male offspring relative to the wildtype. Mean (±SE). *wt* (circle), *novel X* (triangle), and *novel Y* (diamonds). Closed symbols: generation 0 and open symbols: generation 25.

Table S1: Summary of the results from ANOVA analysis of fitting linear models (thorax size and male effect on female fecundity) and linear mixed-effects models (male reproductive fitness, egg-to-adult offspring viability, sex ratio, sperm competition, and total offspring number).

Source	df	F	χ^2	P
Relative male fitness, *Novel X & Y*				
Treatment groups	2	8.50		$2.49e^{-04}$
Experimental block	1	0.16		0.69
Relative male fitness, *novel XY*				
Treatment groups	1	$5e^{-04}$		0.98
Thorax size				
Treatment groups	2	61.01		$<2.2e^{-16}$
Sperm competition, P2				
Treatment groups	2	3.59		*0.03*
Experimental block	1	3.05		0.08
Female longevity				
Treatment groups	2		4.4	0.1
Total number of offspring				
Treatment groups	2	4.10		*0.02*
Experimental block	1	0.16		0.69
Male induced female fecundity				
Treatment groups	2	1.75		0.18
Egg-to-adult viability				
Treatment groups	2	5.77		$4.11e^{-03}$
Experimental block	1	11.36		$1.02e^{-03}$
Sex ratio				
Treatment groups	2	1.55		0.22
Experimental block	1	0.10		0.75

Table S2: Summary of the results from the post-hoc Tukey test of analyses with a significant treatment effect in the linear models.

Source	P
Relative male fitness, *Novel X & Y*	
Novel X – wt	$2.13\ e^{-04}$
Novel Y – wt	$2.53e^{-03}$
Novel X – Novel Y	0.72
Thorax size	
Novel X – wt	$3.46e^{-11}$
Novel Y – wt	0.13
Novel X – Novel Y	$<2.2e^{-16}$
Sperm competition, P2	
Novel X – wt	0.12
Novel Y – wt	*0.03*
Novel X – Novel Y	0.85
Total number of offspring	
Novel X – wt	*0.01*
Novel Y – wt	0.29
Novel X – Novel Y	0.25
Egg-to-adult viability	
Novel X – wt	*0.03*
Novel Y – wt	0.79
Novel X – Novel Y	$4.94e^{-03}$

Table S3: Comparison of the relative fitness of *novel X* and *novel Y* populations within each population cross (Student's t-test). P value is adjusted for multiple testing. Capital letters indicate which population the novel sex chromosome originates from (D: *Dahomey*, I: *Innisfail*, L: LH_M, O: *Odder*) and the novel sex chromosome is denoted by X or Y subscripts.

Crosses	Treatment	Genotype	Mean	df	t	P value
LH_M-Inn	Novel X	L-I_X/XYAA	0.47	46.89	-2.13	0.58
		I-L_X/XYAA				
	Novel Y	L-I_Y/XYAA	0.57			
		I-L_Y/XYAA				
Inn-Odd	Novel X	I-O_X/XYAA	0.52	53.22	1.99	0.77
		O-I_X/XYAA				
	Novel Y	I-O_Y/XYAA	0.44			
		O-I_Y/XYAA				
Odd-Dah	Novel X	O-D_X/XYAA	0.46	46.49	0.72	1.00
		D-O_X/XYAA				
	Novel Y	O-D_Y/XYAA	0.43			
		D-O_Y/XYAA				
Dah-Tas	Novel X	D-T_X/XYAA	0.40	53.30	1.30	1.00
		T-D_X/XYAA				
	Novel Y	D-T_Y/XYAA	0.35			
		T-D_Y/XYAA				
Tas-LH_M	Novel X	T-L_X/XYAA	0.40	39.01	0.51	1.00
		L-T_X/XYAA				
	Novel Y	T-L_Y/XYAA	0.38			
		L-T_Y/XYAA				

Table S4: Comparison of the relative fitness of novel populations with the same novel sex chromosome pair but different autosomes within each population cross (Student's t-test). P value is adjusted for multiple testing. Capital letters indicate which population the novel sex chromosome originates from (D: *Dahomey*, I: *Innisfail*, L: LH_M, O: *Odder*) and the novel sex chromosome is denoted by X or Y subscripts.

Crosses	Genotype	Mean	df	t	P value
LH_M-Inn	L-I_Y/XYAA	0.61	21.47	-1.93	1.00
	I-L_X/XYAA	0.48			
	L-I_X/XYAA	0.48	24.47	-1.01	1.00
	I-L_Y/XYAA	0.53			
Inn-Odd	I-O_Y/XYAA	0.42	25.52	0.94	1.00
	O-I_X/XYAA	0.47			
	I-O_X/XYAA	0.58	24.74	1.88	1.00
	O-I_Y/XYAA	0.46			
Odd-Dah	O-D_Y/XYAA	0.42	25.97	-1.84	1.00
	D-O_X/XYAA	0.33			
	O-D_X/XYAA	0.59	24.49	2.53	0.28
	D-O_Y/XYAA	0.44			
Dah-Tas	D-T_Y/XYAA	0.35	21.98	0.99	1.00
	T-D_X/XYAA	0.41			
	D-T_X/XYAA	0.38	23.93	0.81	1.00
	T-D_Y/XYAA	0.34			
Tas-LH_M	T-L_Y/XYAA	0.32	17.55	1.68	1.00
	L-T_X/XYAA	0.42			
	T-L_X/XYAA	0.39	21.03	-0.65	1.00
	L-T_Y/XYAA	0.43			

Table S5: The change in relative fitness with confidence intervals (CI) calculated by bootstrap for each of the novel populations. The populations marked in bold are significantly different from zero. Annotation for the novel populations is genetic background – origin of sex chromosome (X or Y). D: *Dahomey*, I: *Innisfail*, L: LH_M, O: *Odder*, and T: *Tasmania*.

	ΔFitness	95 % CI
Novel X		
LH_M–T_X	-0.05	-0.15; 0.05
LH_M–I_X	-0.0007	-0.10; 0.10
***Innisfail*–L_X**	**0.16**	**0.08; 0.25**
***Innisfail*–O_X**	**0.27**	**0.17; 0.37**
***Odder*–I_X**	**0.15**	**0.06; 0.23**
***Odder*–D_X**	**0.27**	**0.16; 0.38**
Dahomey–O_X	-0.008	-0.11; 0.09
Dahomey–T_X	0.04	-0.06; 0.14
Tasmania–D_X	0.09	-0.07; 0.26
Tasmania–L_X	0.07	-0.08; 0.21
Novel Y		
LH_M–T_Y	-0.04	-0.16; 0.08
LH_M–I_Y	**0.14**	**0.004; 0.28**
***Innisfail*–L_Y**	**0.22**	**0.12; 0.32**
***Innisfail*–O_Y**	**0.11**	**0.02; 0.20**
***Odder*–I_Y**	**0.14**	**0.05; 0.23**
***Odder*–D_Y**	**0.10**	**0.01; 0.18**
Dahomey–O_Y	0.10	-0.007; 0.21
Dahomey–T_Y	0.02	-0.08; 0.12
Tasmania–D_Y	0.02	-0.14; 0.17
Tasmania–L_Y	0.004	-0.16; 0.16

Table S6: Summary of the results from ANOVA analysis of linear mixed-effects models for the evolved populations.

Source	df	F	P
Relative fitness for *Novel X & Y*			
Treatment groups	2	0.17	0.85
Experimental block	1	29.28	$2.65e^{-07}$
Nested factor	1		1
Sperm competition, P2			
Treatment groups	2	2.78	0.06
Nested factor	1		1
Egg-to-adult viability			
Treatment groups	2	9.50	$1.47e^{-04}$
Experimental block	3	17.45	$1.72e^{-09}$
Nested factor	1		1
Sex ratio			
Treatment groups	2	4.85	$9.22e^{-03}$
Experimental block	3	0.31	0.82
Nested factor	1		1

Table S7: The change in relative fitness between the novel evolved populations and the wild-type background with confidence intervals (CI) calculated by bootstrap. The population marked in bold is significantly different from zero. Annotation for the novel populations is genetic background – origin of sex chromosome (X or Y). D: *Dahomey*, I: *Innisfail*, L: LH_M, and O: *Odder*.

	ΔFitness	95 % CI
Novel X		
Innisfail–L_X	0.07	-0.04; 0.19
***Innisfail*–O_X**	**0.14**	**0.02; 0.27**
Odder–I_X	-0.05	-0.18; 0.09
Odder–D_X	0.03	-0.09; 0.15
Novel Y		
Innisfail–L_Y	0.09	-0.06; 0.23
Innisfail–O_Y	0.10	-0.03; 0.23
Odder–I_Y	0.02	-0.13; 0.16
Odder–D_Y	0.04	-0.09; 0.16

Table S8: Summary of the results from the post-hoc Tukey test of analyses with a significant treatment effect in the linear mixed-effects model.

Source	P
Egg-to-adult viability	
Novel X – wt	0.10
Novel Y – wt	*<0.001*
Novel X – Novel Y	0.09
Sex ratio	
Novel X – wt	0.39
Novel Y – wt	$5.51e^{-03}$
Novel X – Novel Y	0.23

Supplementary Material (Appendices B & C) for: Sexually antagonistic coevolution between the sex chromosomes of *Drosophila melanogaster*. *PNAS*

Katrine K. Lund-Hansen[a], Colin Olito[a], Edward H. Morrow[b,c], & Jessica K. Abbott[a]

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[a] Department of Biology, Section for Evolutionary Ecology, Lund University, Lund 223 62, Sweden.
[b] School of Life Sciences, University of Sussex, Brighton BN1 9QG, United Kingdom.
[c] Department for Environmental and Life Sciences, Karlstad University, 651 88, Karlstad, Sweden.
* Corresponding author e-mail: katrine.lund-hansen@biol.lu.se

Appendix B: Development of the recursions

Our models differ from standard selection models in two important ways: (i) mating is non-random (influenced by males' genotype at **Y**, and (ii) the relative fitness of offspring depends on the parental genotypes rather than their own (i.e., selection in the current generation depends on genotypic frequencies among parents in the previous generation). Here, we develop the recursion equations underlying the deterministic version of the Autosomal and X-linked models described in the main text, as well as the eigenvalues for the relevant boundary equilibria used in subsequent analyses. For the deterministic models below, we assume sufficiently large population sizes that drift can be ignored, in addition to the major assumptions outlined in the main text.

The Autosomal Model

When the compensatory locus is autosomal, we can model the dynamics of the two interacting loci by tracking the frequency of mutant y genotype, q_y, and each of the three autosomal genotypes at the compensatory locus (F_{AA}, F_{Aa}, F_{aa}). We begin with the recursion for the Y-linked locus, **Y**.

Let $(1-q_y)$ and q_y equal the frequency of the wild-type (Y) and mutant (y) genotype among adult males prior to mating, with w_Y^m and w_y^m denoting the relative mating success of the two genotypes respectively. The genotypic frequencies among male gametes at mating are then

$$(1-q_y^m) = \frac{(1-q_y)w_Y^m}{\overline{w}_m} \tag{B1a}$$

$$q_y^m = \frac{q_y w_y^m}{\overline{w}_m}, \tag{B1b}$$

where $\overline{w}_m = (1-q_y)w_Y^m + q_y w_y^m$ is the average fitness of both genotypes with respect to mating success. The resulting genotypic frequencies among offspring after fertilization and viability selection depends on the parent genotypes. We now write the frequency of the three possible genotypes at the autosomal compensatory locus as F_{AA}, F_{Aa}, and F_{aa}. Let $w^o_{Y:AA}$, $w^o_{Y:Aa}$, and $w^o_{Y:aa}$ denote the relative fitness of offspring sired by a wild-type male with each of the three possible female genotypes, with $w^o_{y:AA}$, $w^o_{y:Aa}$, and $w^o_{y:aa}$ representing the same for matings involving mutant males. The genotypic frequencies among offspring after fertilization and selection are then

$$(1-q'_y) = \frac{(1-q_y^m)(F_{AA}w^o_{Y:AA} + F_{Aa}w^o_{Y:Aa} + F_{aa}w^o_{Y:aa})}{\overline{w}_o^{\mathbf{Y}}} \tag{B2a}$$

$$q'_y = \frac{q_y^m(F_{AA}w^o_{y:AA} + F_{Aa}w^o_{Y:Aa} + F_{aa}w^o_{Y:aa})}{\overline{w}_o^{\mathbf{Y}}}, \tag{B2b}$$

where $\overline{w}_o^{\mathbf{Y}}$ represents mean offspring fitness, and is equal to the sum of the numerators of Eq(B2a, B2b).

Recalling that q_y^m denotes the frequency of the mutant y genotype among male gametes, the corresponding recursions for the compensatory locus, **A**, are

$$F'_{AA} = \frac{(1-q_y^m)\left(F_{AA}w^o_{Y:AA} + \frac{F_{Aa}}{2}w^o_{Y:Aa}\right)(1-q_a) + q_y^m\left(F_{AA}w^o_{y:AA} + \frac{F_{Aa}}{2}w^o_{y:Aa}\right)(1-q_a)}{\overline{w}_o^{\mathbf{A}}} \tag{B3a}$$

$$F'_{Aa} = \Bigg((1-q_y^m)\left[\left(F_{AA}w^o_{Y:AA} + \frac{F_{Aa}}{2}w^o_{Y:Aa}\right)q_a + \left(F_{aa}w^o_{Y:aa} + \frac{F_{Aa}}{2}w^o_{Y:Aa}\right)(1-q_a)\right] + q_y^m\left[\left(F_{AA}w^o_{y:AA} + \frac{F_{Aa}}{2}w^o_{y:Aa}\right)q_a + \left(F_{aa}w^o_{y:aa} + \frac{F_{Aa}}{2}w^o_{y:Aa}\right)(1-q_a)\right]\Bigg) \Big/ \overline{w}_o^{\mathbf{A}} \tag{B3b}$$

$$F'_{aa} = \frac{(1-q_y^m)\left(F_{aa}w^o_{Y:aa} + \frac{F_{Aa}}{2}w^o_{Y:Aa}\right)q_a + q_y^m\left(F_{aa}w^o_{y:aa} + \frac{F_{Aa}}{2}w^o_{y:Aa}\right)q_a}{\overline{w}_o^{\mathbf{A}}}, \tag{B3c}$$

where $q_a = F_{aa} + F_{Aa}/2$, and $\overline{w}_o^{\mathbf{A}}$ is the sum of the numerators of Eqs(B3a-c).

Invasion of a mutant y chromosome

During the initial stage of a coevolutionary cycle (invasion and subsequent increase in the frequency of y to fixation in a population fixed for the wild-type compensatory allele), the evolutionary dynamics of the **Y** locus are independent of the genomic location of the compensatory locus. Substituting the fitness expressions defined in Table 1 of the main text, the recursion equation for the expected frequency of the mutant y allele in the next generation for a population initially fixed for the wild-type compensatory allele ($q_a = 0$) reduces to:

$$q'_y = \frac{q_y(1+s_m)(1-s_o)}{1+q_y(s_m(1-s_o)-s_o)}. \tag{B4}$$

From Eq(B4), we see that the mutant y chromosome can invade when $s_m - s_o(1+s_m) > 0$. Under weak selection (dropping terms in $s_m s_o$), we see clearly that invasion of y requires that $\delta = (s_m - s_o) > 0$, the increase in male mating success is greater than the loss of offspring viability relative to wild-type males.

Invasion of autosomal compensatory mutation

To analyze the linear stability of the system of recursion equations, Eqs(B2b, B3a-c) at specific equilibria, we first define the Jacobian matrix:

$$\mathbb{J}_{\mathrm{A}} = \begin{pmatrix} \frac{\partial q'_y}{\partial q_y} & \frac{\partial q'_y}{\partial F_{AA}} & \frac{\partial q'_y}{\partial F_{Aa}} & \frac{\partial q'_y}{\partial F_{aa}} \\ \frac{\partial F'_{AA}}{\partial q_y} & \frac{\partial F'_{AA}}{\partial F_{AA}} & \frac{\partial F'_{AA}}{\partial F_{Aa}} & \frac{\partial F'_{AA}}{\partial F_{aa}} \\ \frac{\partial F'_{Aa}}{\partial q_y} & \frac{\partial F'_{Aa}}{\partial F_{AA}} & \frac{\partial F'_{Aa}}{\partial F_{Aa}} & \frac{\partial F'_{Aa}}{\partial F_{aa}} \\ \frac{\partial F'_{aa}}{\partial q_y} & \frac{\partial F'_{aa}}{\partial F_{AA}} & \frac{\partial F'_{aa}}{\partial F_{Aa}} & \frac{\partial F'_{aa}}{\partial F_{aa}} \end{pmatrix} \tag{B5}$$

If a mutant y chromosome sweeps to fixation prior to the occurrance of a compensatory mutation, completion of a coevolutionary cycle requires that a mutant a allele invades a population initially fixed for y ($q_y = 1$). To determine the conditions for invasion of a into a population initially fixed for y, we evaluated the candidate leading eigenvalue associated with invasion at $\mathbf{A}$, $\lambda_{\mathbf{A}}$ at the boundary equilibrium where $q_y = 1$ and $q_a = 0$.

$$\lambda_{\mathbf{A}}|_{\hat{q}_y=1,\hat{q}_a=0} = \frac{(2 - s_o - h_o s_o)}{(2(1 - s_o))}. \tag{B6}$$

Solving $\lambda_{\mathbf{A}}|_{\hat{q}_y=1,\hat{q}_a=0} - 1 > 0$ yields the intuitive conclusion that invasion of the compensatory allele requires only that there is selection against the wild-type allele (i.e., $h_o < 1$ and $0 < s_o < 1$).

On the other hand, if a compensatory mutation arises before the mutant y chromosome has fixed in the population, we must determine the conditions for invasion of a mutant a allele into a population where q_y is unspecified. Given these initial frequencies, and the fitness expressions provided in Table 1,

$$\lambda_{\mathbf{A}}|_{\hat{q}_a=0} = \frac{2(q_y s_m + 1) - h_c s_c (1 - q_y) - q_y s_o (1 + h_o)(1 + s_m)}{2q_y(s_m(1 - s_o) - s_o) + 2}. \tag{B7}$$

Solving $\lambda_{\mathbf{A}}|_{\hat{q}_a=0} - 1 > 0$ for q_y yields the threshold frequency of the mutant y allele at which selection begins to favour the invasion of the a allele

$$\tilde{q}_y^{\mathrm{A}} = \frac{h_c s_c}{h_c s_c + (s_m - \delta)(1 - h_o)(1 + s_m)}. \tag{B8}$$

The X-linked Model

When the compensatory locus is located on the X chromosome, it is necessary to track the genotypic frequencies of all seven possible pairings of the sex chromosomes: XY, Xy, xY, and xy for males, and XX, Xx, and xx for females. The frequencies of the relevant chromosomes sex chromosomes among male gamete contributed by each male genotype are

$$F_{XY}^g = \frac{F_{XY} w_Y^m}{\overline{w}_m} \tag{B9a}$$

$$F_{Xy}^g = \frac{F_{Xy} w_y^m}{\overline{w}_m} \tag{B9b}$$

$$F_{xY}^g = \frac{F_{xY} w_Y^m}{\overline{w}_m} \tag{B9c}$$

$$F_{xy}^g = \frac{F_{xy} w_y^m}{\overline{w}_m}, \tag{B9d}$$

where, $\overline{w}_m$ is now the sum of the numerators of Eqs(B9a). For simplicity, we also define the overall frequency of the Y and y chromosomes among male gametes as

$$F_{\cdot Y}^g = \frac{(F_{XY} + F_{xY}) w_Y^m}{\overline{w}_m} \tag{B10a}$$

$$F_{\cdot y}^g = \frac{(F_{Xy} + F_{xy}) w_y^m}{\overline{w}_m}. \tag{B10b}$$

The genotypic frequencies among male offspring after fertilization and selection can now be described by the following system of recursions

$$F'_{XY} = \frac{F^g_{\cdot Y}\left(F_{XX}w^o_{Y:XX} + \frac{F_{Xx}}{2}w^o_{Y:Xx}\right)}{\overline{w}^{\mathbf{X}}_m} \tag{B11a}$$

$$F'_{Xy} = \frac{F^g_{\cdot y}\left(F_{XX}w^o_{y:XX} + \frac{F_{Xx}}{2}w^o_{y:Xx}\right)}{\overline{w}^{\mathbf{X}}_m} \tag{B11b}$$

$$F'_{xY} = \frac{F^g_{\cdot Y}\left(F_{xx}w^o_{Y:xx} + \frac{F_{Xx}}{2}w^o_{Y:Xx}\right)}{\overline{w}^{\mathbf{X}}_m} \tag{B11c}$$

$$F'_{xy} = \frac{F^g_{\cdot y}\left(F_{xx}w^o_{y:xx} + \frac{F_{Xx}}{2}w^o_{y:Xx}\right)}{\overline{w}^{\mathbf{X}}_m}, \tag{B11d}$$

where $\overline{w}^{\mathbf{X}}_m$ is the sum of the numerators of Eqs(B11a-d). The genotypic frequencies among female offspring after fertilization and selection are then

$$F'_{XX} = \frac{F^g_{XY}\left(F_{XX}w^o_{Y:XX} + \frac{F_{Xx}}{2}w^o_{Y:Xx}\right) + F^g_{Xy}\left(F_{XX}w^o_{y:XX} + \frac{F_{Xx}}{2}w^o_{y:Xx}\right)}{\overline{w}^{\mathbf{X}}_f} \tag{B12a}$$

$$\begin{aligned} F'_{Xx} = \frac{1}{\overline{w}^{\mathbf{X}}_f}\Bigg[&F^g_{XY}\left(F_{xx}w^o_{Y:xx} + \frac{F_{Xx}}{2}w^o_{Y:Xx}\right) + F^g_{xY}\left(F_{XX}w^o_{Y:XX} + \frac{F_{Xx}}{2}w^o_{Y:Xx}\right) + \\ &F^g_{Xy}\left(F_{xx}w^o_{y:XX} + \frac{F_{Xx}}{2}w^o_{y:Xx}\right) + F^g_{xy}\left(F_{XX}w^o_{y:XX} + \frac{F_{Xx}}{2}w^o_{y:Xx}\right)\Bigg] \end{aligned} \tag{B12b}$$

$$F'_{xx} = \frac{F^g_{xY}\left(F_{xx}w^o_{Y:xx} + \frac{F_{Xx}}{2}w^o_{Y:Xx}\right) + F^g_{xy}\left(F_{xx}w^o_{y:xx} + \frac{F_{Xx}}{2}w^o_{y:Xx}\right)}{\overline{w}^{\mathbf{X}}_f}, \tag{B12c}$$

Invasion of mutant y an X-linked compensatory mutation

To analyze the linear stability of the system of recursion equations, Eqs(B11a-d, B12a-c), at specific equilibria, we first define the Jacobian matrix:

$$\mathbb{J}_{\mathrm{X}} = \begin{pmatrix} \frac{\partial F'_{XY}}{\partial F_{XY}} & \frac{\partial F'_{XY}}{\partial F_{Xy}} & \frac{\partial F'_{XY}}{\partial F_{xY}} & \frac{\partial F'_{XY}}{\partial F_{xy}} & \frac{\partial F'_{XY}}{\partial F_{XX}} & \frac{\partial F'_{XY}}{\partial F_{Xx}} & \frac{\partial F'_{XY}}{\partial F_{xx}} \\ \frac{\partial F'_{Xy}}{\partial F_{XY}} & \frac{\partial F'_{Xy}}{\partial F_{Xy}} & \frac{\partial F'_{Xy}}{\partial F_{xY}} & \frac{\partial F'_{Xy}}{\partial F_{xy}} & \frac{\partial F'_{Xy}}{\partial F_{XX}} & \frac{\partial F'_{Xy}}{\partial F_{Xx}} & \frac{\partial F'_{Xy}}{\partial F_{xx}} \\ \frac{\partial F'_{xY}}{\partial F_{XY}} & \frac{\partial F'_{xY}}{\partial F_{Xy}} & \frac{\partial F'_{xY}}{\partial F_{xY}} & \frac{\partial F'_{xY}}{\partial F_{xy}} & \frac{\partial F'_{xY}}{\partial F_{XX}} & \frac{\partial F'_{xY}}{\partial F_{Xx}} & \frac{\partial F'_{xY}}{\partial F_{xx}} \\ \frac{\partial F'_{xy}}{\partial F_{XY}} & \frac{\partial F'_{xy}}{\partial F_{Xy}} & \frac{\partial F'_{xy}}{\partial F_{xY}} & \frac{\partial F'_{xy}}{\partial F_{xy}} & \frac{\partial F'_{xy}}{\partial F_{XX}} & \frac{\partial F'_{xy}}{\partial F_{Xx}} & \frac{\partial F'_{xy}}{\partial F_{xx}} \\ \frac{\partial F'_{XX}}{\partial F_{XY}} & \frac{\partial F'_{XX}}{\partial F_{Xy}} & \frac{\partial F'_{XX}}{\partial F_{xY}} & \frac{\partial F'_{XX}}{\partial F_{xy}} & \frac{\partial F'_{XX}}{\partial F_{XX}} & \frac{\partial F'_{XX}}{\partial F_{Xx}} & \frac{\partial F'_{XX}}{\partial F_{xx}} \\ \frac{\partial F'_{Xx}}{\partial F_{XY}} & \frac{\partial F'_{Xx}}{\partial F_{Xy}} & \frac{\partial F'_{Xx}}{\partial F_{xY}} & \frac{\partial F'_{Xx}}{\partial F_{xy}} & \frac{\partial F'_{Xx}}{\partial F_{XX}} & \frac{\partial F'_{Xx}}{\partial F_{Xx}} & \frac{\partial F'_{Xx}}{\partial F_{xx}} \\ \frac{\partial F'_{xx}}{\partial F_{XY}} & \frac{\partial F'_{xx}}{\partial F_{Xy}} & \frac{\partial F'_{xx}}{\partial F_{xY}} & \frac{\partial F'_{xx}}{\partial F_{xy}} & \frac{\partial F'_{xx}}{\partial F_{XX}} & \frac{\partial F'_{xx}}{\partial F_{Xx}} & \frac{\partial F'_{xx}}{\partial F_{xx}} \end{pmatrix} \tag{B13}$$

Analyzing the leading eigenvalue of $\mathbb{J}$ evaluated at the boundary equilibrium corresponding to initiation of a coevolutionary cycle (i.e., $q_y = 0$, $q_x = 1$) recovers the same invasion criteria as in the autosomal model ($\delta > 0$).

To determine when selection will favour invasion of the compensatory x allele, we evaluated the candidate leading eigenvalue associated with corresponding invasion at $\mathbf{X}$, $\lambda_{\mathbf{X}}$ at the initial equilibrium where $q_x = 0$, and the q_y is unspecified. Given these initial frequencies, and the fitness expressions provided in Table 1, $\lambda_{\mathbf{X}}|_{\hat{q}_x=0}$ is a large polynomial expression (see the accompanying Mathematica notebook in Appendix X, where this result is derived). However, under additive fitness effects at $\mathbf{X}$ ($h_c = h_o = 1/2$), we can still solve $\lambda_{\mathbf{X}}|_{\hat{q}_x=0} - 1 = 0$ for q_y to give the threshold frequency of the mutant y chromosome at which selection will begin to favour the mutant x compensatory allele. Although the solution is still complicated, it follows the general form

$$\tilde{q}_y^{\mathrm{X}} = \frac{b - \sqrt{b^2 - 8s_c c}}{2c}, \tag{B14}$$

where

$$b = s_m(2 + s_m(2 - s_m(1 - s_m))) - \delta(2 + s_m + s_m^2 + 2s_m^3) + \delta^2(1 + s_m)^2 + s_c(2 + s_m^2 - \delta(1 + s_m)) \tag{B15a}$$

$$c = (s_c + s_m + 3s_m^2 - 3\delta(1 + s_m))(s_m^2 - \delta(1 + s_m)). \tag{B15b}$$

Appendix C: Models of conflict and compensation when male traits affect mating opportunities rather than offspring survival

As noted in the main text, there are three different ways that a Y-linked male-beneficial allele could harm females and possibly generate sexual conflict: (*a*) the allele could recombine onto the X and harm its carriers; (*b*) the allele could harm offspring; or (*c*) the allele could cause males to limit females' mating opportunities (*e.g.*, by harming them). Explanation (*a*) can be discounted for our study because recombination does not occur in *Drosophila melanogaster* males and there is no pseudo-autosomal region on the sex chromosomes; hence there is no opportunity for a Y-linked allele to recombine onto an X chromosome. Explanation (*b*) leads to reduced fitness for both mothers and fathers (creating the conflict), and is what was modeled in the main text and Appendix B. This scenario appears most consistent with our empirical findings that *novel* males enjoyed higher fertilization success while offspring sired by novel-X males had a significantly lower egg-to-adult survival rate. Although not consistent with our empirical findings, explanation (*c*) could also cause sexual conflict. Explanations (*b*) and (*c*) potentially differ in their effects on males and females. Below, we present theoretical models corresponding to explanation (*c*), and a brief analysis to demonstrate key similarities and differences with the models presented in the main text.

Outline of the models

There are many possible ways to model sexual conflict over mating opportunities. Here we present a simple scenario that is roughly consistent with the basic biological details of mating in *D. melanogaster*. As outlined below, the key difference between these models, and those presented in the main text is the structure of the life-cycle, and the phenotypic effects of the mutations.

Here, we assume a simple life cycle in which all adults mate twice within each generation, and no other bouts of selection occur between fertilization and adulthood/mating (*i.e.*, the life-cycle proceeds: *fertilization* $\rightarrow 1^{\text{st}}$ *mating* ($M1$) $\rightarrow 2^{\text{nd}}$ *mating* ($M2$) $\rightarrow$ *fertilization*). For simplicity, we assume that all adults participate in both matings unless prevented from doing so, that females receive enough sperm from any single mating to fertilize all of her eggs, and that females exhibit no mating preference for males based on genotype. We make two additional assumptions based on well documented empirical patterns in *D. melanogaster* lab populations: (*i*) the second male to mate with a given female is able to displace sperm from her previous mate, enabling him to fertilize a proportion, L, of her eggs (7, 5); and (*ii*) female fecundity is increased by multiple mating (*e.g.*, due to effects of seminal fluid proteins; Herndon and Wolfner 4), resulting in increased egg production during $M2$ by a factor, E.

We model the same two alternative genetic systems described in the main text: a Y-linked locus (**Y**, with alleles Y, and y), and a compensatory locus located on either an autosome (**A**, with alleles A, and a) or the X chromosome (**X**, with alleles X, and x) (the Autosomal and X-linked models respectively), in a large population with discrete generations.

The phenotypic effects of the alternative alleles at the two loci influence females' ability to participate in $M2$. For example, suppose that males carrying the mutant y allele express some coercive trait or behavior (*e.g.*, mate harming, delivering a sperm plug, ejaculate proteins, etc.) such that any female they mate with during $M1$ can only participate in $M2$ at a reduced rate, ρ_{ii} (where the subscript ii refers to the female's

genotype at the compensatory locus). Matings involving parental genotypes $[y : AA]$, $[y : Aa]$, and $[y : aa]$ result in female remating rates of $\rho_{AA} = 1 - p$, $\rho_{Aa} = 1 - h_p p$, and $\rho_{aa} = 1$ respectively, where p is the proportion of females that are prevented from participating in $M2$. The benefit to males of carrying the mutant y chromosome arises when they mate with a wild-type female (one carrying no compensatory alleles) during $M1$, in which case he ensures fertilization of the WT female's eggs by preventing her from mating with a different male in $M2$ that would displace his sperm.

The cost to females of mating with a mutant y male is lost mating opportunities. Specifically, because female egg production in $M2$ is larger than in $M1$ by the factor E, females that are prevented from participating in $M2$ lose these additional opportunities to pass their genes on to the next generation. Males also lose the opportunity to secure these additional fertilizations, but it does not affect the relative fitness of the mutant y because both mutant and wild-type males lose an equal number of possible fertilizations.

Table C1 below summarizes the above effects for all possible combinations of mating pair genotypes. We refer readers to the Mathematica (.nb) file available in the online supplementary material for the full system of recursion equations, as well as additional analyses.

Table C1: Summary of phenotypic and fitness effects resulting from all possible combinations of mating pair genotypes at **Y** and the compensatory locus **A** (or **X**).

Female re-mating rate (ρ_{ii})

	Female Genotype		
Male **Y** genotype	AA, XX	Aa, Xx	aa, xx
Y	1	1	1
y	$1 - p$	$1 - h_p p$	1

Proportion of eggs fertilized by males

First Mating ($M1$)			
Y	$(1 - L)$	$(1 - L)$	$(1 - L)$
y	$(1 - \rho_{AA}) + (1 - L)\rho_{AA}$	$(1 - \rho_{Aa}) + (1 - L)\rho_{Aa}$	$(1 - \rho_{aa}) + (1 - L)\rho_{aa}$
Second Mating ($M2$)			
Y	L	L	L
y	$L\rho_{AA}$	$L\rho_{Aa}$	$L\rho_{aa}$

Subscripted ii's denote female genotype at the compensatory locus **A** (or **X**).

Analysis

To identify the conditions under which antagonistic coevolution between the **Y** and **A** (or **X**) loci can occur, and what conditions are necessary for major differences to arise between the X-linked and Autosomal models, we focus our analyses on the evolutionary invasion of rare mutants at each locus individually. Specifically, we analyze the eigenvalues of the Jacobian matrix of the system of equations for the Autosomal and X-linked models, evaluated at some initial equilibrium (6). In parallel with the theoretical results presented in the main text, we focus on three initial equilibria corresponding to key points in a bout of coevolution between the two loci: (*i*) invasion of the mutant y into a population initially fixed for the wild-type allels at both loci; (*ii*) invasion of the compensatory allele (a or x) into a population initially fixed for the mutant y and wild-type compensatory allele (A or X); and (*iii*) invasion of the compensatory allele (a or x) into a population with an arbitrary initial frequency of the mutant y allele.

(i) Invasion of the mutant y allele

For the mutant y chromosome to spread in a population initially fixed for the wild-type alleles (i.e., with initial equilibrium frequencies $\hat{q}_y = 0$ and $\hat{q}_a = 0$ or $\hat{q}_x = 0$), thereby initiating a coevolutionary cycle, the eigenvalue of the Jacobian associated with an increase in q_y must be the leading eigenvlaue (largest in magnitude) and greater than one ($\lambda_L > 1$). For these initial equilibria, both the Autosomal and X-linked models yield the same expression,

$$\lambda_L = \frac{1 + L(E + p)}{1 + LE}, \tag{C1}$$

which is greater than one when $E \geq 0$ and $0 < L, p \leq 1$ are all satisfied, and increases with both L and p, but decreases with E. Hence, two additional ingredients are required for the mutant y chromosome causing male coercive behaviour (*i.e.*, when $p > 0$) to be beneficial: some sperm displacement by the second male to mate with a given female ($L > 0$), and increased female fecundity during the second mating ($E > 0$). After invasion, the beneficial mutant y chromosome is expected to spread to fixation.

The other candidate leading eigenvalue associated with invasion of a compensatory mutation is equal to 1, and is therefore indeterminate for this initial equilibrium. As expected, in the absence of the mutant y chromosome, there is no selection favouring invasion of compensatory mutations.

(ii) Invasion of compensatory mutations when y is fixed

Invasion of a mutant compensatory mutation into a population initially fixed for the mutant y corresponds to initial equilibrium frequencies of $\hat{q}_y = 1$ and $\hat{q}_a = 0$ or $\hat{q}_x = 0$. Here differences between the Autosomal and X-linked models begin to appear. For the Autosomal model, the candiate leading eigenvalue associated with invasion of the mutant a allele is

$$\lambda_L = \frac{2 + LE\big(2 - p(1 + h_p)\big)}{2 + 2LE(1 - p)}. \tag{C2}$$

The candiate leading eigenvalue associated with invasion of the mutant x allele for the X-linked model is quadratic of the form

$$\lambda_L = \frac{-b - \sqrt{b^2 - 4ac}}{2a}, \tag{C3}$$

where $a = 2(LE(1 - h_p p) - 1)$, $b = c = 1 + LE(1 - h_p p)$. The candidate leading eigenvalues for both model are both greater than one when $E \geq 0$ and $0 < L, p, h_p \leq 1$ are all satisfied. That is, the conditions favouring invasion of the compensatory allele when the mutant y chromosome is initially fixed in the population are the same for both the Autosomal and X-linked model.

The Autosomal and X-linked models differ, however, in the strength of selection favouring the compensatory mutations. The selection coefficient for the invading autosomal or X-linked compensatory mutation (s_a and s_x respectively) can be roughly approximated as $\lambda_L - 1$ (6). From Eq(C2) and Eq(C3), selection favouring an X-linked compensatory mutation is always stronger than that for an autosomal one.

(iii) Invasion of compensatory mutations when q_y is arbitrary.

Invasion of a rare mutant compensatory mutation when the frequency of the mutant y chromosome is arbitrary corresponds to initial equilibrium frequencies of $0 \leq \hat{q}_y \leq 1$ and $\hat{q}_a = 0$ or $\hat{q}_x = 0$. For the Autosomal model, the candidate leading eigenvalue associated with invasion of the mutant a allele is

$$\lambda_L = \frac{2 + LE(2 - (1 + h_p)p\hat{q}_y)}{2 + 2LE(1 - p\hat{q}_y)}, \tag{C4}$$

which is greater than one when $E \geq 0$, $0 < L, h_p, p \leq 1$, and $0 < \hat{q}_y \leq 1$ are all satisfied. The corresponding candidate leading eigenvalue for invasion of the mutant x allele in the X-linked model is an unwieldy expression (see supplementary Mathematica notebook), but if the phenotypic effect of the compensatory mutation is additive ($h_p = 1/2$), we find that a rare X-linked compensatory mutation can invade when $0 \leq p \leq 0$ and either $E = 0$, $0 < \hat{q}_y < 0$, $0 < L \leq 1$, and $\underline{OR}$ $E > 0$ and $0 \leq 0 < \hat{q}_y, L \leq 1$. To summarize, an X-linked compensatory mutation can invade whenever an autosomal one can, but also when there is no increased female fecundity during $M2$, provided that the frequency of the mutant y chromosome is intermediate between 0 and 1. As in the previous scenario, the relative magnitude of selection favouring an X-linked compensatory mutation is always greater than that favouring an Autosomal one.

Implications

Three general implications emerge from our analytic results for the model of antagonistic coevolution over mating opportunities:

First, the models predict that sexual conflict over mating opportunities (scenario *c*) can cause coevolutionary cycles to occur between a male-beneficial mutation at a Y-linked locus (*e.g.*, governing some coercive trait/behaviour) and a compensatory mutation at another locus located elsewhere in the genome. Hence, antagonistic coevolution can potentially occur for a much broader suite of traits/conflicts than we found in our empirical study of *D. melanogaster*, and that we model in the main text.

Second, our analytic results suggest that in scenarios where the mutant y chromosome has already invaded (*i.e.*, scenarios *ii* and *iii* above), selection favouring the compensatory mutation will always be stronger if it is located on the X chromosome than on an autosome. This result holds whether we model

conflict over offspring fitness (scenario *a* in the main text), or mating opportunities (scenario *c* modeled here), and strongly suggests that the fixation probability of new compensatory mutations will be higher when they are X-linked than Autosomal. That is, the models of antagonistic coevolution over mating opportunities are expected to give similar results to those presented in Fig. 4B for the model of conflict over offspring survival).

Third, our X-linked and Autosomal models of sexual conflict over mating opportunities are not expected to result in different behaviour when compensatory evolution is fast vs. slow (*i.e.*, is limited by mutational variation or not). An important feature of conflict over mating opportunities is that there is positive selection for compensatory mutations as soon as the mutant y chromosome invades (*i.e.*, whenever $q_y > 0$), which is different from the models of conflict over offspring survival, where the mutant y chromosome had to reach a threshold frequency, $\tilde{q}_y$ before compensatory mutations were favoured. Ultimately, this difference between the models arises from the 'cost of compensation' that was included in the main models. When females carrying compensatory mutations pay some fitness cost when mating with wild-type males, it prevents the compensatory mutation from being immediately favoured once the mutant y arises in the population. Hence, without a cost of compensation, coevolutionary cycles expected to behave similarly whether or not the male-beneficial and compensatory mutations are likely to co-segregate in the population.

It is perhaps worth noting, however, that if a cost of comensation is incorporated in the models of conflict over mating opportunities, the phenomenon of a threshold frequency, $\tilde{q}_y$ re-emerges. Overall, our theoretical models suggest that under most circumstances, our predictions regarding the behavior of antagonistic coevolutionary cycles between sex-chromosomes are robust to the cause of conflict (offspring survival vs. restricting mating opportunities). The only major difference between the scenarios depends on whether or not there is a cost of compensation for females.

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Sexually antagonistic coevolution between the sex chromosomes of *Drosophila melanogaster:* *Theoretical models of sexual conflict over offspring viability*

Katrine Lund – Hansen[1,2,*,+], Colin Olito[1,+], Edward *H*. Morrow[2], and Jessica *K*. Abbott[1]

[1]Department of Biology, Section for Evolutionary Ecology, Lund University, Lund 223 62, Sweden.

[2]School of Life Sciences, University of Sussex, Brighton BN1 9QG, United Kingdom.

[*]Corresponding author e-mail: Katrine.Lund-Hansen@biol.lu.se.

[+]Equal contributions.

In this notebook we provide the deterministic recursions and key analytic results for the population genetic models described in the main article. Additional R code for Wright-Fisher simulations are available online at https://github.com/colin-olito/sexChromCoAdapt. Please report any problems or bugs using the GitHub issue tracker available at this website, or send an email to colin.olito@gmail.-com.

Y-linked locus influencing male fertilization success, Autosomal compensatory locus

In[•]:=
```
ClearAll["Global`*"]
```

- Deterministic recursions

Y-linked locus influencing male fertilization success

- *Genotypic frequencies among adults (before mating)*

In[•]:=
```
(* FY, Fy *)
```

- Genotypic frequencies among male gametes

In[•]:=
```
wgbar[Fy_][wgY_, wgy_] := ((1 - Fy) * wgY) + (Fy * wgy)
FgY[Fy_][wgY_, wgy_] := ((1 - Fy) * wgY) / wgbar[Fy][wgY, wgy];
Fgy[Fy_][wgY_, wgy_] := (Fy * wgy) / wgbar[Fy][wgY, wgy];
```

- Genotypic frequencies among offspring after fertilization and selection

```
In[•]:= wobar[Fy_][wgY_, wgy_][FAA_, FAa_, Faa_][woAAY_, woAaY_, woaaY_][woAAy_, woAay_,
          woaay_] := (FgY[Fy][wgY, wgy] * ((FAA * woAAY) + (FAa * woAaY) + (Faa * woaaY))) +
          (Fgy[Fy][wgY, wgy] * ((FAA * woAAy) + (FAa * woAay) + (Faa * woaay)))

        FoY[Fy_][wgY_, wgy_][FAA_, FAa_, Faa_][woAAY_, woAaY_, woaaY_][
          woAAy_, woAay_, woaay_] :=
                FgY[Fy][wgY, wgy] * ((FAA * woAAY) + (FAa * woAaY) + (Faa * woaaY))
        ------------------------------------------------------------------------;
        wobar[Fy][wgY, wgy][FAA, FAa, Faa][woAAY, woAaY, woaaY][woAAy, woAay, woaay]

        Foy[Fy_][wgY_, wgy_][FAA_, FAa_, Faa_][woAAY_, woAaY_, woaaY_][
          woAAy_, woAay_, woaay_] :=
                Fgy[Fy][wgY, wgy] * ((FAA * woAAy) + (FAa * woAay) + (Faa * woaay))
        ------------------------------------------------------------------------;
        wobar[Fy][wgY, wgy][FAA, FAa, Faa][woAAY, woAaY, woaaY][woAAy, woAay, woaay]
```

- Overall recursion for frequency of *y* allele in next generation

```
In[•]:= qyPr[Fy_][wgY_, wgy_][FAA_, FAa_, Faa_][woAAY_, woAaY_, woaaY_][
          woAAy_, woAay_, woaay_] :=
        Foy[Fy][wgY, wgy][FAA, FAa, Faa][woAAY, woAaY, woaaY][woAAy, woAay, woaay];
      Δqy[Fy_][wgY_, wgy_][FAA_, FAa_, Faa_][woAAY_, woAaY_, woaaY_][woAAy_, woAay_, woaay_] :=
        qyPr[Fy][wgY, wgy][FAA, FAa, Faa][woAAY, woAaY, woaaY][woAAy, woAay, woaay] - qy;
```

Autosomal compensatory locus influencing offspring survival

- Genotypic frequencies among adult females (before mating)

```
(*FAA,FAa,Faa*)
```

■ Genotypic frequencies among daughters after fertilization and selection

```
In[•]:= woAutobar[Fy_][wgY_, wgy_][FAA_, FAa_, Faa_][woAAY_, woAaY_, woaaY_][
          woAAy_, woAay_, woaay_] :=
        (FgY[Fy][wgY, wgy] * (((FAA * woAAY) + (FAa * woAaY)/2) * (FAA + FAa/2)) +
           Fgy[Fy][wgY, wgy] * (((FAA * woAAy) + (FAa * woAay)/2) * (FAA + FAa/2))) +
         (((FgY[Fy][wgY, wgy] * ((((FAA * woAAY) + (FAa * woAaY)/2) * (Faa + FAa/2)) +
                 (((Faa * woaaY) + (FAa * woAaY)/2) * (FAA + FAa/2)))) +
            Fgy[Fy][wgY, wgy] * ((((FAA * woAAy) + (FAa * woAay)/2) * (Faa + FAa/2)) +
                 (((Faa * woaay) + (FAa * woAay)/2) * (FAA + FAa/2))))) +
          (FgY[Fy][wgY, wgy] * (((Faa * woaaY) + (FAa * woAaY)/2) * (Faa + FAa/2)) +
             Fgy[Fy][wgY, wgy] * (((Faa * woaay) + (FAa * woAay)/2) * (Faa + FAa/2))));

      FoAA[Fy_][wgY_, wgy_][FAA_, FAa_, Faa_][woAAY_, woAaY_, woaaY_][
          woAAy_, woAay_, woaay_] :=
        (FgY[Fy][wgY, wgy] * (((FAA * woAAY) + (FAa * woAaY)/2) * (FAA + FAa/2)) +
           Fgy[Fy][wgY, wgy] * (((FAA * woAAy) + (FAa * woAay)/2) * (FAA + FAa/2)))/
        woAutobar[Fy][wgY, wgy][FAA, FAa, Faa][woAAY, woAaY, woaaY][woAAy, woAay, woaay];

      FoAa[Fy_][wgY_, wgy_][FAA_, FAa_, Faa_][woAAY_, woAaY_, woaaY_][
          woAAy_, woAay_, woaay_] :=
        (FgY[Fy][wgY, wgy] * ((((FAA * woAAY) + (FAa * woAaY)/2) * (Faa + FAa/2)) +
             (((Faa * woaaY) + (FAa * woAaY)/2) * (FAA + FAa/2))) +
           Fgy[Fy][wgY, wgy] * ((((FAA * woAAy) + (FAa * woAay)/2) * (Faa + FAa/2)) +
             (((Faa * woaay) + (FAa * woAay)/2) * (FAA + FAa/2))))/
        woAutobar[Fy][wgY, wgy][FAA, FAa, Faa][woAAY, woAaY, woaaY][woAAy, woAay, woaay];

      Foaa[Fy_][wgY_, wgy_][FAA_, FAa_, Faa_][woAAY_, woAaY_, woaaY_][
          woAAy_, woAay_, woaay_] :=
        (FgY[Fy][wgY, wgy] * (((Faa * woaaY) + (FAa * woAaY)/2) * (Faa + FAa/2)) +
           Fgy[Fy][wgY, wgy] * (((Faa * woaay) + (FAa * woAay)/2) * (Faa + FAa/2)))/
        woAutobar[Fy][wgY, wgy][FAA, FAa, Faa][woAAY, woAaY, woaaY][woAAy, woAay, woaay];
```

■ Analytic Results

```
In[•]:= Clear[JacobianMat]
```

- Define the Jacobian

```
In[•]:= JacobianMat[Fy_: Fy][wgY_: wgY, wgy_: wgy][FAA_: FAA, FAa_: FAa, Faa_: Faa][woAAY_: woAAY,
          woAaY_: WoAaY, woaaY_: woaaY][woAAy_: woAAy, woAay_: woAay, woaay_: woaay] = Outer[D,
        {qyPr[Fy][wgY, wgy][FAA, FAa, Faa][woAAY, woAaY, woaaY][woAAy, woAay, woaay],
         FoAA[Fy][wgY, wgy][FAA, FAa, Faa][woAAY, woAaY, woaaY][woAAy, woAay, woaay],
         FoAa[Fy][wgY, wgy][FAA, FAa, Faa][woAAY, woAaY, woaaY][woAAy, woAay, woaay],
         Foaa[Fy][wgY, wgy][FAA, FAa, Faa][woAAY, woAaY, woaaY][woAAy, woAay, woaay]},
        {Fy, FAA, FAa, Faa}];
```

- Invasion of mutant y chromosome: initial frequencies of *y* and *a* alleles are 0

```
In[•]:= Clear[J]
        J = JacobianMat[0][1, 1 + sm][1, 0, 0][1, 1 - hc sc, 1 - sc][1 - so, 1 - ho so, 1] // Simplify
        Dimensions[J]
```

$$\text{Out[•]= } \left\{\{-(1+\text{sm})\ (-1+\text{so}),\ 0,\ 0,\ 0\},\ \left\{0,\ 0,\ -1+\frac{\text{hc sc}}{2},\ -2+\text{sc}\right\},\ \left\{0,\ 0,\ 1-\frac{\text{hc sc}}{2},\ 2-\text{sc}\right\},\ \{0,\ 0,\ 0,\ 0\}\right\}$$

$$\text{Out[•]= } \{4,\ 4\}$$

Have a look at Eigenvalues

```
In[•]:= Clear[λ]
        λ = Eigenvalues[J] // FullSimplify
```

$$\text{Out[•]= } \left\{0,\ 0,\ 1-\frac{\text{hc sc}}{2},\ -(1+\text{sm})\ (-1+\text{so})\right\}$$

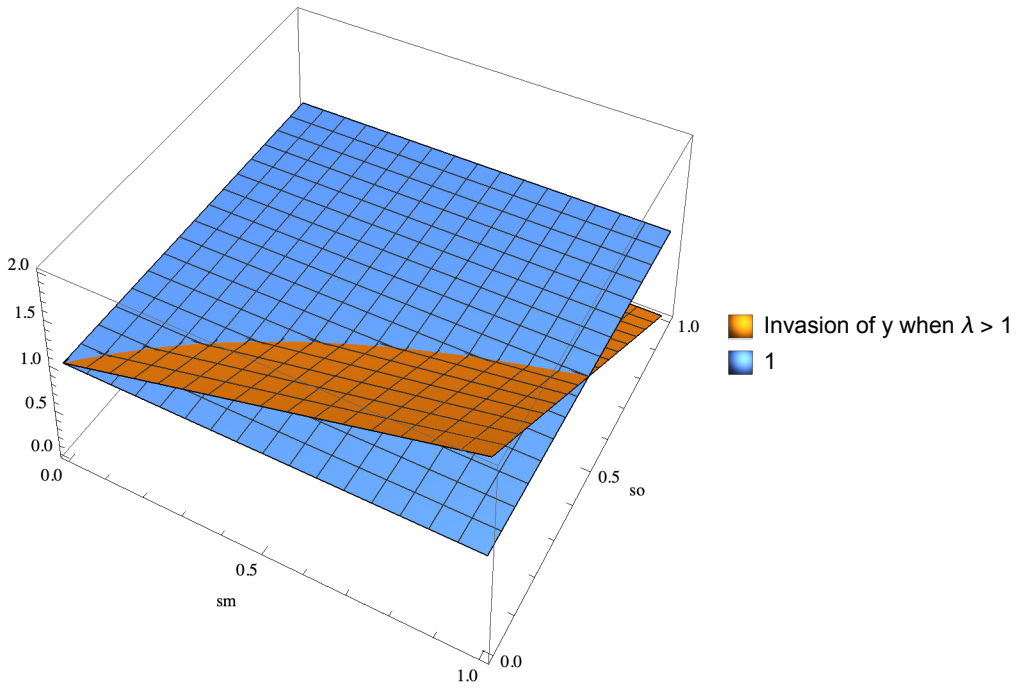
```
In[•]:= λ[[3]] // Expand
        λ[[4]] /. qy → 0 // Expand
```

$$\text{Out[•]= } 1-\frac{\text{hc sc}}{2}$$

$$\text{Out[•]= } 1+\text{sm}-\text{so}-\text{sm so}$$

```
In[•]:= Plot3D[{1 + sm - so - sm so, 1}, {sm, 0, 1}, {so, 0, 1}, AxesLabel → Automatic,
         AspectRatio → 1, PlotLegends → {"Invasion of y when λ > 1", "1"}]
```

Out[•]=



Defining $\delta = s_m - s_o$, to $O(s_m s_o)$, we have $\lambda_L = 1 + \delta$, and the mutant y chromosome can invade when $\delta > 0$.

- Invasion of mutant y chromosome: initial frequency of y is arbitrary, initial frequency of a is 0

```
In[•]:= Clear[J]
        J = JacobianMat[qy][1, 1 + sm][1, 0, 0][1, 1 - hc sc, 1 - sc][
            1 - (sm - δ), 1 - ho (sm - δ), 1] // Simplify
        Clear[λ]
        λ = Eigenvalues[J] // Simplify
```

$$\text{Out[•]= } \left\{\left\{\frac{1 - sm^2 + \delta + sm\,\delta}{\left(-1 + qy\left(sm^2 - \delta - sm\,\delta\right)\right)^2}, 0, \frac{(-1 + qy)\,qy\,(1 + sm)\left(hc\,sc\,(-1 + sm - \delta) + \left(-1 + ho\right)(sm - \delta)\right)}{\left(-1 + qy\left(sm^2 - \delta - sm\,\delta\right)\right)^2}, \frac{(-1 + qy)\,qy\,(1 + sm)\,(-sm + sc\,(-1 + sm - \delta) + \delta)}{\left(-1 + qy\left(sm^2 - \delta - sm\,\delta\right)\right)^2}\right\},\right.$$
$$\left\{0, 0, \frac{2 + hc\,(-1 + qy)\,sc + qy\left(-\left(1 + ho\right)sm^2 + \left(1 + ho\right)\delta + sm\left(1 + ho\,(-1 + \delta) + \delta\right)\right)}{-2 + 2\,qy\left(sm^2 - \delta - sm\,\delta\right)}, \frac{2 + (-1 + qy)\,sc + qy\left(sm - sm^2 + \delta + sm\,\delta\right)}{-1 + qy\left(sm^2 - \delta - sm\,\delta\right)}\right\},$$
$$\left\{0, 0, \frac{-2 + hc\,(sc - qy\,sc) + qy\left(\left(1 + ho\right)sm^2 - \left(1 + ho\right)\delta - sm\left(1 + ho\,(-1 + \delta) + \delta\right)\right)}{-2 + 2\,qy\left(sm^2 - \delta - sm\,\delta\right)}, \frac{2 + (-1 + qy)\,sc + qy\left(sm - sm^2 + \delta + sm\,\delta\right)}{1 + qy\left(-sm^2 + \delta + sm\,\delta\right)}\right\}, \{0, 0, 0, 0\}\Big\}$$

$$\text{Out[•]= } \left\{0, 0, \frac{1 - sm^2 + \delta + sm\,\delta}{\left(-1 + qy\left(sm^2 - \delta - sm\,\delta\right)\right)^2}, \frac{-2 + hc\,(sc - qy\,sc) + qy\left(\left(1 + ho\right)sm^2 - \left(1 + ho\right)\delta - sm\left(1 + ho\,(-1 + \delta) + \delta\right)\right)}{-2 + 2\,qy\left(sm^2 - \delta - sm\,\delta\right)}\right\}$$

```
In[•]:= Series[1 - sm^2 + δ + sm δ / (-1 + qy (sm^2 - δ - sm δ))^2, {δ, 0, 1}] // FullSimplify
        Series[1 - sm^2 / (-1 + qy sm^2)^2 - (1 + sm) (1 + qy (-2 + sm^2)) δ / (-1 + qy sm^2)^3, {sm, 0, 1}] // FullSimplify
        (1 + δ - 2 qy δ) + (δ - 2 qy δ) sm // Factor
```

$$\text{Out[•]= } \frac{1 - sm^2}{\left(-1 + qy\,sm^2\right)^2} - \frac{(1 + sm)\left(1 + qy\left(-2 + sm^2\right)\right)\delta}{\left(-1 + qy\,sm^2\right)^3} + O[\delta]^2$$

$$\text{Out[•]= } (1 + \delta - 2\,qy\,\delta) + (\delta - 2\,qy\,\delta)\,sm + O[sm]^2$$

$$\text{Out[•]= } 1 + \delta - 2\,qy\,\delta + sm\,\delta - 2\,qy\,sm\,\delta$$

```
In[•]:= 1 + δ (1 - 2 qy)
```

$$\text{Out[•]= } 1 + (1 - 2\,qy)\,\delta$$

```
In[•]:= (-2 + hc (sc - qy sc) + qy ((1 + ho) sm^2 - (1 + ho) δ - sm (1 + ho (-1 + δ) + δ))) / (-2 + 2 qy (sm^2 - δ - sm δ))
```

$$\text{Out[•]= } \frac{-2 + hc\,(sc - qy\,sc) + qy\left(\left(1 + ho\right)sm^2 - \left(1 + ho\right)\delta - sm\left(1 + ho\,(-1 + \delta) + \delta\right)\right)}{-2 + 2\,qy\left(sm^2 - \delta - sm\,\delta\right)}$$

```
In[•]:= Reduce[(-2 + hc (sc - qy sc) + qy ((1 + ho) sm^2 - (1 + ho) δ - sm (1 + ho (-1 + δ) + δ)))/(-2 + 2 qy (sm^2 - δ - sm δ)) == 1, qy]

Out[•]= (sc == 0 && ho == 1 && - sm - sm^2 + qy sm^3 + qy sm^4 + δ +
      sm δ - qy sm δ - 3 qy sm^2 δ - 2 qy sm^3 δ + qy δ^2 + 2 qy sm δ^2 + qy sm^2 δ^2 ≠ 0) ||
   ((sm == -1 || sm == δ) && sc == 0 && 1 + qy sm ≠ 0) ||
   (hc sc + sm - ho sm + sm^2 - ho sm^2 - δ + ho δ - sm δ + ho sm δ ≠ 0 &&
     qy == hc sc / (hc sc + sm - ho sm + sm^2 - ho sm^2 - δ + ho δ - sm δ + ho sm δ) &&
     - hc sc - sm + ho sm - sm^2 + ho sm^2 + hc sc sm^2 + δ - ho δ - hc sc δ + sm δ - ho sm δ - hc sc sm δ ≠ 0) ||
   ((sm == -1 || sm == δ) && sc ≠ 0 && hc == 0 && 1 + qy sm ≠ 0) ||
   ((1 + sm) (sm - δ) ≠ 0 && ho == 1 && sc ≠ 0 && hc == 0 && -1 + qy sm^2 - qy δ - qy sm δ ≠ 0)
```

For $0 \leq s_m, s_c, h_o, h_c \leq 1$ the relevant solution presented as $\tilde{q}_y^A$ in the text is:

```
In[•]:= qyTildeA = hc sc / (hc sc + sm - ho sm + sm^2 - ho sm^2 - δ + ho δ - sm δ + ho sm δ);
```

Under additive fitness effects $h_o = h_c = h = \frac{1}{2}$, $\tilde{q}_y^A$ simplifies the the expression used to produce Fig. 4A:

```
In[•]:= qyTildeA /. hc → 1/2 /. ho → 1/2 // FullSimplify

Out[•]= sc / (sc + (1 + sm) (sm - δ))
```

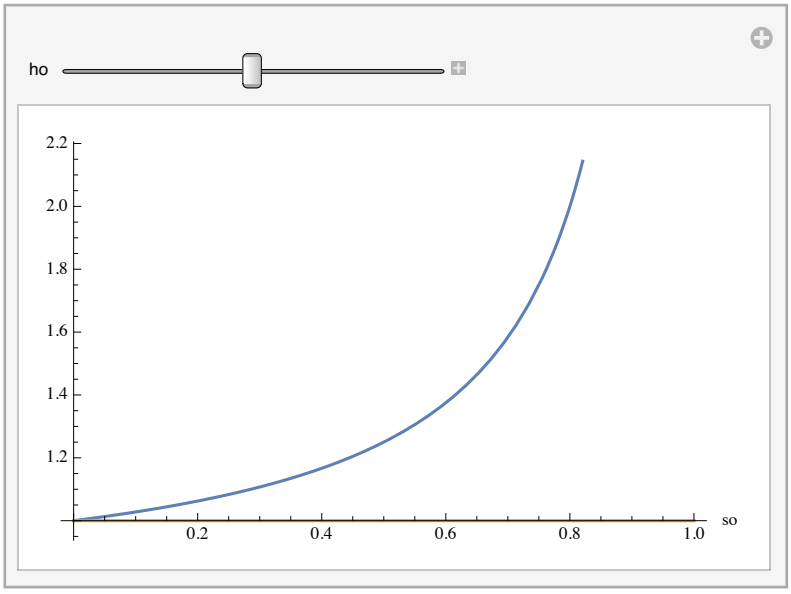
- Invasion of compensatory mutation: population initially fixed for mutant y, and frequency of $a = 0$

```
In[•]:= Clear[J]
        J = JacobianMat[1][1, 1 + sm][1, 0, 0][1, 1 - hc sc, 1 - sc][1 - so, 1 - ho so, 1] // Simplify;
        Clear[λ]
        λ = Eigenvalues[J] // Simplify

Out[•]= {1 / (1 + sm - so - sm so), 0, 0, (-2 + so + ho so) / (2 (-1 + so))}

In[•]:= FullSimplify[(-2 + so + ho so) / (2 (-1 + so))] == FullSimplify[(2 - so - ho so) / (2 (1 - so))]

Out[•]= True

In[•]:= (2 - (1 + ho) sm + δ + ho δ) / (2 - 2 sm + 2 δ) /. ho -> 1/2 // FullSimplify

Out[•]= 1/4 (3 + 1 / (1 - sm + δ))

In[•]:= Reduce[(-2 + so + ho so) / (2 (-1 + so)) > 1]

Out[•]= (so < 0 && ho > 1) || (0 < so < 1 && ho < 1) || (so > 1 && ho > 1)
```

As expected, when the mutant y chromosome is initially fixed in the population, the compensatory allele is always able to invade for relevant parameter values ($0 \leq h_o \leq 1$).

In[•]:=
```
Manipulate[Plot[{(-2 + so + ho so)/(2 (-1 + so)), 1}, {so, 0, 1}, AxesLabel → Automatic], {{ho, 1/2}, 0, 1}]
```

Out[•]=



Y-linked locus influencing male fertilization success, X-linked compensatory locus

In[•]:=
```
ClearAll["Global`*"]
```

- Deterministic recursions

Y-linked locus influencing male fertilization success

- Genotypic frequencies among male gametes

In[•]:=
```
wgbar[FXY_, FXy_, FxY_, Fxy_][wgY_, wgy_] := (FXY + FxY) * wgY + (FXy + Fxy) * wgy

FgY[FXY_, FXy_, FxY_, Fxy_][wgY_, wgy_] := (FXY + FxY) * wgY / wgbar[FXY, FXy, FxY, Fxy][wgY, wgy];
Fgy[FXY_, FXy_, FxY_, Fxy_][wgY_, wgy_] := (FXy + Fxy) * wgy / wgbar[FXY, FXy, FxY, Fxy][wgY, wgy];

FgXY[FXY_, FXy_, FxY_, Fxy_][wgY_, wgy_] := FXY * wgY / wgbar[FXY, FXy, FxY, Fxy][wgY, wgy];
FgXy[FXY_, FXy_, FxY_, Fxy_][wgY_, wgy_] := FXy * wgy / wgbar[FXY, FXy, FxY, Fxy][wgY, wgy];
FgxY[FXY_, FXy_, FxY_, Fxy_][wgY_, wgy_] := FxY * wgY / wgbar[FXY, FXy, FxY, Fxy][wgY, wgy];
Fgxy[FXY_, FXy_, FxY_, Fxy_][wgY_, wgy_] := Fxy * wgy / wgbar[FXY, FXy, FxY, Fxy][wgY, wgy];
```

X-linked compensatory locus

- Genotypic frequencies among offspring after fertilization and selection

Males

```
In[•]:= wmbar[FXY_, FXy_, FxY_, Fxy_][wgY_, wgy_][FXX_, FXx_, Fxx_][woXXY_, woXxY_, woxxY_][
          woXXy_, woXxy_, woxxy_] :=
        (FgY[FXY, FXy, FxY, Fxy][wgY, wgy] * (FXX * woXXY + (FXx * woXxY)/2)) +
         (Fgy[FXY, FXy, FxY, Fxy][wgY, wgy] * (FXX * woXXy + (FXx * woXxy)/2)) +
         (FgY[FXY, FXy, FxY, Fxy][wgY, wgy] * (Fxx * woxxY + (FXx * woXxY)/2)) +
         (Fgy[FXY, FXy, FxY, Fxy][wgY, wgy] * (Fxx * woxxy + (FXx * woXxy)/2))

      FXYPr[FXY_, FXy_, FxY_, Fxy_][wgY_, wgy_][FXX_, FXx_, Fxx_][woXXY_, woXxY_, woxxY_][
          woXXy_, woXxy_, woxxy_] :=
        (FgY[FXY, FXy, FxY, Fxy][wgY, wgy] * (FXX * woXXY + (FXx * woXxY)/2))/
         wmbar[FXY, FXy, FxY, Fxy][wgY, wgy][FXX, FXx, Fxx][woXXY, woXxY, woxxY][
          woXXy, woXxy, woxxy];

      FXyPr[FXY_, FXy_, FxY_, Fxy_][wgY_, wgy_][FXX_, FXx_, Fxx_][woXXY_, woXxY_, woxxY_][
          woXXy_, woXxy_, woxxy_] :=
        (Fgy[FXY, FXy, FxY, Fxy][wgY, wgy] * (FXX * woXXy + (FXx * woXxy)/2))/
         wmbar[FXY, FXy, FxY, Fxy][wgY, wgy][FXX, FXx, Fxx][woXXY, woXxY, woxxY][
          woXXy, woXxy, woxxy];

      FxYPr[FXY_, FXy_, FxY_, Fxy_][wgY_, wgy_][FXX_, FXx_, Fxx_][woXXY_, woXxY_, woxxY_][
          woXXy_, woXxy_, woxxy_] :=
        (FgY[FXY, FXy, FxY, Fxy][wgY, wgy] * (Fxx * woxxY + (FXx * woXxY)/2))/
         wmbar[FXY, FXy, FxY, Fxy][wgY, wgy][FXX, FXx, Fxx][woXXY, woXxY, woxxY][
          woXXy, woXxy, woxxy];

      FxyPr[FXY_, FXy_, FxY_, Fxy_][wgY_, wgy_][FXX_, FXx_, Fxx_][woXXY_, woXxY_, woxxY_][
          woXXy_, woXxy_, woxxy_] :=
        (Fgy[FXY, FXy, FxY, Fxy][wgY, wgy] * (Fxx * woxxy + (FXx * woXxy)/2))/
         wmbar[FXY, FXy, FxY, Fxy][wgY, wgy][FXX, FXx, Fxx][woXXY, woXxY, woxxY][
          woXXy, woXxy, woxxy];
```

Females

```
In[•]:= wfbar[FXY_, FXy_, FxY_, Fxy_][wgY_, wgy_][FXX_, FXx_, Fxx_][woXXY_, woXxY_, woxxY_][
    woXXy_, woXxy_, woxxy_] :=
   (FgXY[FXY, FXy, FxY, Fxy][wgY, wgy] * (FXX * woXXY + (FXx * woXxY)/2) +
     FgXy[FXY, FXy, FxY, Fxy][wgY, wgy] * (FXX * woXXy + (FXx * woXxy)/2)) +
   (((FgXY[FXY, FXy, FxY, Fxy][wgY, wgy] * (Fxx * woxxY + (FXx * woXxY)/2) +
       FgxY[FXY, FXy, FxY, Fxy][wgY, wgy] * (FXX * woXXY + (FXx * woXxY)/2)) +
     (FgXy[FXY, FXy, FxY, Fxy][wgY, wgy] * (Fxx * woxxy + (FXx * woXxy)/2) +
       Fgxy[FXY, FXy, FxY, Fxy][wgY, wgy] * (FXX * woXXy + (FXx * woXxy)/2))) +
   (FgxY[FXY, FXy, FxY, Fxy][wgY, wgy] * (Fxx * woxxY + (FXx * woXxY)/2) +
     Fgxy[FXY, FXy, FxY, Fxy][wgY, wgy] * (Fxx * woxxy + (FXx * woXxy)/2));

FXXPr[FXY_, FXy_, FxY_, Fxy_][wgY_, wgy_][FXX_, FXx_, Fxx_][woXXY_, woXxY_, woxxY_][
    woXXy_, woXxy_, woxxy_] :=
   (FgXY[FXY, FXy, FxY, Fxy][wgY, wgy] * (FXX * woXXY + (FXx * woXxY)/2) +
     FgXy[FXY, FXy, FxY, Fxy][wgY, wgy] * (FXX * woXXy + (FXx * woXxy)/2)) /
   wfbar[FXY, FXy, FxY, Fxy][wgY, wgy][FXX, FXx, Fxx][woXXY, woXxY, woxxY][
    woXXy, woXxy, woxxy];

FXxPr[FXY_, FXy_, FxY_, Fxy_][wgY_, wgy_][FXX_, FXx_, Fxx_][woXXY_, woXxY_, woxxY_][
    woXXy_, woXxy_, woxxy_] :=
   (1 / wfbar[FXY, FXy, FxY, Fxy][wgY, wgy][FXX, FXx, Fxx][woXXY, woXxY, woxxY][woXXy,
       woXxy, woxxy]) ((FgXY[FXY, FXy, FxY, Fxy][wgY, wgy] * (Fxx * woxxY + (FXx * woXxY)/2) +
       FgxY[FXY, FXy, FxY, Fxy][wgY, wgy] * (FXX * woXXY + (FXx * woXxY)/2)) +
     (FgXy[FXY, FXy, FxY, Fxy][wgY, wgy] * (Fxx * woxxy + (FXx * woXxy)/2) +
       Fgxy[FXY, FXy, FxY, Fxy][wgY, wgy] * (FXX * woXXy + (FXx * woXxy)/2)));

FxxPr[FXY_, FXy_, FxY_, Fxy_][wgY_, wgy_][FXX_, FXx_, Fxx_][woXXY_, woXxY_, woxxY_][
    woXXy_, woXxy_, woxxy_] :=
   (FgxY[FXY, FXy, FxY, Fxy][wgY, wgy] * (Fxx * woxxY + (FXx * woXxY)/2) +
     Fgxy[FXY, FXy, FxY, Fxy][wgY, wgy] * (Fxx * woxxy + (FXx * woXxy)/2)) /
   wfbar[FXY, FXy, FxY, Fxy][wgY, wgy][FXX, FXx, Fxx][woXXY, woXxY, woxxY][
    woXXy, woXxy, woxxy];
```

- Analytic Results
- Define the Jacobian

```
In[•]:= Clear[JacobianMat]
JacobianMat[FXY_:FXY, FXy_:FXy, FxY_:FxY, Fxy_:Fxy][wgY_:wgY, wgy_:wgy][
      FXX_:FXX, FXx_:FXx, Fxx_:Fxx][woXXY_:WoXXY, woXxY_:woXxY, woxxY_:woxxY][
    woXXy_:woXXy, woXxy_:woXxy, woxxy_:woxxy] = Outer[D,
    {FXYPr[FXY, FXy, FxY, Fxy][wgY, wgy][FXX, FXx, Fxx][woXXY, woXxY, woxxY][
      woXXy, woXxy, woxxy],
     FXyPr[FXY, FXy, FxY, Fxy][wgY, wgy][FXX, FXx, Fxx][woXXY, woXxY, woxxY][
      woXXy, woXxy, woxxy],
     FxYPr[FXY, FXy, FxY, Fxy][wgY, wgy][FXX, FXx, Fxx][woXXY, woXxY, woxxY][
      woXXy, woXxy, woxxy],
     FxyPr[FXY, FXy, FxY, Fxy][wgY, wgy][FXX, FXx, Fxx][woXXY, woXxY, woxxY][
      woXXy, woXxy, woxxy],
     FXXPr[FXY, FXy, FxY, Fxy][wgY, wgy][FXX, FXx, Fxx][woXXY, woXxY, woxxY][
      woXXy, woXxy, woxxy],
     FXxPr[FXY, FXy, FxY, Fxy][wgY, wgy][FXX, FXx, Fxx][woXXY, woXxY, woxxY][
      woXXy, woXxy, woxxy],
     FxxPr[FXY, FXy, FxY, Fxy][wgY, wgy][FXX, FXx, Fxx][woXXY, woXxY, woxxY][
      woXXy, woXxy, woxxy]}, {FXY, FXy, FxY, Fxy, FXX, FXx, Fxx}];
```

- Invasion of mutant y chromosome: initial frequencies of y and x alleles are 0

```
In[•]:= Clear[J]
J = JacobianMat[1, 0, 0, 0][1, 1 + sm][1, 0, 0][1, 1, 1][
    1 - so, 1 - ho so, 1] // FullSimplify
Dimensions[
  J]
```

$$\text{Out[•]}= \left\{\left\{0,\ (1+\text{sm})\ (-1+\text{so}),\ 0,\ (1+\text{sm})\ (-1+\text{so}),\ 0,\ -\frac{1}{2},\ -1\right\},\right.$$
$$\{0,\ -(1+\text{sm})\ (-1+\text{so}),\ 0,\ -(1+\text{sm})\ (-1+\text{so}),\ 0,\ 0,\ 0\},$$
$$\left\{0,\ 0,\ 0,\ 0,\ 0,\ \frac{1}{2},\ 1\right\},\ \{0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0\},$$
$$\left\{0,\ 0,\ -1,\ (1+\text{sm})\ (-1+\text{so}),\ 0,\ -\frac{1}{2},\ -1\right\},$$
$$\left.\left\{0,\ 0,\ 1,\ -(1+\text{sm})\ (-1+\text{so}),\ 0,\ \frac{1}{2},\ 1\right\},\ \{0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0\}\right\}$$

```
Out[•]= {7, 7}
```

Have a look at Eigenvalues

```
In[•]:= Clear[λ]
λ = Eigenvalues[J] // FullSimplify
```

$$\text{Out[•]}= \left\{1,\ -\frac{1}{2},\ 0,\ 0,\ 0,\ 0,\ -(1+\text{sm})\ (-1+\text{so})\right\}$$

```
In[•]:= λ[[7]]
```

$$\text{Out[•]}= -(1+\text{sm})\ (-1+\text{so})$$

As expected, we get the same result for invasion of the mutant y chromosome as we did for the Autosomal model.

```
In[•]:= (1 + sm) (1 - so) // Expand
```

$$\text{Out[•]=}\quad 1 + sm - so - sm\,so$$

- Invasion of mutant y chromosome allele: initial frequency of y is arbitrary, initial frequency of x is 0.

```
In[•]:= Clear[J]
        J = JacobianMat[(1 - qy), qy, 0, 0][1, 1 + sm][1, 0, 0][1, 1 - hc sc, 1 - sc][
              1 - so, 1 - ho so, 1] /. hc → 1 / 2 /. ho → 1 / 2 // Simplify
        Clear[λ]
        λ = Eigenvalues[J] /. FXY → (1 - qy) /. FXy → qy // FullSimplify
```

$$\text{Out[•]=}\quad \left\{\left\{-\frac{qy\,(1+sm)\,(-1+so)}{(-1+qy\,(sm\,(-1+so)+so))^2},\ -\frac{(-1+qy)\,(1+sm)\,(-1+so)}{(-1+qy\,(sm\,(-1+so)+so))^2},\right.\right.$$
$$-\frac{qy\,(1+sm)\,(-1+so)}{(-1+qy\,(sm\,(-1+so)+so))^2},\ -\frac{(-1+qy)\,(1+sm)\,(-1+so)}{(-1+qy\,(sm\,(-1+so)+so))^2},$$
$$0,\ -\frac{(-1+qy)\,(sc-2\,(1+qy\,sm)+qy\,sc\,(-2+sm\,(-1+so)+so))}{4\,(-1+qy\,(sm\,(-1+so)+so))^2},$$
$$\left.\frac{(-1+qy)\,(1+(-1+qy)\,sc+qy\,sm)}{(-1+qy\,(sm\,(-1+so)+so))^2}\right\},\ \left\{\frac{qy\,(1+sm)\,(-1+so)}{(-1+qy\,(sm\,(-1+so)+so))^2},\right.$$
$$\frac{(-1+qy)\,(1+sm)\,(-1+so)}{(-1+qy\,(sm\,(-1+so)+so))^2},\ \frac{qy\,(1+sm)\,(-1+so)}{(-1+qy\,(sm\,(-1+so)+so))^2},\ \frac{(-1+qy)\,(1+sm)\,(-1+so)}{(-1+qy\,(sm\,(-1+so)+so))^2},$$
$$0,\ -\frac{qy\,(1+sm)\,\left(2-3\,so+2\,sc\,(-1+qy+so-qy\,so)+qy\,\left(so^2+sm\,\left(2-3\,so+so^2\right)\right)\right)}{4\,(-1+qy\,(sm\,(-1+so)+so))^2},$$
$$\left.\frac{qy\,(1+sm)\,(1+(-1+qy)\,sc+qy\,sm)\,(-1+so)}{(-1+qy\,(sm\,(-1+so)+so))^2}\right\},$$
$$\left\{0,0,0,0,0,-\frac{(-1+qy)\,(-2+sc)}{-4+4\,qy\,(sm\,(-1+so)+so)},\frac{-1+qy+sc-qy\,sc}{-1+qy\,(sm\,(-1+so)+so)}\right\},$$
$$\left\{0,0,0,0,0,\frac{qy\,(1+sm)\,(-2+so)}{-4+4\,qy\,(sm\,(-1+so)+so)},-\frac{qy\,(1+sm)}{-1+qy\,(sm\,(-1+so)+so)}\right\},$$
$$\left\{0,0,\frac{1}{-1+qy\,(sm\,(-1+so)+so)},-\frac{(1+sm)\,(-1+so)}{-1+qy\,(sm\,(-1+so)+so)},0,\right.$$
$$\left.\frac{2+(-1+qy)\,sc-qy\,(sm\,(-2+so)+so)}{-4+4\,qy\,(sm\,(-1+so)+so)},\frac{1+(-1+qy)\,sc+qy\,sm}{-1+qy\,(sm\,(-1+so)+so)}\right\},$$
$$\left\{0,0,\frac{1}{1-qy\,(sm\,(-1+so)+so)},\frac{(1+sm)\,(-1+so)}{-1+qy\,(sm\,(-1+so)+so)},0,\right.$$
$$\left.\left.\frac{-2+sc-qy\,sc+qy\,(sm\,(-2+so)+so)}{-4+4\,qy\,(sm\,(-1+so)+so)},\frac{-1+sc-qy\,sc-qy\,sm}{-1+qy\,(sm\,(-1+so)+so)}\right\},\{0,0,0,0,0,0,0\}\right\}$$

$$\text{Out[•]=}\quad \left\{0,0,0,0,-\frac{(1+sm)\,(-1+so)}{(-1+qy\,(sm\,(-1+so)+so))^2},\right.$$
$$\left(-2+sc-qy\,sc+qy\,(sm\,(-2+so)+so)-\sqrt{\left(36+(-1+qy)^2\,sc^2+4\,qy\,sm\,(18+(8+qy)\,sm)-\right.\right.\right.$$
$$\left.\left.\left.4\,qy\,(1+sm)\,(13+(12+qy)\,sm)\,so+qy\,(16+qy)\,(1+sm)^2\,so^2-\right.\right.\right.$$
$$\left.\left.\left.2\,(-1+qy)\,sc\,(-10+qy\,(sm\,(-2+so)+so))\right)\right)\Big/(-8+8\,qy\,(sm\,(-1+so)+so)),$$
$$\left(-2+sc-qy\,sc+qy\,(sm\,(-2+so)+so)+\sqrt{\left(36+(-1+qy)^2\,sc^2+4\,qy\,sm\,(18+(8+qy)\,sm)-\right.\right.\right.$$
$$\left.\left.\left.4\,qy\,(1+sm)\,(13+(12+qy)\,sm)\,so+qy\,(16+qy)\,(1+sm)^2\,so^2-\right.\right.\right.$$
$$\left.\left.\left.2\,(-1+qy)\,sc\,(-10+qy\,(sm\,(-2+so)+so))\right)\right)\Big/(-8+8\,qy\,(sm\,(-1+so)+so))\right\}$$

```
In[•]:= λ[[5]] // FullSimplify
        λ[[6]] // FullSimplify
        λ[[7]] // FullSimplify
```

$$\text{Out[•]=}\ -\frac{(1+sm)\,(-1+so)}{(-1+qy\,(sm\,(-1+so)+so))^2}$$

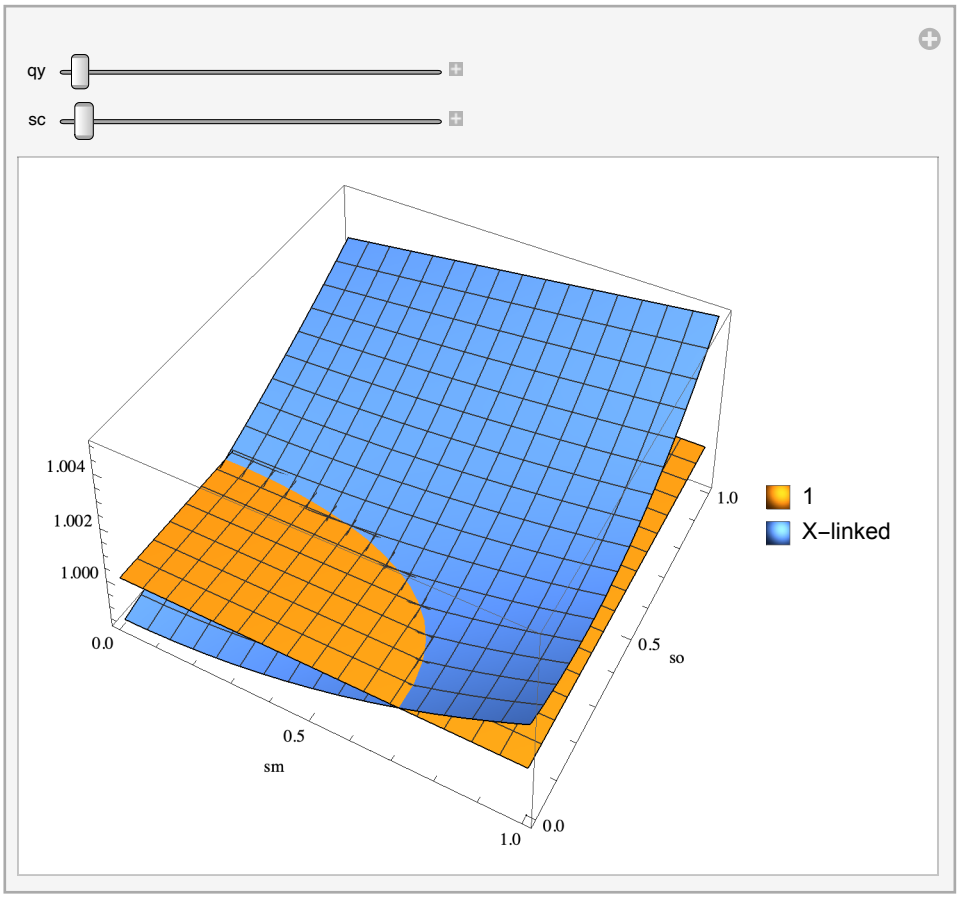
$$\text{Out[•]=}\ \left(-2+sc-qy\,sc+qy\,(sm\,(-2+so)+so)-\sqrt{\left(36+(-1+qy)^2\,sc^2+4\,qy\,sm\,(18+(8+qy)\,sm)-4\,qy\,(1+sm)\,(13+(12+qy)\,sm)\,so+qy\,(16+qy)\,(1+sm)^2\,so^2-2\,(-1+qy)\,sc\,(-10+qy\,(sm\,(-2+so)+so))\right)}\right)\Big/(-8+8\,qy\,(sm\,(-1+so)+so))$$

$$\text{Out[•]=}\ \left(-2+sc-qy\,sc+qy\,(sm\,(-2+so)+so)+\sqrt{\left(36+(-1+qy)^2\,sc^2+4\,qy\,sm\,(18+(8+qy)\,sm)-4\,qy\,(1+sm)\,(13+(12+qy)\,sm)\,so+qy\,(16+qy)\,(1+sm)^2\,so^2-2\,(-1+qy)\,sc\,(-10+qy\,(sm\,(-2+so)+so))\right)}\right)\Big/(-8+8\,qy\,(sm\,(-1+so)+so))$$

The eigenvalue associated withe invasion of the compensatory *x* allele is λ[[6]]:

```
In[•]:= Manipulate[Plot3D[{1, (-2 + sc - qy sc + qy (sm (-2 + so) + so) -
              √((36 + (-1 + qy)^2 sc^2 + 4 qy sm (18 + (8 + qy) sm) - 4 qy (1 + sm) (13 + (12 + qy) sm) so +
                 qy (16 + qy) (1 + sm)^2 so^2 - 2 (-1 + qy) sc (-10 + qy (sm (-2 + so) + so))))) /
           (-8 + 8 qy (sm (-1 + so) + so))}, {sm, 0, 1}, {so, 0, 1}, AxesLabel → Automatic,
         AspectRatio → 1, PlotLegends → {1, "X-linked"}], {{qy, 0.01},
         0, 1}, {{sc, 0.005}, 0, 0.25}]
```

Out[•]=




```
In[•]:=
```

```
In[•]:= Assuming[0 ≤ sc ≤ 1 && 0 ≤ so ≤ 1 && 0 ≤ qy ≤ 1 && sc ∈ ℝ && so ∈ ℝ && qy ∈ ℝ,
  Solve[(-2 + sc - qy sc + qy (sm (-2 + so) + so) -
      √(36 + (-1 + qy)^2 sc^2 + 4 qy sm (18 + (8 + qy) sm) - 4 qy (1 + sm) (13 + (12 + qy) sm) so +
        qy (16 + qy) (1 + sm)^2 so^2 - 2 (-1 + qy) sc (-10 + qy (sm (-2 + so) + so)))) /
    (-8 + 8 qy (sm (-1 + so) + so)) == 1, qy]] // FullSimplify
```

```
Out[•]= {{qy → (2 sc - sc sm + 2 sm^2 + (2 + sc - 3 sm) (1 + sm) so +
      (1 + sm)^2 so^2 - √(-8 sc (sm (-1 + so) + so) (sc - 2 sm + 3 (1 + sm) so) +
        (2 sc - sc sm + 2 sm^2 + (2 + sc - 3 sm) (1 + sm) so + (1 + sm)^2 so^2)^2)) /
    (2 (sm (-1 + so) + so) (sc - 2 sm + 3 (1 + sm) so))},
  {qy → (2 sc - sc sm + 2 sm^2 + (2 + sc - 3 sm) (1 + sm) so + (1 + sm)^2 so^2 +
      √(-8 sc (sm (-1 + so) + so) (sc - 2 sm + 3 (1 + sm) so) +
        (2 sc - sc sm + 2 sm^2 + (2 + sc - 3 sm) (1 + sm) so + (1 + sm)^2 so^2)^2)) /
    (2 (sm (-1 + so) + so) (sc - 2 sm + 3 (1 + sm) so))}}
```

Compare with equivalent case for the Autosomal model

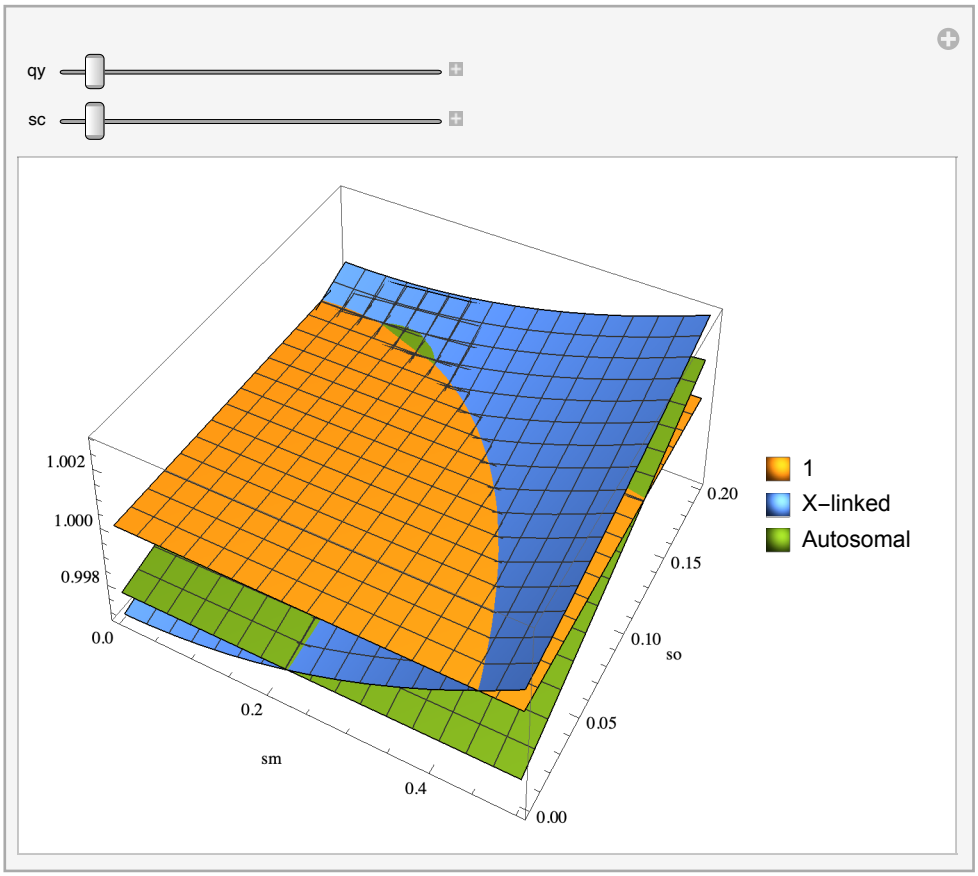
```
In[•]:= (*(-2+hc (sc-qy sc)+(1+ho) qy so+qy sm (-2+so+ho so))/(-2+2 qy (sm (-1+so)+so))*)
  (-2 + hc (sc - qy sc) + (1 + ho) qy so + qy sm (-2 + so + ho so))/(-2 + 2 qy (sm (-1 + so) + so)) /. hc → 1 / 2 /. ho → 1 / 2 //
  FullSimplify
```

```
Out[•]= (-4 + sc - qy sc - 4 qy sm + 3 qy (1 + sm) so)/(-4 + 4 qy (sm (-1 + so) + so))
```

```
In[•]:= λaAuto = (-4 + sc - qy sc - 4 qy sm + 3 qy (1 + sm) so)/(-4 + 4 qy (sm (-1 + so) + so));
  λaXlinked =
    (-2 + sc - qy sc + qy (sm (-2 + so) + so) - √(36 + (-1 + qy)^2 sc^2 + 4 qy sm (18 + (8 + qy) sm) -
        4 qy (1 + sm) (13 + (12 + qy) sm) so + qy (16 + qy) (1 + sm)^2 so^2 -
        2 (-1 + qy) sc (-10 + qy (sm (-2 + so) + so)))) / (-8 + 8 qy (sm (-1 + so) + so));
```

```
In[•]:= Manipulate[Plot3D[{1, (-2 + sc - qy sc + qy (sm (-2 + so) + so) -
          √(36 + (-1 + qy)^2 sc^2 + 4 qy sm (18 + (8 + qy) sm) - 4 qy (1 + sm) (13 + (12 + qy) sm) so +
             qy (16 + qy) (1 + sm)^2 so^2 - 2 (-1 + qy) sc (-10 + qy (sm (-2 + so) + so)))) /
        (-8 + 8 qy (sm (-1 + so) + so)), (-4 + sc - qy sc - 4 qy sm + 3 qy (1 + sm) so) / (-4 + 4 qy (sm (-1 + so) + so))},
       {sm, 0, 0.5}, {so, 0, 0.2}, AxesLabel → Automatic,
       AspectRatio → 1,
       PlotLegends → {1, "X-linked", "Autosomal"}],
      {{qy, 0.05}, 0, 1}, {{sc, 0.01}, 0, 0.2}]
```

Out[•]=




```
In[•]:= Solve[λaAuto == 1, qy] // FullSimplify
        Solve[λaXlinked == 1, qy] // FullSimplify
```

Out[•]= $\left\{\left\{qy \to \frac{sc}{sc + so + sm\ so}\right\}\right\}$

```
Out[•]= {{qy → (2 sc - sc sm + 2 sm^2 + (2 + sc - 3 sm) (1 + sm) so +
            (1 + sm)^2 so^2 - √(-8 sc (sm (-1 + so) + so) (sc - 2 sm + 3 (1 + sm) so) +
               (2 sc - sc sm + 2 sm^2 + (2 + sc - 3 sm) (1 + sm) so + (1 + sm)^2 so^2)^2)) /
          (2 (sm (-1 + so) + so) (sc - 2 sm + 3 (1 + sm) so))},
        {qy → (2 sc - sc sm + 2 sm^2 + (2 + sc - 3 sm) (1 + sm) so + (1 + sm)^2 so^2 +
            √(-8 sc (sm (-1 + so) + so) (sc - 2 sm + 3 (1 + sm) so) +
               (2 sc - sc sm + 2 sm^2 + (2 + sc - 3 sm) (1 + sm) so + (1 + sm)^2 so^2)^2)) /
          (2 (sm (-1 + so) + so) (sc - 2 sm + 3 (1 + sm) so))}}
```

Dynamic version of Fig. 1A: Frequency of mutant *y* at which compensatory *x* allele is favoured by selection.

In[•]:=
```
Manipulate[Plot[{sc/(sc + so + sm so),
  (2 sc - sc sm + 2 sm^2 + 2 so + sc so - sm so + sc sm so - 3 sm^2 so + so^2 + 2 sm so^2 + sm^2 so^2 -
     Sqrt[(-2 sc + sc sm - 2 sm^2 - 2 so - sc so + sm so - sc sm so + 3 sm^2 so - so^2 - 2 sm so^2 - sm^2 so^2)^2 -
       8 sc (-sc sm + 2 sm^2 + sc so - 5 sm so + sc sm so - 5 sm^2 so + 3 so^2 + 6 sm so^2 + 3 sm^2 so^2)]) /
   (2 (-sc sm + 2 sm^2 + sc so - 5 sm so + sc sm so - 5 sm^2 so + 3 so^2 + 6 sm so^2 + 3 sm^2 so^2))},
 {sm, 0, 0.2}, PlotRange → {0, 1}, AxesLabel → {"s_m", "q̃_y"},
 AspectRatio → 1,
 PlotLegends → {"q̃_y Autosomal", "q̃_y X-linked"}],
{{sc, 0.02}, 0, 0.2}, {{so, 0.02}, 0, 0.2}]
```

Out[•]=