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1 The rates of introgression and barriers to genetic exchange between  
2 hybridizing species: sex chromosomes vs. autosomes

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## 15 ABSTRACT

16 Interspecific crossing experiments have shown that sex chromosomes play a major role in reproductive isolation between  
17 many pairs of species. However, their ability to act as reproductive barriers, which hamper interspecific genetic exchange,  
18 has rarely been evaluated quantitatively compared to Autosomes. This genome-wide limitation of gene flow is essential  
19 for understanding the complete separation of species, and thus speciation. Here, we develop a mainland-island model of  
20 secondary contact between hybridizing species of an XY (or ZW) sexual system. We obtain theoretical predictions for the  
21 frequency of introgressed alleles, and the strength of the barrier to neutral gene flow for the two types of chromosomes  
22 carrying multiple interspecific barrier loci. Theoretical predictions are obtained for scenarios where introgressed alleles  
23 are rare. We show that the same analytical expressions apply for sex chromosomes and autosomes, but with different  
24 sex-averaged effective parameters. The specific features of sex chromosomes (hemizyosity and absence of recombination  
25 in the heterogametic sex) lead to reduced levels of introgression on the X (or Z) compared to autosomes. This effect can be  
26 enhanced by certain types of sex-biased forces, but it remains overall small (except when alleles causing incompatibilities  
27 are recessive). We discuss these predictions in the light of empirical data comprising model-based tests of introgression  
28 and cline surveys in various biological systems.

## 29 INTRODUCTION

30 Speciation is a process of gradual accumulation of reproductive barriers in the genome, ultimately leading to a cessation of  
31 gene flow between groups of individuals forming distinct biological species (Dobzhansky 1937). The extent to which barrier  
32 loci reduce interspecific gene flow is a central factor in the study of speciation, as it allows the strength of reproductive  
33 isolation to be quantified as populations diverge (see Ravinet et al. 2017 for a review). Another major advance in the  
34 genetics of speciation has been the discovery, by interspecific crossing experiments, of two extremely robust patterns: (i)  
35 “Haldane’s rule” (Haldane 1922; Schilthuizen et al. 2011), i.e. in species with sex-specific reduced fitness of F1 hybrids,  
36 the affected sex is generally heterogametic; and (ii) the “large-X/Z effect” (Dobzhansky 1936), i.e. the disproportionate  
37 density on the X or the Z chromosome of barrier loci causing hybrid sterility or inviability. These “rules of speciation”  
38 (Coyne and Orr 1989) suggest the existence of universal mechanisms associated with sex chromosomes that may promote  
39 speciation. However, these two lines of research (quantifying the barrier strength vs characterizing the genetic basis of  
40 speciation) have, for the most part, been conducted independently. In particular, there is room for improvement in the  
41 empirical estimation of the barrier to interspecific gene flow due to specific chromosomes, as this information is mainly  
42 available only for model species (Payseur et al. 2018). In consequence, the ability of sex chromosomes to act as barriers  
43 that impede interspecies gene exchange has been little evaluated quantitatively or in a systematic way.

44  
45 The recent explosion of genomic data in the field of speciation has provided empirical evidence across a diversity of  
46 species with different sex chromosome systems (i.e. XY under male heterogamety, and ZW under female heterogamety,  
47 Bachtrog et al. 2014). Indeed, the presence of reproductive barriers along the genome reduces interspecific gene flow  
48 around each barrier locus, leading to a local increase of interspecific differentiation relative to the rest of the genome. This  
49 effect has been widely used to scan genomes for regions that are abnormally differentiated between species, in search of  
50 loci involved in speciation. These studies collectively show a systematically stronger differentiation of sex chromosomes  
51 (X or Z) compared to the autosomes (Presgraves 2018), even after correcting for the different effective population sizes  
52 of sex chromosomes and autosomes. This indicates that sex chromosomes may play a leading role in the isolation of  
53 nascent species. However, caution must be exercised when interpreting this pattern, as measures of relative differentiation  
54 (such as  $F_{ST}$ ; Weir and Cockerham 1984) are sensitive to the level of diversity within species, and whether or not gene  
55 flow occurred during species divergence (Charlesworth 1998; Cruickshank and Hahn 2014). Complementary approaches  
56 addressing this limitation are based on simple summary statistics of absolute divergence (such as  $D_{XY}$ ; Nei and Li 1979),  
57 cline analyses (Barton and Hewitt 1985) and speciation model inferences (Sousa and Hey 2013), but they do not reveal  
58 clear-cut patterns (see Discussion and Table 3).

59  
60 Three main theories have been proposed to explain why sex chromosomes might act as stronger interspecies barriers  
61 than autosomes. First, sex chromosomes may accumulate interspecific barrier loci faster than autosomes. The faster-X  
62 theory (Charlesworth et al. 1987) predicts that this would be the case if incompatible loci first appear as recessive or  
63 partially recessive beneficial mutations (or under a much wider range of dominance levels, if the effective population size

64 for the X relative to autosomes is high enough; Vicoso and Charlesworth 2009). This is because the hemizyosity of the  
65 sex chromosome, i.e. the fact that the heterogametic sex has only one copy of the X/Z, makes selection more efficient  
66 by unmasking sex-linked recessive mutations. Most theoretical and empirical work so far deals with the faster-X theory;  
67 however, empirical evidence in favor of greater efficacy of selection on the sex chromosome is mixed (Meisel and Connallon  
68 2013; but see Charlesworth et al. 2018, who provide fairly solid evidence for adaptive faster-X effects in *Drosophila*).  
69 Instead, other studies have suggested that the accumulation of slightly deleterious mutations may be a major cause of  
70 faster-X/Z, especially in birds, where the variance of male reproductive success strongly decreases the effective population  
71 size of the sex chromosome relative to autosomes, increasing genetic drift (Mank et al. 2010).

72 Another theory, which was initially dismissed but has recently returned to the limelight, is that barrier loci may occur  
73 more readily on the sex chromosomes, because the absence of recombination between the X and the Y (or the Z and  
74 the W) makes them more susceptible to segregation conflicts in the heterogametic sex (e.g. Bachtrog et al. 2019). Since  
75 segregation distorters and their suppressors co-evolve within populations independently, changes in these interactions in  
76 hybrids may cause incompatibilities (Frank 1991; Hurst and Pomiankowski 1991; and see Phadnis and Orr 2009, for an  
77 example of a X-linked meiotic driver associated with hybrid sterility in *Drosophila*). This mechanism thus contrasts with  
78 the classical view that interspecies incompatibilities first appear within species as beneficial mutations (Presgraves 2010).

79 The last theory does not require that sex chromosomes harbor more barrier loci than autosomes, but simply that  
80 they are more exposed to selection, such that sex-linked incompatibilities cause more deleterious effects (than autosomal  
81 incompatibilities) in hybrids. This process has been formalized by the dominance theory (Turelli and Orr 1995, 2000),  
82 which is also based on the hemizyosity of the sex chromosome and assumes that most incompatible mutations act re-  
83 cessively in hybrids. However, it has been experimentally tested (and validated) almost exclusively in *Drosophila* (e.g.  
84 Masly and Presgraves 2007; Cattani and Presgraves 2012; Llopart et al. 2018, but see Matsubara et al. 2015 for a study in  
85 rice). Assessing its generality remains difficult, due to the experimental challenge of measuring the dominance of mutations.

86  
87 The present work aims to understand the role of the X or Z chromosome (note that we do not consider the Y or W)  
88 in the formation of new biological species by focusing on the impact of sex-linked reproductive barriers on the reduction  
89 of gene flow between hybridizing species, rather than on their rate of establishment during species divergence. To do this,  
90 we extend the theoretical framework of Barton and Bengtsson who modeled the introgression of incompatible autosomal  
91 genomic blocks between hybridizing species (Barton 1983), and quantified their effect on the effective migration rate at  
92 a linked neutral marker (Barton and Bengtsson 1986). With multiple barrier loci, Barton and Bengtsson showed that  
93 neutral gene flow is significantly reduced over most of the genome only if the number of loci involved in reproductive  
94 isolation is large enough that any neutral marker becomes closely linked to at least one barrier locus. While they showed  
95 that this result is little influenced by the position of the neutral marker along a given chromosome, the effect of being  
96 located on the sex chromosomes rather than on autosomes was not addressed.

97 Yet, for the above reasons, one may expect to see differences between the two types of chromosomes: in the heteroga-  
98 matic sex, the greater efficacy of selection against deleterious mutations and the lack of recombination over much of the X  
99 or Z are expected to reduce the rate of introgression of sex-linked neutral markers relative to autosomal ones, even when

100 there is no difference between the number, density or selective effects of sex-linked and autosomal incompatibilities. More-  
101 over, because sex chromosomes are involved in sex determination, they are inherited differently between males and females  
102 and thus spend different periods of time in each sex, making them sensitive to sex-specific evolutionary forces (Hedrick  
103 2007), such as sex-biased migration, sex-biased selection, or achiasmy (the absence of recombination in the entire genome  
104 of the heterogametic sex, which causes the X/Z to have more recombination than the autosomes; e.g. see Betancourt  
105 et al. 2004). Since these evolutionary forces determine the intensity of the barrier to gene flow (Barton and Bengtsson  
106 1986), their sex specificity must be taken into account in order to quantify the role of sex chromosomes in speciation. An  
107 additional complication comes from the phenomenon of dosage compensation that has been found in many species with  
108 a degenerated sex chromosome (Mank 2013). The degeneration of the Y (or the W) causes a dose difference between the  
109 sexes, so that sex-linked loci are transcribed half as much in the heterogametic sex. Dosage compensation then consists of  
110 restoring similar levels of the X (or Z) gene product in males and females. Among the diversity of mechanisms that have  
111 evolved (Gu and Walters 2017), some may lead to stronger selection against hybrids, and thus should enhance the ability  
112 of sex chromosomes to act as interspecific barriers.

113

114 To date, only a handful of theoretical studies have examined the role of sex chromosomes in speciation. The dominance  
115 theory (Turelli and Orr 1995, 2000) offered a simple explanation for the two “rules of speciation”, building upon the  
116 allopatric model of postzygotic isolation elaborated by Dobzhansky (1937) and Muller (1940). This seminal model involves  
117 deleterious epistasis between alleles at different loci (i.e., Dobzhansky-Muller incompatibilities, or DMIs). In a model  
118 of parapatry, Hoellinger and Hermisson (2017) found that X-linked DMIs are more easily maintained in the presence  
119 of interspecies gene flow than autosomal DMIs. All these studies address the question of the origin, maintenance or  
120 accumulation of incompatibilities, but do not explicitly quantify their long-term effect as barriers to neutral gene flow.  
121 In fact, it may be that DMIs are generally ineffective at maintaining genome-wide differentiation in the face of gene flow  
122 (while they can create strong selection against F1 and F2 hybrids) as shown by simulations (Lindtke and Buerkle 2015).

123 In contrast, Muirhead and Presgraves (2016) modeled the effect of a locally deleterious mutation (either intrinsically  
124 incompatible with the local genome, or extrinsically incompatible with the local habitat) on gene flow at a linked neutral  
125 marker. Under a weak migration/strong selection regime, they showed that selection against incompatible mutations  
126 reduces the flow of neutral markers more strongly on the sex chromosomes than on autosomes, the effect being greater  
127 when mutations are recessive.

128 While all these studies are in line with a reduced introgression probability on the sex chromosomes relative to au-  
129 toosomes, they are limited to single/pairs of barrier loci, even though speciation is a complex, and probably multigenic,  
130 process. Thus, it is essential to develop more realistic models that account for the effects of multiple linked barrier loci.  
131 This can have important implications; for example, Barton (1983) showed that linkage between incompatible autosomal  
132 alleles, which governs the extent to which they act as a single unit of selection, is a key determinant of their level of  
133 introgression and the barrier to gene flow they induce. A multilocus theory would thus allow us to better understand the  
134 differences between sex-linked and autosomal interspecies barriers, and the causes underlying these differences, such as  
135 the role of recombination. In addition, these models provide predictions of introgression patterns along chromosomes, and



136 thus are critical to capture the signature of polygenic barriers in genomic data.

137

138 Here, we address this gap by extending previous multilocus predictions (Barton 1983; Barton and Bengtsson 1986) to  
139 the case of sex chromosomes. We introduce a model where chromosomes contain multiple interspecies incompatibilities,  
140 and quantify (i) their frequency on sex chromosomes relative to autosomes in populations under migration-selection balance  
141 and (ii) the effect of selection against these incompatibilities on the neutral gene flow between two hybridizing species. Our  
142 aim is to understand (i) to what extent the particularities of the sex chromosomes: hemizygoty and lack of recombination  
143 on the X/Z in the heterogametic sex (we ignore pseudoautosomal regions) affect their ability to hinder interspecific gene  
144 flow, and (ii) the influence of sex-biased processes and dosage compensation in different sexual systems (XY and ZW). Our  
145 model is general in the sense that it applies to both male heterogamety (males are XY and females are XX) and female  
146 heterogamety (females are ZW and males are ZZ), and it allows us to address the effects of various kinds of sex-specificity  
147 by introducing sex-specific parameters. Unless otherwise stated, we assume an XY system, as the results apply to ZW  
148 systems by interchanging males and females.

## 149 MODEL AND METHODS

150 We consider a population with males and females. The homogametic sex carries two copies of the sex chromosome (X or  
151 Z) and two copies of each autosome; the heterogametic sex carries one copy of the sex chromosome and two copies of each  
152 autosome. We focus on X/Z chromosomes (or regions of them) where there are no homologous genes on the Y/W, and  
153 which do not undergo recombination in the heterogametic sex.

154 Hybridisation between two incompletely isolated species occurs in a mainland-island setting. Migration is assumed to  
155 be one-way from a donor to a recipient species, although our results hold for two-way exchange as long as migration is weak  
156 and introgressed alleles rare. We model the introgression of a single medium-sized block of genome with multiple loci that  
157 influence fitness in the recipient species. We assume that the two species are fixed for different alleles at these loci, and  
158 that each introduced allele is deleterious in the recipient species, either because it is not adapted to the local environment  
159 or to the local genetic background. Throughout we neglect *de novo* mutations and segregating variants (with effects on  
160 fitness) that may be private to one of the two species. We also neglect the effect of loosely linked or unlinked incompatible  
161 alleles (spread across the entire genome), since these would affect introgression of both autosomal and X-linked blocks to  
162 a similar extent via an increase in the fitness variation and a resultant reduction in the effective population size (Barton  
163 1995). We thus focus here on a comparison of the introgression dynamics of a single X-linked vs autosomal block.

164

165 In each generation, a fraction  $m_F$  of females and a fraction  $m_M$  of males are replaced by immigrant females and males  
166 respectively. Immigrants are drawn from a donor species which is fixed for the deleterious block. Thus, if the block is  
167 on the X, immigrant females introduce two identical copies and immigrant males a single copy into the recipient species,  
168 while if it is autosomal, any immigrant introduces two copies of the deleterious block. The block is assumed to carry  $L$   
169 equally spaced loci with deleterious alleles of equal effect, which multiplicatively reduce individual fitness in the recipient

170 species. Thus the relative fitness of a female heterozygous for  $y$  and homozygous for  $y'$  X-linked introgressed alleles is  
171  $v_F(y, y') = e^{-y s_F - y' s_{hom, F}}$ , while the fitness of a male who carries  $y$  introgressed alleles is  $v_M(y) = e^{-y s_M}$ . Here,  $s_F$   
172 and  $s_{hom, F}$  denote heterozygous and homozygous effects per allele in females, and  $s_M$  the hemizygous effect in males;  
173 unlike in the more standard notation where  $h s$  refers to the heterozygous effect. Our choice of notation is motivated  
174 by the fact that, to the first order in  $sL$ , introgression depends only on heterozygous effects and is independent of the  
175 dominance coefficient,  $h$  (see below). Fitness in the autosomal case can be defined analogously. The per generation rate  
176 of recombination between adjacent selected loci is denoted by  $c_F$  in females (for either autosomal or X-linked blocks) and  
177  $c_M$  in males (only for autosomes; no recombination occurs between the X and Y in males). Unless stated otherwise, we  
178 always assume autosomal recombination in the heterogametic sex.

179 We also analyze the introgression of a neutral marker linked to the deleterious genomic block. For simplicity, we focus  
180 on the so-called “rod” model of Barton and Bengtsson (1986), where the neutral marker lies at one end of the block, with  
181 the rate of recombination between the neutral marker and the terminal selected locus  $\alpha c_F$  in females and  $\alpha c_M$  in males.  
182 Here  $\alpha$  parameterizes the map distance between the neutral marker and nearest selected locus, relative to the interlocus  
183 map distance. Thus, in this model, the neutral marker can escape into the recipient background via a single recombination  
184 event. Our analysis can be generalized to other marker configurations, following Barton and Bengtsson (1986) (e.g. the  
185 “embedded” model, Figure S7).

186

187 We derive analytical results by assuming that: (i) the frequency of the introgressing block (and its descendant frag-  
188 ments) is low so that matings between individuals carrying introgressed material can be neglected, and (ii) the deleterious  
189 block is short enough that multiple crossovers within the deleterious block can also be neglected. This implies that any  
190 individual male or female carries at most a single deleterious block. In particular, immigrant females (who carry two copies  
191 of the selected blocks) must necessarily pass on only one of these to their offspring (who are likely to inherit the other  
192 chromosome from a father bearing no introgressed material). Thus, in this regime, introgression is largely independent of  
193 the homozygous effect of deleterious alleles and is governed by the heterozygous (in females) and hemizygous (in males)  
194 selective effects.

195

196 In the following, we first present dynamical equations for the evolution of deleterious fragments of the introduced genome  
197 and solve these at equilibrium (i.e., migration-selection-recombination balance) to obtain analytical predictions for the  
198 equilibrium frequencies of deleterious X-linked blocks. We then calculate the effective migration rate of neutral markers  
199 into the recipient species, thus quantifying the reduction in gene flow at a neutral marker due to linkage to interspecific  
200 barrier loci. We also test these analytical expressions (i.e., the exact formulae and the weak-selection approximations)  
201 against individual-based simulations. We first analyze a basic model assuming co-dominance, multiplicative effects and  
202 no sex-specific forces, and then extend to sex-specificities, dominance and epistasis. Key notation is summarized in Table  
203 1.

## 204 Introgression of multilocus deleterious blocks

### 205 Autosomal introgression:

206 We consider how selection against multiple deleterious alleles influences introgression of an incompatible autosomal block  
 207 in a regime where the vast majority of individuals carry at most one fragment of the block. While Barton (1983) considered  
 208 the simplest case of haploid introgression and no sex differences, we generalize to the case where selection coefficients,  
 209 migration rates and recombination rates may differ between males and females (see Appendix A, Supp. Text). For small  
 210 parameters (i.e.  $sL$ ,  $cL$ ,  $m \ll 1$ ), we can take a continuous time limit by approximating  $e^{-sy} \approx 1 - sy$  and, neglecting  
 211 all terms that are second order in small parameters, e.g.,  $O(s^2)$ ,  $O(ms)$  etc. Then the frequencies  $P_A(y, t)$  of introgressed  
 212 fragments with  $y$  deleterious alleles at time  $t$  satisfy dynamical equations which are identical to those in Barton (1983)  
 213 but with  $s_A$ ,  $m_A$  and  $c_A$  given by the sex-averaged rates ( $s_A = \frac{s_F + s_M}{2}$ ,  $m_A = \frac{m_F + m_M}{2}$ ,  $c_A = \frac{c_F + c_M}{2}$ ; see Table 2):

$$\frac{\partial P_A(y, t)}{\partial t} = -[s_A y + c_A(y - 1)] P_A(y) + 2c_A \sum_{y'=y+1}^L P_A(y') + m_A \delta_{y,L} \quad (1)$$

214 Here  $\delta_{y,L}$  equals 1 for  $y = L$ , and is zero otherwise. The first term represents the loss of the introgressed fragment  
 215 due to selection against deleterious alleles and backcrossing with the recipient species; the second term corresponds to  
 216 the creation of the fragment by splitting of larger fragments; the third term represents the influx of the full block (with  
 217  $L$  deleterious alleles) due to migration from the donor species. Note that eq. (1) is linear in  $P_A(y)$  since we assume  
 218 introgressing blocks to be rare and terms of the form  $P_A(y)P_A(y')$  (which arise from mating between individuals carrying  
 219 introgressing blocks) to be negligible. For the same reason, we can ignore terms of order  $m_A^2$ ; thus our results also hold  
 220 for weak bidirectional migration.

221 Eq. (1) can be solved to obtain the block frequencies  $P_A(y)$  at equilibrium and the average equilibrium frequency of  
 222 deleterious alleles (averaged over all loci on the autosomal block):  $\bar{p}_A = \sum_{y=1}^L P_A(y) \frac{y}{L}$  (see eq. (12) in Appendix A).

223  
 224 A key parameter governing introgression is  $\theta_A = \frac{s_A}{c_A}$ , the ratio of selective effect to recombination rate per locus, which  
 225 determines the extent of coupling between loci (Barton 1983). For large  $L$ , the average deleterious allele frequency  $\bar{p}_A$  can  
 226 be approximated as:

$$\bar{p}_A \sim \begin{cases} \frac{m_A}{S_A} L^{1-2\theta_A} & \theta_A \ll 1 \\ \frac{m_A}{2S_A} (\gamma + \log[4L]) & \theta_A = 1 \\ \frac{m_A}{S_A} \left(1 + \frac{1}{\theta_A}\right) & \theta_A \gg 1 \end{cases} \quad (2)$$

227 where  $S_A = s_A L$  and  $\gamma = 0.577216$  is Euler's constant. One can define the effective selection coefficient,  $s_A^* = \frac{m_A}{\bar{p}_A}$ , as  
 228 the selective pressure at a single locus that would be needed to produce the observed average frequency of the deleterious  
 229 alleles (Barton 1983). By extension, one can also define the effective number of loci  $n_{eA} = \frac{s_A^*}{s_A}$ , that, if fully linked, would  
 230 have produced the same effect on deleterious allele frequency as the full deleterious block (Figure S1). Note that in the  
 231 limit  $c \rightarrow 0$ , i.e., for perfect linkage between deleterious alleles,  $n_{eA}$  must approach  $L$ .

232 For  $\theta_A \gg 1$ , (i.e., very strong coupling between loci), the deleterious allele frequency depends only on the net selective  
 233 disadvantage  $S_A = s_A L$  of the block, and is independent of the number of loci on the block (for large  $L$ ). It also follows  
 234 that the effective selection coefficient per locus, given by  $S_A/(1 + \frac{1}{\theta_A})$ , approaches the net selective effect  $S_A$  for very  
 235 large values of  $\theta_A$ . In contrast, for  $\theta_A \ll 1$  (i.e., weak coupling between loci), each locus experiences an effective selective  
 236 disadvantage (given by  $S_A L^{2\theta_A - 1} = s_A L^{2\theta_A}$ ) which is proportional to its own selective effect  $s_A$ , but also depends weakly  
 237 on  $L$  (the number of selected loci that are loosely linked to it). Crucially, in this regime, the effective selection per  
 238 locus decreases and the deleterious allele frequency increases as we consider introgression scenarios where the same total  
 239 selective effect is due to a larger and larger number of loci of weaker effect (i.e., on increasing  $L$  while keeping  $S_A = s_A L$  and  
 240  $C_A = c_A L$  constant). Thus for weak coupling between loci, this analysis (which assumes that deleterious allele frequency  
 241 is low) remains valid only for small values of  $L$ , more specifically for  $\frac{m_A}{S_A} L^{1-2\theta_A} \ll 1$ .

### 242 X-linked introgression:

243 We now analyze introgression of an X-linked block. Let  $P_F(y)$  and  $P_M(y)$  denote the fraction of females and males  
 244 who carry a single copy of, i.e., are respectively heterozygous and hemizygous for, an X-linked introgressed block with  $y$   
 245 deleterious alleles. Thus, in a population of size  $N$  with a 1 : 1 sex ratio, the number of X-linked blocks with  $y$  deleterious  
 246 alleles must be  $\frac{N}{2}(P_F(y) + P_M(y))$ , while the total number of X chromosomes is  $\frac{3}{2}N$ . Thus, the frequency of X-linked  
 247 introgressed blocks with  $y$  deleterious alleles is given by  $P_X(y) = \frac{P_F(y) + P_M(y)}{3}$ . In Appendix A, we derive dynamical  
 248 equations for the evolution of  $P_F(y, t)$  and  $P_M(y, t)$ . In the continuous time limit, i.e., assuming  $sL, cL \ll 1$ , and  $m \ll s$ ,  
 249 these can be combined into a single equation for the evolution of  $P_X(y, t)$ :

$$\frac{\partial P_X(y, t)}{\partial t} = - \left[ \frac{2s_F}{3}y + \frac{s_M}{3}y + \frac{2c_F}{3}(y-1) \right] P_X(y) + \frac{4c_F}{3} \sum_{y'=y+1}^L P_X(y') + \left( \frac{2m_F}{3} + \frac{m_M}{3} \right) \delta_{y,L} \quad (3a)$$

$$250 \quad P_F(y) = 2P_X(y) \quad P_M(y) = P_X(y) \quad (3b)$$

251  
 252 This equation is identical in form to eq. (1) (which describes the dynamics of the autosomal block distribution), but  
 253 with  $s_A$  replaced by  $s_X = \frac{2s_F + s_M}{3}$ ,  $m_A$  by  $m_X = \frac{2m_F + m_M}{3}$  and  $c_A$  by  $c_X = \frac{2c_F}{3}$  (see Table 2). The sex-averaged effective  
 254 parameters  $m_X$ ,  $s_X$  and  $c_X$  are weighted sums of the corresponding male and female parameters. Female migration con-  
 255 tributes twice as much as male migration to  $m_X$  simply because any female migrant introduces two X-linked blocks while  
 256 a male migrant carries one. Similarly, the sex-averaged selection coefficient  $s_X$  is the sum of male and female components,  
 257 with the contribution of  $s_F$ , the heterozygous selective effect per allele in females, being twice that of  $s_M$ , the hemizygous  
 258 effect in males. Note that this is despite the fact that almost all females, with the exception of immigrant females, carry  
 259 only one copy of any X-linked deleterious block (under the assumption of low migration). The 2:1 contributions emerge  
 260 nevertheless because there are twice as many females who are heterozygous (for any X-linked deleterious block) as males  
 261 who are hemizygous ( $P_M(y) \sim \frac{P_F(y)}{2}$ , see eq. (3b)): this, in turn, is simply because females can inherit the block from  
 262 either parent, while males only inherit it from their mothers. Thus any X-linked deleterious allele spends twice as much  
 263 time in the heterozygous state in females than in the hemizygous state in males, causing it to be more sensitive to selection

264 in females. The 2:1 contributions of female and male recombination rates (where the latter is assumed to be zero) to the  
 265 sex-averaged recombination fraction  $c_X$  can be explained similarly.

266

267 Equation (3a) can be solved to obtain an explicit expression for the equilibrium  $P_X(y)$  (details in Appendix A):

$$P_X(L) = \frac{m_X}{s_X} \frac{\theta_X}{(L-1) + \theta_X L}, \quad P_X(y) = 2 \frac{m_X}{s_X} \frac{\prod_{i=1}^{L-y-1} [\theta_X(y+i) + (y+i+1)]}{\prod_{i=0}^{L-y} [\theta_X(y+i) + (y+i-1)]} \theta_X \quad y < L \quad (4)$$

268 Here  $\theta_X = \frac{s_X}{c_X}$  is a measure of the strength of coupling between deleterious alleles on the X chromosome. As in the  
 269 autosomal case, equation (4) can be used to calculate the average frequency of deleterious alleles, averaged over all loci  
 270 on the X chromosome:  $\bar{p}_X = \sum_{y=1}^L P_X(y) \frac{y}{L}$ . Since eq. (1) and (3) have the same form, this is given by the same expressions  
 271 as in eq. (2), but with  $s_X$  and  $m_X$  replaced by  $s_A$  and  $m_A$  respectively. As before, we expect our analysis to become  
 272 inaccurate when the X chromosome contains a large number of weakly linked and weakly selected deleterious alleles, i.e.,  
 273 for  $\theta_X \lesssim 1$  and  $\frac{m_X}{s_X} L^{1-2\theta_X} \ll 1$ .

274

275 Equations (1) and (3) reveal a simple correspondence between introgression on autosomes and on X chromosomes,  
 276 even when there is multilocus selection against introgression (for weak selection and recombination:  $sL, cL \ll 1$ ). More  
 277 specifically, they suggest that the distribution of lengths of X-linked introgressing blocks should be nearly identical to  
 278 the distribution of autosomal introgressing blocks, upon comparing autosomes and sex chromosomes with  $\frac{m_X}{s_X} = \frac{m_A}{s_A}$  and  
 279  $\theta_X = \theta_A$  (this prediction is verified in simulations, see Figure S2A). Conversely, if we compare autosomal and X-linked  
 280 blocks with identical genetic architectures and no sex-bias, then we have  $\theta_X = \frac{3}{2}\theta_A$ , i.e., deleterious alleles on the X  
 281 are more strongly coupled to each other (than on autosomes), resulting in lower rates of introgression. We quantify this  
 282 precisely in the Results using our analytical expressions for  $P_X(y)$  and  $P_A(y)$  (eq. (4)).

283 Note that this correspondence between X-linked and autosomal introgression also applies to the dynamics of block  
 284 length distributions (eq. (3a), see also Figure S2B for verification in simulations). This implies that the time-dependent  
 285 distribution of block lengths can be described by the same mathematical results as for the autosomal case (Appendix in  
 286 Baird 1995). Thus we do not consider dynamics any further here, and focus only on patterns at equilibrium.

## 287 Multilocus barrier to neutral introgression

### 288 Autosomal barrier:

289 We next investigate how deleterious genomic blocks act as genetic barriers to neutral gene flow by calculating the rate  
 290 at which an allele at a linked neutral marker (fixed in the donor species) increases in the recipient species, where it is  
 291 initially absent. We again assume that introgression is rare and that multiple crossovers within the deleterious block can  
 292 be neglected. Therefore, the neutral marker can only be found in the recipient species associated with a consecutive series  
 293 of between 0 and  $L$  locally deleterious alleles, and paired with a chromosome of the recipient species.

294

295 Following Bengtsson (1985), the strength of the barrier ( $b_A$ ) is defined as  $\frac{m_A}{me_A}$ , where  $m_A$  is the raw migration rate  
 296 on autosomes and  $me_A$  is the effective migration rate defined as  $\frac{\delta u_A(0)}{\Delta u_A}$ . Here,  $\Delta u_A$  is the difference in allele frequency of  
 297 the neutral marker between the donor and recipient species (and is 1, by definition), while  $\delta u_A(0)$  is the rate of increase  
 298 of the neutral marker in the recipient background. The rate of increase  $\delta u_A(0)$  can be calculated by tracking the rate  
 299 at which the neutral marker is transferred between different genetic backgrounds (with different numbers of deleterious  
 300 alleles) at equilibrium (Barton and Bengtsson 1986; see also Appendix B, Supp. Text). For  $sL, cL \ll 1$ , the autosomal  
 301 barrier strength  $b_A$  under this “rod” configuration can be written as (Barton and Bengtsson 1986):

$$b_A = \frac{\Gamma\left(L + \frac{\alpha + \theta_A}{1 + \theta_A}\right) \Gamma\left(\frac{\alpha}{1 + \theta_A}\right)}{\Gamma\left(L + \frac{\alpha}{1 + \theta_A}\right) \Gamma\left(\frac{\alpha + \theta_A}{1 + \theta_A}\right)} \quad (5)$$

302 where  $\Gamma$  refers to the Gamma function.

303 The barrier strength is thus determined by the coupling coefficient ( $\theta_A = \frac{s_A}{c_A}$ ) and the proximity of the neutral marker  
 304 to the selected block ( $\alpha$ ). The dependence of the barrier strength on  $\theta_A$  is qualitatively similar to the dependence of  
 305 the equilibrium frequency of locally deleterious alleles. When  $\theta_A$  is large ( $sL \gg cL$ ), all that matters in order for  
 306 the neutral marker to introgress is to recombine away from the entire deleterious block in a single step, while when  $\theta_A$  is  
 307 small ( $sL \ll cL$ ), pairwise interactions between the neutral marker and each selected locus modulate neutral introgression.

308

309 Concerning the proximity of the neutral marker to the incompatible block, the smaller  $\alpha$ , the stronger the barrier,  
 310 since the neutral marker is then closely linked to the block. When  $\alpha \ll 1$ , the barrier strength is proportional to the effect  
 311 of the closest deleterious locus, but the additional effect of the other deleterious loci on the block reduces gene flow well  
 312 below the single-locus expectation. When  $\alpha \gg L$ , the deleterious block acts as a single unit with regard to the neutral  
 313 marker, and thus the barrier strength is close to that produced by a single locus with total effect  $sL$  at a distance  $\alpha c$  from  
 314 the marker.

### 315 X-linked barrier:

316 We now consider the case of a neutral marker on the X chromosome. We obtain an explicit expression for the strength  
 317 of the barrier to neutral X-linked gene flow, under the same assumptions as in the autosomal case (see Appendix B for  
 318 further details, Supp. Text). This is the same expression as in eq. (5), but with  $\theta_A$  substituted by  $\theta_X$ :

$$b_X = \frac{\Gamma\left(L + \frac{\alpha + \theta_X}{1 + \theta_X}\right) \Gamma\left(\frac{\alpha}{1 + \theta_X}\right)}{\Gamma\left(L + \frac{\alpha}{1 + \theta_X}\right) \Gamma\left(\frac{\alpha + \theta_X}{1 + \theta_X}\right)} \quad (6)$$

319 In Appendix B (Supp. Text), we show that, in the limit of very tight linkage between barrier loci (for which we expect  
 320 the deleterious block to act as a single locus with net effect  $S_X = s_X L$ ), our results for barrier strength are consistent  
 321 with those of Muirhead and Presgraves (2016), who consider a neutral marker linked to a *single* incompatible locus.

## 322 Model extensions

323 An important feature of the above equations is that any kind of sex bias can be encapsulated by the composite parameters  
324  $\theta = \frac{s}{c}$  and  $\frac{m}{s}$ , at least for weak migration, selection and recombination. Thus equations (3) and (4) describe X-linked  
325 introgression in various scenarios.

### 326 Dosage compensation and sex-biased forces:

327 Dosage compensation is likely to influence fitness effects of incompatibilities in complex ways that are poorly understood.  
328 For simplicity, we focus on two different fitness models, which represent two extreme possibilities. We first analyze a “basic  
329 model” with co-dominant alleles in which heterozygous selective effects in females are assumed to be half the corresponding  
330 homozygous effects and equal to the hemizygous selective effects in males (i.e.,  $s_M = s_F = s_{hom,F}/2$ ). This parameter  
331 setting for our “basic model” is justified to the extent that we first want to focus solely on the effects of lower effective  
332 recombination on the sex chromosomes. Moreover, it gives the most conservative estimate of how much barrier strength is  
333 increased on the sex chromosome relative to autosomes, because stronger deleterious effects in males (relative to females)  
334 further enhance the strength of coupling ( $\theta$ ) on the X relative to autosomes (if  $s_F$  and  $c$  are comparable for the two  
335 chromosome types).

336 We then consider a more realistic parameter setting, where we explicitly account for the fact that a single deleterious  
337 allele is (typically) more detrimental in the heterogametic sex than a single deleterious allele in the homogametic sex. This  
338 setting follows from a model with dosage compensation, where the fitness of a male carrying a hemizygous XY deleterious  
339 block is the same as that of a female carrying a homozygous XX block (i.e.,  $s_M = s_{hom,F} > s_F$  for sex-linked alleles).  
340 Under this assumption, the selection coefficient in males,  $s_M$ , is larger in magnitude than in the “basic model”, due to the  
341 larger amounts of deleterious gene products in the heterogametic sex. This model can describe any mechanism of dosage  
342 compensation which leads to similar total levels of activity of the sex chromosome in both sexes. It is thus reasonable for  
343 groups in which dosage compensation evolved by up-regulation of the X (or Z) in the homogametic sex. But it can also  
344 be applied to groups in which the X (or Z) is initially over-expressed in both sexes, then secondarily down-regulated in  
345 the homogametic sex. Note that the assumptions about the influence of dosage compensation on selective effects can be  
346 easily relaxed; our mathematical results are very general and can be directly applied to any model of dosage compensation  
347 (which would result in different  $s_F/s_{hom,F}$  and  $s_F/s_M$  ratios).

348 We then model sex-specific selection on deleterious alleles by assuming stronger selection coefficients on XY males (i.e.,  
349  $s_M > s_F$ ) or on ZZ males (i.e.,  $s_F > s_M$  in our XY notation). We study sex-biased migration by setting  $m_M \neq m_F$ , and  
350 achiasmy by setting  $c_M = 0$  on autosomes in males (or  $c_F = 0$  in a ZW system), such that there is no recombination on  
351 any chromosome in the heterogametic sex.

### 352 Beyond the weak selection approximation:

353 The analytical predictions presented so far assume that selection against hybrids is weak, i.e.,  $S = sL \ll 1$ , (for a given  $\theta$ ,  
354  $m/S$ , and  $L$ ). Under weak selection, the frequency of hemizygous males (carrying a particular X-linked deleterious block)  
355 is found to be half the corresponding frequency of heterozygous females, i.e.,  $P_M(y) \sim P_F(y)/2$ . This forms the basis of

356 a relatively simple description of X-linked introgression in terms of sex-averaged X-linked parameters (equations (4) and  
357 (6)).

358 Under stronger selection, homozygous female immigrants may have strongly reduced fitness as compared to hemizy-  
359 gous males: this reduces the relative contribution of females to the next generation, increasing the ratio  $P_M(y)/P_F(y)$   
360 above 1/2. Moreover, this effect is sensitive to the dominance coefficient of deleterious alleles ( $h$ ), which determines the  
361 relative fitness of homozygous and heterozygous females. However, even under these scenarios (i.e. strong selection and  
362 dominance/recessivity), male and female genotypic frequencies satisfy a set of linear equations, as long as the frequency of  
363 deleterious alleles in the recipient species is sufficiently low that second order terms in  $P(y)$  can be neglected. In Appen-  
364 dices A and B (Supp. Text), we present these more general predictions for the equilibrium frequencies  $P_F(y)$  and  $P_M(y)$   
365 as well as for the barrier strengths, for both autosomal and X-linked introgression. These are more accurate when selection  
366 against hybrids is strong (see Results), but they are considerably more involved than the weak-selection expressions in  
367 eqs. (4) and (6), and cannot be expressed in terms of sex-averaged effective parameters (i.e.  $s_X$ ,  $m_X$ ,  $c_X$ ).

## 368 Individual-based simulations

369 The analytical expressions for the deleterious allele frequency (eq. (4) which is valid for  $sL$ ,  $cL$  and  $m \ll 1$ ; eq. (16) in  
370 Appendix A which is also valid for strong selection) and for the barrier strength (eq. (6); eq. (30) in Appendix B) assume  
371 that: (i) drift is negligible ( $Ns \gg 1$ ); (ii) multiple crossovers are rare ( $cL \ll 1$ ); and (iii) introgressed alleles are rare  
372 enough that most individuals carry at most one introgressed block. The last assumption is the most critical, as its validity  
373 depends on the value of  $\theta$ . Therefore, we compare our analytical results to forward-in-time individual-based simulations  
374 (see Supp. Text for more details) with  $Ns \gg 1$  and  $cL \ll 1$ , but in different coupling regimes, for which assumption (iii)  
375 may not hold.

## 376 RESULTS

377 Extending previous results (Barton 1983; Barton and Bengtsson 1986) to the case of sex chromosomes, we have derived  
378 analytical expressions for the equilibrium frequency of multilocus sex-linked incompatibilities, and their strength as barriers  
379 against gene exchange between two hybridizing species (eqs. (4) and (6), see also Appendices A and B). We now focus  
380 on the biological consequences of there being different sex-averaged effective parameters (Table 2 and S1). We summarize  
381 the influence of (i) the genetic architecture of the barrier between species, (ii) sex-biased forces and dosage compensation,  
382 and (iii) other model extensions (dominance and epistasis, see Supp. Text). We restrict attention to the effect of each  
383 factor separately, although combined effects are discussed for the interpretation of empirical data (see Discussion). To be  
384 consistent with the mathematical simplifications in our model (see Model and Methods), we choose  $sL$  to be in the range  
385 0.01 to 0.1,  $cL$  to be in the range 0.025 to 0.25 and migration to be weak relative to selection coefficients ( $m = 0.001$ ). In  
386 simulations, we take population sizes to be sufficiently large ( $N = 10^5$ ) to conform with the deterministic regime.



## 387 Genetic architecture of the barrier between species

### 388 Effects of coupling between barrier loci, and of the migration-to-selection ratio:

389 Validating Barton (1983)'s autosomal results, we find that the equilibrium frequency of deleterious alleles decreases as  
390 coupling between selected loci becomes stronger, i.e., as  $\theta$  increases (Figure 1A). When coupling between barrier loci is  
391 strong ( $\theta_A = 2$ , red; Figure 1A), the deleterious block acts as the unit of selection, so that the deleterious allele frequency  
392 in the recipient species ( $\bar{p}_A$ ) is lower than under intermediate coupling ( $\theta_A = 1$ , black; Figure 1A). When coupling is  
393 weak ( $\theta_A = 0.2$ , blue; Figure 1A), recombination breaks the genomic block down to smaller sub-blocks of weaker selective  
394 disadvantage, resulting in a much higher equilibrium frequency of the introgressing alleles. Note that when  $\theta < 1$  and  
395  $L$  is large, the analytical predictions break down:  $\bar{p}_A$  obtained by simulations (open symbols) is much higher than the  
396 predicted values (lines and filled symbols). Coupling has a similar effect on gene flow at a neutral marker, i.e. effective  
397 neutral migration decreases as  $\theta$  increases (and the barrier strength correspondingly increases; Figure 2), which corrob-  
398 orates Barton and Bengtsson (1986)'s autosomal results. The equilibrium frequency of deleterious alleles in the recipient  
399 species shows a strong dependence on  $\frac{m}{S}$  (the ratio of the migration rate to the selective effect  $S = sL$  of the full block) and  
400 is in fact equal to  $\frac{m}{S}$  when selected loci are completely linked. Even with incomplete linkage (i.e., strong to intermediate  
401 coupling), equilibrium allele frequency is nearly proportional to  $\frac{m}{S}$ : for example, the autosomal equilibrium frequency for  
402 low  $\frac{m}{S}$  ( $\frac{m_A}{S_A} = 0.1$ , blue; Figure 1B) is  $\sim 10$  times higher relative to that in the high  $\frac{m}{S}$  regime ( $\frac{m_A}{S_A} = 0.01$ , red; Figure  
403 1B), nearly independently of the number of barrier loci.

404  
405 We show that these patterns also hold for the sex chromosomes, but with a consistently lower equilibrium frequency  
406 and higher barrier strength (Figure 1A and Figure 2). This is because deleterious alleles on the sex chromosome are more  
407 strongly coupled to each other than on autosomes (i.e.,  $\theta_X = \frac{3}{2}\theta_A$ ; Table S1), while  $\frac{m}{S}$  ratios are equal for autosomes and  
408 sex chromosomes (i.e.,  $\frac{m_X}{S_X} = \frac{m_A}{S_A}$ ; Table S1), assuming identical genetic architectures of hybrid incompatibility for both  
409 (i.e. same  $s_F$ ,  $s_M$ ,  $c_F$  and  $L$ ). These relationships result in a lower sex-linked equilibrium frequency when the barrier is  
410 multilocus ( $\frac{\bar{p}_X}{\bar{p}_A} < 1$  for  $L > 1$ ; Figure 1A), and a higher barrier strength on the sex chromosome even when there is a  
411 single barrier locus ( $\frac{b_X}{b_A} > 1$  for  $L \geq 1$ ; Figure 2), since the neutral marker and the selected locus are then more strongly  
412 linked. Note that the difference between X-linked and autosomal introgression probabilities is quite pronounced for low  
413 to intermediate values of  $\theta$  (Figure 1A), but becomes weaker for  $s_F, s_M \gg c$ , i.e.,  $\theta \gg 1$  (red; Figure S4A), even though  
414 X-linked incompatibilities are more strongly coupled than autosomal ones (i.e.,  $\theta_X = \frac{3}{2}\theta_A$ ) in both regimes. This simply  
415 reflects the fact that equilibrium frequencies are nearly independent of the exact value of the coupling strength, when this  
416 is large, i.e., when selection acts on the block as a whole.

### 417 Number of barrier loci on the incompatible block:

418 The dependence of the equilibrium frequency of introgressing alleles on the number of barrier loci,  $L$ , is qualitatively  
419 different in different coupling regimes. In the strong coupling regime ( $\theta \gg 1$ ), the equilibrium frequency of sex-linked and  
420 autosomal alleles is largely governed by the net selective disadvantage of the block ( $S = sL$ ), and increases only weakly

421 with  $L$  for fixed  $S$  (red; Figure 1A and Figure S4A). For smaller  $\theta$ ,  $\bar{p}_A$  and  $\bar{p}_X$  both increase with  $L$  (black, Figure 1A);  
422 correspondingly, the barrier strengths,  $b_A$  and  $b_X$ , decrease (Figure S3). For very weak coupling ( $\theta \ll 1$ ), the equilibrium  
423 deleterious allele frequency increases so strongly with  $L$  that our predictions break down at large  $L$  (blue; Figure 1A).  
424 As before, this is because in the  $\theta \ll 1$  regime, any locus is only weakly affected by other selected loci and the effective  
425 selection coefficient per allele is close to the raw selective effect  $s$ . The latter decreases as we consider genetic architectures  
426 with larger numbers of more weakly selected loci (from left to right along the x axis in Figure 1).

427

428 We also predict that the ratio of autosomal and X-linked deleterious allele frequencies and barrier strengths should  
429 increase with the number of barrier loci involved ( $L$ ), for weak coupling between selected loci. In this regime, the  
430 effective strength of selection against individual loci scales as  $L^{2\theta_X}$  and  $L^{2\theta_A}$  for X-linked and autosomal deleterious alleles  
431 respectively; thus, the ratio of equilibrium frequencies scales as  $L^{2(\theta_A - \theta_X)}$  for  $\theta_A, \theta_X \ll 1$ . Since coupling between  
432 X-linked alleles is stronger, this means that the ratio of X-linked alleles to autosomal alleles must decrease as the barrier  
433 becomes more polygenic. For example, for  $\theta_A = 0.2$  and  $\theta_X = 0.3$ , introgression rates on the sex chromosome are  $\sim 0.63$   
434 times that of the autosomes when the incompatible block is polygenic ( $L = 100$ , blue; Figure 1A), while, as expected, they  
435 are identical for both chromosomes with a single barrier locus ( $L = 1$ ). The same qualitative pattern is observed for the  
436 ratio of barrier strengths:  $\frac{b_X}{b_A} \sim 2$  for  $L = 100$  while it is  $\sim 1.5$  for  $L = 1$ , when the neutral marker is close to the block  
437 ( $\alpha = 0.01$ , black; Figure 2). In contrast, under strong coupling (e.g.  $\theta_A = 10$  and  $\theta_X = 15$ ), the differences between the  
438 two types of chromosomes become negligible (red; Figure S4). Note that for the barrier strength, only the extreme cases  
439 of a single strongly deleterious locus ( $L = 1$ ) and many weakly deleterious loci ( $L = 100$ ) are considered hereafter.

#### 440 **Distance of the neutral marker from the incompatible block:**

441 We have modeled a situation in which a neutral marker lies at one end of an incompatible genomic block (“rod” configu-  
442 ration). The barrier strength always decreases as a function of the distance between the neutral marker and the closest  
443 selected locus on both autosomes and the sex chromosomes. This is because the ability of a neutral marker to escape  
444 from its incompatible genomic background increases with its map distance from the selected block. For example, under  
445 intermediate coupling (black; Figure 2), a multilocus autosomal barrier can reduce the effective migration rate by a factor  
446 of  $\sim 20$  for a neutral marker at a relative distance of  $\alpha = 0.01$  from the closest barrier locus, while this factor drops to  
447 less than  $\sim 2$  at a fraction distance  $\alpha = 1$ ; these numbers are respectively  $\sim 35$  and  $\sim 2.5$  in the sex chromosome case.

448

449 This pattern also holds in the more general case of a neutral marker embedded in the selected block (“embedded” con-  
450 figuration, Figure S7). In agreement with previous work on autosomes (Barton and Bengtsson 1986) and sex chromosomes  
451 (Muirhead and Presgraves 2016), the barrier at the center is up to one order of magnitude stronger than the barrier at the  
452 edge of the block, because two recombination events (instead of one) are required for the neutral marker to escape from  
453 the deleterious background. Moreover, as the predictions concerning sex-linked barriers can again be obtained from the  
454 corresponding autosomal predictions by a change in sex-averaged effective parameters, our general conclusion that these  
455 impede neutral flow more strongly than autosomal barriers still holds in the ‘embedded’ simulations. However, this effect

456 is more extreme than in the “rod” configuration (e.g.  $\frac{b_X}{b_A} \sim 3$  instead of  $\sim 2$  for  $L = 100$  and  $\alpha = 0.01$ ; Figure S7).

## 457 Dosage compensation and sex-biased forces

### 458 Dosage compensation:

459 The basic model considered so far assumes that the hemizygous selective effect of a deleterious allele in a XY male (or  
460 a ZW female) is half as strong as the homozygous effect in a XX female (or a ZZ male). However, various molecular  
461 mechanisms of dosage compensation have evolved independently in various clades in response to these imbalances (Gu  
462 and Walters 2017). These mechanisms may boil down to a fitness model where the effect of a single copy of a sex-linked  
463 allele in hemizygous males is similar to that of two copies in homozygous females ( $s_M = s_{hom,F} = 2s$  and  $s_F = s$  for  
464  $h = 0.5$ ). In such a situation, we have  $\theta_X = 2\theta_A$  and  $\frac{m_X}{S_X} = \frac{3}{4} \frac{m_A}{S_A}$  (Table S1), which results in a lower X-to-autosome  
465 ratio of introgressed allele frequencies for weak to intermediate coupling between selected loci ( $\frac{\bar{p}_X}{\bar{p}_A} \sim 0.4$  instead of  $\sim 0.66$   
466 in the basic model with  $L = 100$ , compare red vs. black; Figure 3A). This holds for single and multilocus barriers (i.e.,  
467 for  $L \geq 1$ ), since not only coupling between sex-linked alleles is stronger than in the basic model, but also  $m/S$  on the  
468 sex chromosomes is weaker than on autosomes. Likewise, the influence of dosage compensation on neutral gene flow is  
469 to increase the ratio of barrier strengths relative to the basic model ( $\frac{b_X}{b_A} \sim 3$  instead of  $\sim 2$  with  $L = 100$  and  $\alpha = 0.01$ ,  
470 compare red vs. black; Figure 4A).

### 471 Achiasmy:

472 In some species, such as certain dipterans or lepidopterans, recombination is absent throughout the genome in the het-  
473 erogametic sex (i.e. their meiosis is achiasmatic, so  $c_M = 0$  on autosomes and sex chromosomes in an XY sexual system;  
474 Satomura et al. 2019). As sex-linked introgressing alleles (whether on the X or Z) spend one third of their time in the  
475 heterogametic sex, while autosomal alleles spend an equal amount of time in both sexes (at least under weak selection),  
476 achiasmy leads to a lower effective recombination rate of autosomal compared to sex-linked incompatibility loci (all else  
477 being equal; Langley et al. 1988, Betancourt et al. 2004). In this case, we have  $\theta_X = \frac{3}{4}\theta_A$  (Table S1), i.e. the coupling  
478 between barrier loci is stronger on autosomes than on sex chromosomes. As a consequence, achiasmatic species exhibit  
479 higher introgression of sex chromosomes than autosomes when barriers are multilocus ( $\sim 1.2$  times higher with  $L = 100$ ,  
480 blue; Figure 3A), and their barrier strength is accordingly weaker ( $\sim 0.6$  that of autosomes with  $L = 100$  and  $\alpha = 0.01$ ,  
481 blue; Figure 4A).

### 482 Sex-biased migration:

483 Differences in migration rates between sexes are commonly observed in nature, with the heterogametic sex prone to higher  
484 migration in mammals and birds (Greenwood 1980; Trochet et al. 2016), although there are many exceptions and patterns  
485 are less known in other taxa. Sex-biased migration affects only the introgression of the sex-linked alleles (to lowest order  
486 in  $sL$  and  $cL$ ), because immigrants of the heterogametic and homogametic sex introduce different numbers of sex-linked  
487 blocks, but the same number of autosomal blocks. Under male-biased migration in a XY system (or female-biased in a ZW

488 system), the migration-to-selection ratio is thus lower for sex-linked alleles than for autosomal alleles (e.g. if  $m_M = 3m_F$ ,  
489 then  $\frac{m_X}{S_X} = \frac{5}{6} \frac{m_A}{S_A}$ ; Table S1). Compared to the basic model, this results in a lower X-to-autosome ratio for  $L \geq 1$  and  
490 weak to intermediate coupling ( $\frac{\bar{p}_X}{\bar{p}_A} \sim 0.55$  instead of  $\sim 0.66$  with  $L = 100$ , red; Figure 3B). Note that the barrier strength  
491 is predicted to be insensitive to sex-bias in migration to lowest order in  $sL$  and  $cL$  (see eqs. (5) and (6)). However, for  
492 stronger selection, sex-biased migration can have a very weak effect on barrier strength, which is predicted by eqs. (22)  
493 and (30) (Appendix B, and Figure 4B). Opposite patterns are found when migration is female-biased in a XY system (e.g.  
494 if  $m_F = 3m_M$ , then  $\frac{m_X}{S_X} = \frac{7}{6} \frac{m_A}{S_A}$ ; Table S1), or male-biased in a ZW system.

#### 495 Sex-biased selection:

496 In most species, the strength of sexual selection is stronger in males than in females (Singh and Punzalan 2018). Since  
497 twice as many females carry a single copy of any X-linked introgressed allele as males (i.e., any X-linked incompatible  
498 allele spends twice as much time in females as in males), while the reverse is true for the Z chromosome, male-biased  
499 sexual selection will lead to opposite effects in the two sexual systems. For example, if selection is twice as strong on XY  
500 males as on females ( $s_M = 2s_F$ ), we have  $\theta_X = \frac{4}{3}\theta_A$  and  $\frac{m_X}{S_X} = \frac{9}{8} \frac{m_A}{S_A}$  (Table S1), i.e. weaker coupling between barrier loci  
501 and a stronger migration-to-selection ratio on the sex chromosome compared to autosomes, than in the basic model. This  
502 results in a higher X-to-autosome equilibrium frequency compared to the basic model ( $\frac{\bar{p}_X}{\bar{p}_A} \sim 0.9$  instead of  $\sim 0.66$  with  
503  $L = 100$ , red; Figure 3C), and a correspondingly lower ratio of barrier strengths ( $\frac{b_X}{b_A} \sim 1.5$  instead of  $\sim 2$  with  $L = 100$   
504 and  $\alpha = 0.01$ , red; Figure 4C). On the contrary, when ZZ males are under stronger selection than females ( $s_F = 2s_M$ ,  
505 using our XY notation), we have  $\theta_Z = \frac{5}{3}\theta_A$  and  $\frac{m_Z}{Z} = \frac{9}{10} \frac{m_A}{S_A}$  (Table S1), resulting in lower Z-to-autosome equilibrium  
506 frequencies compared to the basic model (Figure 3C) and a correspondingly higher barrier strength (Figure 4C).

#### 507 Dominance of barrier loci

508 In Figures 1-4, we have shown results for individual-based simulations in which incompatible mutations have co-dominant  
509 effects ( $h = 0.5$ ), and compared these with our analytical predictions, which are actually independent of dominance as  
510 long as selection is weak, i.e.,  $sL$  and  $cL \ll 1$  (see Table S1 and Appendix A). We now examine how dominance affects  
511 introgression patterns by considering either partial recessivity ( $h = 0.1$ ) or full dominance ( $h = 1.0$ ) of the incompatible  
512 effects, and by varying their homozygous deleterious effect ( $s_{hom}$ ) while keeping the heterozygous selective effect ( $s$ ) con-  
513 stant. This allows us to test the influence of  $h = s/s_{hom}$  (i.e., the ratio of heterozygous to homozygous effects) for a given  
514 heterozygous effect.

515  
516 Recessivity of deleterious alleles implies that homozygous blocks are far more deleterious than in the co-dominant  
517 model ( $s_{hom} = 0.5$  for  $h = 0.1$  vs  $s_{hom} = 0.1$  for  $h = 0.5$ ), resulting in a strong selective disadvantage for first generation  
518 immigrants of the homogametic sex (in case of sex-linked blocks) or of both sexes (in case of the autosomal blocks). This  
519 leads to lower levels of introgression for both the sex chromosome and autosomes (blue; Figure S5B). Moreover, total  
520 migration is more strongly reduced in autosomes (due to the reduced fitness in both male and female migrants) relative  
521 to sex chromosomes (since only female migrants have substantially lower fitness than in the co-dominant model). Thus,

522 the X-to-autosome introgression ratio is higher than that in the co-dominant case (Figure S5B), and the ratio of barrier  
523 strengths is correspondingly smaller (Figure S6B). However, it is worth noting that introgression remains low overall in our  
524 model, and so homozygous blocks are rare in the population. They are actually neglected in the weak selection approxi-  
525 mation (lines) and only enter into the more general analytical predictions (filled symbols) via the fitness of homozygous  
526 migrants (of the homogametic sex); however, they are fully considered in the simulations (empty symbols). Therefore, the  
527 influence of dominance ( $h$ ) on equilibrium frequencies and barrier strengths is by itself weak: what matters to the first  
528 order is only the heterozygous effects ( $s$ ) of the incompatible alleles.

529

530 If we also assume dosage compensation, then the migration-to-selection ratio ( $\frac{m}{s}$ ) of X-linked recessive alleles is only  
531  $\frac{1}{4}$  that of the autosomes (compared to  $\frac{3}{4}$  when alleles are co-dominant, see Table S1), and coupling between incompatible  
532 alleles ( $\theta$ ) is 6 times stronger on the X chromosome relative to autosomes (compared to 2 times when alleles are co-  
533 dominant, see Table S1). Importantly, these two effects cause a much lower X-to-autosome introgression ratio than under  
534 co-dominance ( $\frac{\bar{p}_X}{\bar{p}_A} \sim 0.1$  instead of  $\sim 0.4$  with  $L = 100$ , blue; Figure S5C), and also a much greater X-to-autosome ratio  
535 of barrier strengths ( $\frac{b_X}{b_A} \sim 20$  instead of  $\sim 3.5$  with  $L = 100$  and  $\alpha = 0.01$ , blue; Figure S6C). This point in the parameter  
536 space is important to consider as it corresponds to very different X-linked effects in hemizygous males and heterozygous  
537 females, which is the basis of the dominance theory (Turelli and Orr 1995, 2000).

538

539 Under complete dominance of incompatible alleles, homozygous blocks are just as deleterious as heterozygous blocks  
540 ( $s_{hom} = 0.05$  for  $h = 1.0$ ). This leads to slightly higher levels of introgression for both chromosome types in the  
541 simulations (red; Figure S5B), compared to the co-dominant model. With dosage compensation, the relationship between  
542 the composite parameters is  $\theta_X = \frac{3}{2}\theta_A$  and  $\frac{m_X}{s_X} = \frac{m_A}{s_A}$  (Table S1); therefore dominant X-linked alleles introgress more  
543 than co-dominant alleles (red; Figure S5C), and act as weaker barrier to gene flow (red; Figure S6C).

## 544 DISCUSSION

545 Previous work has considered the effect of divergence at large numbers of autosomal loci on genetic exchange between  
546 hybridizing species (Barton 1983; Barton and Bengtsson 1986; Baird 1995). Here, we have extended the analysis to sex  
547 chromosomes (X or Z). Under weak selection, predictions for the introgression of multilocus deleterious blocks (eq. 4), and  
548 their strength as barriers to neutral gene flow (eq. 6), are governed by the same equations as for autosomes, (eqs. 12 and  
549 5), but with different sex-averaged effective parameters  $m_X$ ,  $s_X$  and  $c_X$ . We show that all sex-linked effective parameters  
550 have the form  $a_X = \frac{2a_F + a_M}{3}$ , i.e., they are weighted sums of female and male components,  $a_F$  and  $a_M$ . This implies that  
551 evolutionary forces that affect males and females differently can be readily encapsulated by these effective parameters,  
552 and their biological consequences predicted. An extreme example is that of the X-linked recombination rate,  $c_X$ , which is  
553  $\frac{2c_F}{3}$ , since males do not contribute at all to recombination on the X.

554

555 These expressions for sex-linked effective parameters are identical to those proposed by Hedrick (2007) (see also Haldane

1926; Avery 1984), based on the argument that females carry two copies of the X chromosome (versus a single copy in males) and thus are associated with a weight that is twice that of males. However, the reason underlying the 2:1 female and male contributions to  $s_X$  and  $c_X$  in our model is somewhat different, since both males and females, with the exception of first-generation female immigrants, typically carry a single copy of any X-linked introgressed allele (assuming low introgression). As we demonstrate above, X-linked alleles nevertheless spend twice as much time in females as in males because twice as many females are heterozygous for X-linked deleterious alleles as males (are hemizygous). However, very strong selection against hybrids elevates the ratio  $P_M(y)/P_F(y)$  above 1/2 (see equation 15b, Appendix A), causing this simple description in terms of sex-averaged X-linked parameters to break down: analytical predictions can still be derived by separately considering genotypic frequencies in males and females.

### Conditions for an oversized role of sex chromosomes in speciation

We find that the stronger coupling of incompatible alleles on the sex chromosome reduces the escape probability of neutral markers, and thus enhances their barrier strength by a factor of  $\sim 1.5$  to  $\sim 3$  relative to autosomes under optimal conditions (i.e. when the neutral marker is close to a multilocus barrier). This result mirrors earlier predictions for single and two-locus barriers under a regime where introgressed alleles are rare (Muirhead and Presgraves 2016), and for the maintenance of DMIs in parapatry (Hoellinger and Hermisson 2017). Although it is a moderate effect, this process is very general as it applies to species diverging in parapatry under various scenarios (including dosage compensation, sex-biased migration and sex-biased selection; see below), and regardless of the position of the neutral marker along the chromosome. However it may not be sufficient to explain the major role of sex chromosomes in the speciation of achiasmatic species such as *Drosophila*, where an opposite effect is expected (i.e. X-linked barriers are weaker than autosomal ones) if achiasmy is the sole sex-biased process acting.

The greatest sex chromosome-to-autosome ratio for the barrier strengths ( $\frac{b_X}{b_A} \sim 20$ ) occurs when incompatible alleles are recessive and dosage compensated (Figure S6C), in agreement with the model of Muirhead and Presgraves (2016). In contrast, Hoellinger and Hermisson (2017) did not find a large difference in the outcomes between co-dominant and recessive DMIs. We note that in their model the level of dominance is not associated with the single-locus effects but with the two-locus DMI itself, and so the fitness setting is unlike ours. Importantly, under our model assumptions, dominance (i.e. the ratio of heterozygous to homozygous fitness) itself has little effect as it affects only the selective disadvantage of migrants in the first generation (and of rare homozygous individuals). In essence, recessivity and dosage compensation cause hemizygous deleterious effects of X-linked alleles in males to be much stronger than the corresponding heterozygous effects in females. This is in line with the dominance theory (Turelli and Orr 1995, 2000), which proposes that if most incompatible alleles act recessively in hybrids, then their deleterious effects would be more exposed on the sex chromosomes (as they are hemizygous in males). Therefore, selection more efficiently removes recessive incompatible alleles from the sex chromosome, and eventually imposes a stronger barrier to neutral flow.

A new prediction that emerges from our multilocus model is that polygenic barriers comprised of many weakly coupled

591 alleles result in a greater sex chromosome-to-autosome difference than strong single-locus barriers (although the absolute  
592 strength of the barrier is stronger on the latter case). Therefore, the genetic architecture of reproductive isolation affects the  
593 extent of introgression on sex chromosomes relative to autosomes, which may have important implications. QTL mapping  
594 studies for sterility and/or inviability of hybrids typically detect a few large-effect loci (Maheshwari and Barbash 2011),  
595 but numerous smaller-effect incompatibilities may be commonly underestimated in these studies. Detailed backcrossing  
596 experiments have actually shown that each introgressed block might contain multiple linked barrier loci each required for  
597 reproductive isolation to appear (for a review see Fraïsse et al. 2014). Such polygenic barriers between species, if common,  
598 may contribute to the oversized role of sex chromosomes in speciation.

## 599 **The influence of dosage compensation**

600 Dosage compensation mechanisms are diverse among taxa (Gu and Walters 2017). In dipterans and hemipterans, imbal-  
601 ance of the sex-linked gene dose is solved by an up-regulation of the sex chromosome in the heterogametic sex, while in  
602 nematodes and mammals, there is a down-regulation of the sex chromosome in the homogametic sex in response to an early  
603 over-expression in both sexes (Charlesworth 1978). These evolutionary mechanisms are equivalent in terms of fitness effects  
604 as long as they lead to similar total levels of activity of the X (or Z) in males and females. We have shown that dosage  
605 compensation (e.g., the X expression is doubled in XY males) reinforces the barrier strength ratio of sex chromosomes to  
606 autosomes compared to the basic setting (Figure 4A), consistent with findings of Hoellinger and Hermisson (2017). This is  
607 because a single X-linked incompatible allele in males is selected against twice as strongly as a single X-linked allele in (het-  
608 erozygous) females, while selection against hemizygous males and heterozygous females is equally strong in the basic model.

609  
610 However, various complications exist. First, there is a controversy over whether there has indeed first been up-  
611 regulation of the X (or Z) in both sexes in response to degeneration of the Y (or W), followed by down-regulation in  
612 the homogametic sex (e.g. see the work by Mahajan and Bachtrog 2015 in *Tribolium*). Second, there are indications  
613 that dosage compensation is partial in some clades like in birds (Graves 2014). This means that the average expression  
614 of Z-linked dosage-sensitive genes in females is typically below that of the two copies in males. Third, the random  
615 inactivation of one of the two X chromosomes in mammalian females makes the situation even more complicated, as  
616 females are functionally hemizygous, but at the cell level (except in marsupials where paternal X inactivation makes  
617 females phenotypically hemizygous at the individual level, like males; Watson and Demuth 2012). Therefore, the effect of  
618 a heterozygous mutation on fitness depends on whether gene expression is cell autonomous (likely causing semi-dominance  
619 for fitness, Mank et al. 2010) or not. Finally, although our dosage compensation model assumptions are in line with  
620 precedents in the theoretical literature (e.g. Avery 1984), to the best of our knowledge, there is no direct proof that  
621 supports these.

## 622 **The influence of sex-biased forces**

623 Evolutionary parameters governing introgression and barrier strength (i.e.,  $m$ ,  $s$  and  $c$ ) may differ significantly between the  
624 sexes (Hedrick 2007). One general trend is that migration is biased towards the heterogametic sex (males in mammals and

625 females in birds; Greenwood 1980; Trochet et al. 2016). As the heterogametic sex carries only one copy of the X (or Z) vs.  
626 two for each autosome, this type of sex-bias leads to a deficit of the number of migrating X (or Z) relative to autosomes.  
627 Accordingly, we observed that both male-biased migration (in XY systems) and female-biased migration (in ZW systems)  
628 reduce the deleterious equilibrium frequency in the recipient species (Figure 3B, and see Hoellinger and Hermisson 2017  
629 for a similar effect on stability conditions of DMIs in parapatry); thus we do not expect systematic differences between the  
630 two sexual systems. Moreover, in the weak selection limit ( $sL \ll 1$ ), the barrier strength is predicted to be independent of  
631 sex-biased migration (see eqs. (5) and (6)); even under stronger selection, the ratio  $m_F/m_M$  has only an extremely weak  
632 effect on  $b$  (see Figure 4B; eqs. (22) and (30)).

633

634 Second, quantitative differences in recombination rate between sexes are common (heterochiasmy; Lenormand and  
635 Dutheil 2005). In some cases, recombination is totally absent in the heterogametic sex (Satomura et al. 2019), causing  
636 the effects of linkage to be stronger on autosomes relative to the sex chromosome. As sex-linked alleles spend less time  
637 in the heterogametic sex ( $\sim \frac{1}{3}$  of their time in XY males or in ZW females) than autosomal alleles ( $\frac{1}{2}$  of their time),  
638 achiasmy enhances the opportunity for neutral alleles to escape from the sex-linked incompatible loci. As a consequence,  
639 incompatibilities on the sex chromosomes become weaker interspecies barriers than autosomes (Figure 4A, qualitatively  
640 consistent with the observations of Muirhead and Presgraves 2016). Again, this effect is small and should not produce  
641 systematic differences between the X and the Z chromosomes, but is still important to consider in speciation genetics as  
642 many detailed studies have been done in *Drosophila*, where achiasmy is widespread.

643

644 Third, sexual selection tends to be stronger in males relative to females (Mallet et al. 2011; Sharp and Agrawal 2013;  
645 Singh and Punzalan 2018). If sexual selection contributes to reproductive isolation between incipient species, then the  
646 impact of male-biased sexual selection should vary between XY systems (where males are heterogametic and an X-linked  
647 allele spends  $\frac{1}{3}$  of its time in males) and ZW systems (where males are homogametic and a Z-linked allele spends  $\frac{2}{3}$  of  
648 its time in males). In agreement with this prediction, and assuming a two-fold selection bias toward males, we show that  
649 the barrier strength of the Z relative to autosomes is a little increased compared to the basic model, while for the X it is  
650 correspondingly decreased (Figure 4C).

## 651 **Insights from empirical data**

652 Next-generation data from a diversity of organisms has provided methods to scan genomes for interspecies reproductive  
653 barriers, and contrast autosomes with sex chromosomes. Provided that speciation takes place in the presence of gene flow,  
654 we expect incompatibilities and their surrounding loci to exhibit a lower rate of interspecific introgression than the rest of  
655 the genome (Roux et al. 2013; Sethuraman et al. 2019). In contrast, if speciation occurs in allopatry, it becomes impossible  
656 to locate barrier loci, because interspecies divergence is then a simple function of the time elapsed since isolation, which  
657 is shared by the whole genome (Wilkinson-Herbots 2008). By putting together population genomic studies of speciation,  
658 Presgraves (2018)'s meta-analysis provides useful insights into the role of sex chromosomes (X or Z) in speciation. He  
659 shows that sex chromosomes have a systematically higher differentiation level relative to autosomes in 101 species pairs



660 for which  $F_{ST}$  was reported. However, as acknowledged by the author, the impacts on genomic divergence of confounding  
661 factors occurring within species (like demographic events or linked selection) are hard to tell apart from the effect of  
662 selection against migrants between species, especially when summary statistics are applied.

663

664 To address this issue, we report in Table 3 empirical studies that statistically evaluate speciation models from genomic  
665 data (at the two extremes: a model of allopatric speciation vs a model where speciation is opposed by continuous gene  
666 flow; Sousa and Hey 2013), and then test whether sex chromosomes are more resistant to interspecies introgression than  
667 autosomes by estimating the introgression rate for each type of chromosome. Since cline analyses also provide unambiguous  
668 estimates of barrier strength (Barton and Hewitt 1985), and simulation studies have shown that incompatible sex-linked  
669 alleles flow more slowly than autosomal alleles across a cline (Wang 2013; Hvala et al. 2018; Sciuchetti et al. 2018), we also  
670 include them in Table 3, together with studies inferring introgressed tracts from phased genomes (Lawson et al. 2012).  
671 We investigate a wide range of species (34 species pairs, including 16 XY systems and 18 ZW systems) characterized by  
672 diverse mechanisms of dosage compensation and sex-biased forces.

673 Overall, patterns are mixed, with  $\sim 50\%$  of the studies showing lower effective migration rates, steeper clines and/or  
674 lower fraction of introgressed tracts on the sex chromosomes compared to the autosomes ( $\frac{6}{13}$  in XY studies and  $\frac{9}{17}$  in  
675 ZW studies). This apparently inconclusive result hides a strong heterogeneity between clades. In XY systems, almost all  
676 mammalian species exhibit stronger X-linked barriers relative to autosomes, in agreement with our theoretical predictions.  
677 Dosage compensation by X chromosome inactivation in mammals might contribute to this effect, while the prevalence  
678 of male-biased sexual selection would act in the opposite direction. On the contrary, none of the studies in *Drosophila*  
679 (except one) shows that the X chromosome is less exchangeable between species than autosomes. This is in line with the  
680 fact that the species studied are achiasmatic, although this effect might be counterbalanced by the widespread mechanism  
681 of dosage compensation in *Drosophila* (X up-regulation in males). In ZW systems, birds present a balanced mixture of  
682 the two patterns across the six orders examined, which prevents us from drawing any definite conclusions. On the one  
683 side, the prevalence of male-biased sexual selection in birds is expected to boost the strength of the Z-linked barriers.  
684 On the other side, birds may only have partial dosage compensation of Z-linked genes, which would counteract this  
685 effect. Dosage compensation is highly debated in birds, and more generally, it is unclear how the diverse dosage compensa-  
686 tion mechanisms alter the fitness effects of incompatibilities; therefore our interpretations should be regarded as suggestive.

687

688 These patterns are preliminary, and should be taken with caution for several reasons. A first caveat is the heterogeneity  
689 across studies in both their genetic markers (number and type) and statistical methods. A second concern is the taxonomic  
690 sampling, which is overweighted by some clades (e.g. bird species) or some genera within clades (e.g. *Drosophila*), and  
691 likely influenced by positive results; altogether these factors may introduce systematic biases in the global picture. Third,  
692 information concerning the mechanisms of dosage compensation or the existence of sex bias is generally reported for a  
693 handful of species and then generalized at the level of the order; this precludes a definite test of their impact. Moreover,  
694 the balance of power among sex-biased evolutionary forces will depend on their respective magnitudes, which is very hard  
695 to measure in nature. Fourth, the effect of sex chromosome linkage, dosage compensation and sex bias on the capacity

696 of sex chromosomes to better resist interspecies introgression may be too subtle to be observed in real data. Actually,  
697 the main determinant of the barrier strength between species remains the genetic architecture of reproductive isolation  
698 (i.e., the number of barrier loci and their level of recessivity and/or epistasis). Whether or not this differs between sex  
699 chromosomes and autosomes remains an open question. Finally, our predictions (which rely on the crucial assumption  
700 of rare introgression) may not fully apply to these empirical studies where the frequency of introgressing alleles must be  
701 sufficiently high to be detected. Table 3 definitely calls for further comparative analyses that hopefully would provide  
702 more robust patterns and a better understanding of their cause.

## 703 **Limitations of the model**

704 The present model investigates whether the chromosomal location of reproductive barriers (i.e., sex chromosomes vs.  
705 autosomes) influences their capacity to impede interspecies genetic exchange, and ultimately, promote or undermine  
706 speciation. However, the rate of accumulation of barrier loci during species divergence may also vary with chromosomal  
707 location: the “faster-X theory” (Charlesworth et al. 1987) predicts that sex chromosomes (X or Z) evolve more rapidly than  
708 autosomes, and as a result, harbor more interspecific barriers. Here, we cannot shed light on the faster-X effect, because  
709 the number of reproductive barriers is a parameter of the model; we do not actually model the processes that cause them  
710 to accumulate in the first place. Extending our work to model an initial divergence phase would help understand to what  
711 extent the faster-X effect contributes to the role of sex chromosomes in speciation.

712 Our model of genetic exchange in a mainland-island setting rests on the assumption that the rate of migration is low  
713 relative to selection, so that hybrids remain rare. However, to account for the clinal structure of hybrid zones (Barton  
714 and Hewitt 1985) and the intermediate frequency of incompatible alleles at their center, it will be necessary to extend our  
715 model to more realistic spatial geometries, such as a stepping-stone model or a continuous habitat. This requires different  
716 theoretical frameworks that track genomes with multiple introgressed blocks (see Barton 1983, and Barton and Bengtsson  
717 1986), which is beyond the scope of this study. Moreover, in this situation, dominance of the incompatible alleles will  
718 become important; thus deviations from the simple expectation that introgression in the sex-linked case is the same as  
719 introgression in the autosomal case with appropriately rescaled parameters could arise.

720

721 Our analytical treatment is deterministic, i.e., assumes that populations are large enough that genetic drift can be  
722 neglected, which is, of course, unrealistic in most natural populations. Also, the outcomes of a hybridization event (where  
723 one or a few foreign genomes are introduced and randomly split by recombination) are stochastic (Baird et al. 2003). An  
724 extension of the model to include these stochastic processes will thus be necessary to understand the short-term evolution  
725 of hybrid genomes.

726

727 Another assumption is that all selected alleles are exclusively, and equally, deleterious in the recipient species. A  
728 more realistic model would consider allelic effects that vary along the chromosome (i.e., from globally adaptive to locally  
729 deleterious). Sachdeva and Barton (2018) studied this scenario by analyzing the introgression dynamics of an autosomal  
730 block with linked adaptive and deleterious variants with infinitesimal effects. Importantly, they demonstrated that in

731 this case, deleterious variants can attain high frequency in the recipient species via hitchhiking with genomic blocks with  
732 net positive effect (see also Bisschop et al. 2019 for a simulation study). Thus it would be informative to extend the  
733 present model to examine how hitchhiking with globally adaptive variants influences the relative barrier strengths of sex  
734 chromosomes vs autosomes.

735

736 Finally, we would also need to take into account the genome-wide reduction in the effective neutral gene flow due  
737 to loosely linked loci (which should affect different chromosomes to similar extents), and would thus further reduce the  
738 difference between sex chromosomes and autosomes in species with large genomes.

## 739 DATA AVAILABILITY STATEMENT

740 SLiM simulation codes for the basic model and notebooks for the maths analyses are available as **Supplementary Codes:**

741 SuppCode-1a: SLiM code for autosomal equilibrium frequencies

742 SuppCode-1b: SLiM code for X-linked equilibrium frequencies

743 SuppCode-1c: SLiM code for autosomal barrier strengths (rod configuration)

744 SuppCode-1d: SLiM code for X-linked barrier strengths (rod configuration)

745 SuppCode-1e: SLiM code for autosomal barrier strengths (embedded configuration)

746 SuppCode-1f: SLiM code for X-linked barrier strengths (embedded configuration)

747 SuppCode-2a: Mathematica notebook for equilibrium frequencies

748 SuppCode-2b: Mathematica notebook for barrier strengths

749

### 750 **Supplementary Texts:**

751 1. Appendix A: Distribution of lengths of introgressing blocks

752 2. Appendix B: Strength of a multilocus barrier to the flow at a neutral marker

753 3. Model extensions: Epistasis among barrier loci

754 4. Detailed methods: Individual-based simulations

755

### 756 **Supplementary Figures:**

757 Figure S1. Effective number of loci

758 Figure S2. Correspondence between autosomal and X-linked frequencies

759 Figure S3. Effect of the number of barrier loci on average equilibrium frequencies and barrier strength at the neutral  
760 marker

761 Figure S4. Effect of very strong coupling on average equilibrium frequencies and barrier strength at the neutral marker

762 Figure S5. Effect of model extensions on average equilibrium frequencies

763 Figure S6. Effect of model extensions on barrier strength at the neutral marker

764 Figure S7. Effect of the “rod” vs “embedded” configurations on barrier strength at the neutral marker

765

766 **Supplementary Tables:**

767 Table S1. Predictions for different evolutionary scenarios

768 **AUTHOR CONTRIBUTIONS**

769 C.F. designed the project, C.F. and H. S. derived the analytical and numerical results, C.F. conducted the simulations.  
770 C.F. and H.S. wrote the manuscript.

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777 **REFERENCES**

- 778 Avery, P. J., 1984 The population genetics of haplo-diploids and X-linked genes. *Genetics Res.* 44: 321–341.  
779 <https://doi.org/10.1017/S0016672300026550>
- 780 Bachtrog, D., J. E. Mank, C. L. Peichel, M. Kirkpatrick, S. P. Otto, *et al.*, 2014 Sex determination: why so many ways  
781 of doing it? *PLoS Biol.* 12: e1001899. <https://doi.org/10.1371/journal.pbio.1001899>
- 782 Bachtrog, D., S. Mahajan, and R. Bracewell, 2019 Massive gene amplification on a recently formed *Drosophila* Y chromo-  
783 some. *Nat. Ecol. Evol.* 3: 1587–1597. <https://doi.org/10.1038/s41559-019-1009-9>
- 784 Backström, N., and Ü. Väli, 2011 Sex- and species- biased gene flow in a spotted eagle hybrid zone. *BMC Evol. Biol.* 11:  
785 100. <https://doi.org/10.1186/1471-2148-11-100>
- 786 Baird, S., 1995 A simulation study of multilocus clines. *Evolution* 49: 1038–1045. <https://doi.org/10.1111/j.1558-5646.1995.tb04431.x>
- 788 Baird, S., N. Barton, and A. Etheridge, 2003 The distribution of surviving blocks of an ancestral genome. *Theor.*  
789 *Popul. Biol.* 64: 451–471. [https://doi.org/10.1016/S0040-5809\(03\)00098-4](https://doi.org/10.1016/S0040-5809(03)00098-4)
- 790 Baiz, M. D., P. K. Tucker, and L. Cortés-Ortiz, 2019 Multiple forms of selection shape reproductive isolation in a  
791 primate hybrid zone. *Mol. Ecol.* 28: 1056–1069. <https://doi.org/10.1111/mec.14966>
- 792 Barton, N., 1983 Multilocus clines. *Evolution* 37: 454–471.

- 793 Barton, N., 1995 Linkage and the limits to natural selection. *Genetics* 140: 821–841.
- 794 Barton, N., and B. O. Bengtsson, 1986 The barrier to genetic exchange between hybridising populations. *Heredity* 57:  
795 357–376. <https://doi.org/10.1038/hdy.1986.135>
- 796 Barton, N., and G. M. Hewitt, 1985 Analysis of hybrid zones. *Annu. Rev. Ecol. Evol. Syst* 16: 113–148.
- 797 Bengtsson, B. O., 1985 The flow of genes through a genetic barrier, pp. 31–42 in *Evolution essays in honour of John*  
798 *Maynard Smith*, edited by J. J. Greenwood, P. H. Harvey and M. Slatkin. Cambridge University Press,  
799 Cambridge.
- 800 Betancourt, A. J., Y. Kim, and H. A. Orr, 2004 A pseudohitchhiking model of X vs. autosomal diversity. *Genetics* 168:  
801 2261–2269. <https://doi.org/10.1534/genetics.104.030999>
- 802 Bisschop, G., D. Setter, M. Rafajlovic, S. J. Baird, and K. Lohse, 2020 The impact of global selection on local adaptation  
803 and reproductive isolation. *Phil. Trans. R. Soc. B* 375: 20190531. <http://doi.org/10.1098/rstb.2019.0531>
- 804 Bourgeois, Y. X., J. A. Bertrand, B. Delahaie, H. Holota, C. Thébaud, and B. Milá, 2018 Local adaptation and sexual  
805 selection drive intra-island diversification in a songbird lineage: differential divergence in autosomes and sex  
806 chromosomes. *bioRxiv* 353771. (Preprint posted June 22, 2018). <https://doi.org/10.1101/353771>
- 807 Carling, M. D., I. J. Lovette, and R. T. Brumfield, 2010 Historical divergence and gene flow: coalescent analyses of mito-  
808 chondrial, autosomal and sex-linked loci in *Passerina* buntings. *Evolution* 64: 1762–1772. <https://doi.org/10.1111/j.1558-5646.2010.00954.x>  
809 5646.2010.00954.x
- 810 Carneiro, M., J. A. Blanco-Aguilar, R. Villafuerte, N. Ferrand, and M. W. Nachman, 2010 Speciation in the European  
811 rabbit (*Oryctolagus cuniculus*): islands of differentiation on the X chromosome and autosomes. *Evolution* 64:  
812 3443–3460. <https://doi.org/10.1111/j.1558-5646.2010.01092.x>
- 813 Cattani, M. V. and D. C. Presgraves, 2012 Incompatibility between X chromosome factor and pericentric heterochro-  
814 matic region causes lethality in hybrids between *Drosophila melanogaster* and its sibling species. *Genetics* 191:  
815 549–559. <https://doi.org/10.1534/genetics.112.139683>
- 816 Charlesworth, B., 1978 Model for evolution of Y chromosomes and dosage compensation. *Proc. Natl. Acad. Sci. U.S.A.*  
817 75: 5618–5622. <https://doi.org/10.1073/pnas.75.11.5618>
- 818 Charlesworth, B., 1998 Measures of divergence between populations and the effect of forces that reduce variability. *Mol.*  
819 *Biol. Evol.* 15: 538–543. <https://doi.org/10.1093/oxfordjournals.molbev.a025953>
- 820 Charlesworth, B., J. L. Campos, and B. C. Jackson, 2018 Faster-X evolution: Theory and evidence from *Drosophila*. *Mol.*  
821 *Ecol.* 27: 3753–3771. <https://doi.org/10.1111/mec.14534>
- 822 Charlesworth, B., J. A. Coyne, and N. H. Barton, 1987 The relative rates of evolution of sex chromosomes and autosomes.  
823 *Am. Nat.* 130: 113–146. <https://doi.org/10.1086/284701>

- 824 Chu, J.-H., D. Wegmann, C.-F. Yeh, R.-C. Lin, X.-J. Yang, *et al.*, 2013 Inferring the geographic mode of speciation by  
825 contrasting autosomal and sex-linked genetic diversity. *Mol. Biol. Evol.* 30: 2519–2530. <https://doi.org/10.1093/molbev>
- 826 Coyne, J., and A. H. Orr, 1989 Two rules of speciation, pp. 180–207 in *Speciation and its consequences*, edited by D.  
827 Otte and J. Endler. Sinauer Associates, Sunderland, MA.
- 828 Cruickshank, T. E., and M. W. Hahn, 2014 Reanalysis suggests that genomic islands of speciation are due to reduced  
829 diversity, not reduced gene flow. *Mol. Ecol.* 23: 3133–3157. <https://doi.org/10.1111/mec.12796>
- 830 Dalquen, D. A., T. Zhu, and Z. Yang, 2017 Maximum likelihood implementation of an Isolation-with-Migration model  
831 for three species. *Syst. Biol.* 66: 379–398. <https://doi.org/10.1093/sysbio/syw063>
- 832 Dhimi, K. K., L. Joseph, D. A. Roshier, and J. L. Peters, 2016 Recent speciation and elevated Z-chromosome dif-  
833 ferentiation between sexually monochromatic and dichromatic species of Australian teals. *J. Avian Biol.* 47:  
834 92–102. <https://doi.org/10.1111/jav.00693>
- 835 Dobzhansky, T. H., 1936 Studies on hybrid sterility. II. Localization of sterility factors in *Drosophila pseudoobscura*  
836 hybrids. *Genetics* 21: 113.
- 837 Dobzhansky, T. H., 1937 *Genetics and the origin of species*. Columbia University Press, New York City.
- 838 Fraïsse, C., J. Elderfield, and J. Welch, 2014 The genetics of speciation: are complex incompatibilities easier to evolve?  
839 *J. Evol. Biol.* 27: 688–699. <https://doi.org/10.1111/jeb.12339>
- 840 Frank, S. A., 1991 Divergence of meiotic drive-suppression systems as an explanation for sex-biased hybrid sterility  
841 and inviability. *Evolution* 45: 262–267. <https://doi.org/10.1111/j.1558-5646.1991.tb04401.x>
- 842 Fuller, Z. L., C. J. Leonard, R. E. Young, S. W. Schaeer, and N. Phadnis, 2018 Ancestral polymorphisms explain the  
843 role of chromosomal inversions in speciation. *PLoS Genet.* 14: e1007526. <https://doi.org/10.1371/journal.pgen.1007526>
- 844 Galla, S. J., and J. A. Johnson, 2015 Differential introgression and effective size of marker type influence phylogenetic  
845 inference of a recently divergent avian group (Phasianidae: *Tympanuchus*). *Mol. Phylogenetics Evol.* 84:  
846 1–13. <https://doi.org/10.1016/j.ympev.2014.12.012>
- 847 Geneva, A. J., C. A. Muirhead, S. B. Kingan, and D. Garrigan, 2015 A new method to scan genomes for introgression  
848 in a secondary contact model. *PLoS One* 10: e0118621. <https://doi.org/10.1371/journal.pone.0118621>
- 849 Ginsberg, P. S., D. P. Humphreys, and K. A. Dyer, 2019 Ongoing hybridization obscures phylogenetic relationships in  
850 the *Drosophila subquinaria* species complex. *J. Evol. Biol.* 32: 1093–1105. <https://doi.org/10.1111/jeb.13512>
- 851 Graves, J. A. M., 2014 Avian sex, sex chromosomes, and dosage compensation in the age of genomics. *Chromosome*  
852 *Res.* 22: 45–57. <https://doi.org/10.1007/s10577-014-9409-9>
- 853 Greenwood, P. J., 1980 Mating systems, philopatry and dispersal in birds and mammals. *Anim. Behav.* 28: 1140–1162.  
854 [https://doi.org/10.1016/S0003-3472\(80\)80103-5](https://doi.org/10.1016/S0003-3472(80)80103-5)

- 855 Gu, L., and J. R. Walters, 2017 Evolution of sex chromosome dosage compensation in animals: a beautiful theory,  
856 undermined by facts and bedeviled by details. *Genome Biol. Evol.* 9: 2461–2476. <https://doi.org/10.1093/gbe/evx154>
- 857 Haldane, J. B. S., 1922 Sex ratio and unisexual sterility in hybrid animals. *J. Genet.* 12: 101–109. <https://doi.org/10.1007/BF0298>
- 858 Haldane, J. B. S., 1926 A mathematical theory of natural and artificial selection. Part III. *Proc. Camb. Philos. Soc.* 23:  
859 363–372. <https://doi.org/10.1017/S0305004100015176>
- 860 Hedrick, P. W., 2007 Sex: differences in mutation, re-  
861 combination, selection, gene flow, and genetic drift. *Evolution* 61: 2750–2771. <https://doi.org/10.1111/j.1558-5646.2007.00250.x>
- 862 Herrig, D. K., A. J. Modrick, E. Brud, and A. Llopart, 2014 Introgression in the *Drosophila subobscura* - *D. madeirensis*  
863 sister species: evidence of gene flow in nuclear genes despite mitochondrial differentiation. *Evolution* 68:  
864 705–719. <https://doi.org/10.1111/evo.12295>
- 865 Hoellinger, I., and J. Hermisson, 2017 Bounds to parapatric speciation: a Dobzhansky-Muller incompatibility model in-  
866 volving autosomes, X chromosomes, and mitochondria. *Evolution* 71: 1366–1380. <https://doi.org/10.1111/evo.13223>
- 867 Hogner, S., S. A. Sæther, T. Borge, T. Bruvik, A. Johnsen, and G.-P. Sætre, 2012 Increased divergence but reduced  
868 variation on the Z chromosome relative to autosomes in *Ficedula* flycatchers: differential introgression or the  
869 faster-Z effect? *Ecol. Evol.* 2: 379–396. <https://doi.org/10.1002/ece3.92>
- 870 Hooper, D. M., S. C. Griffith, and T. D. Price, 2019 Sex chromosome inversions enforce reproductive isolation across  
871 an avian hybrid zone. *Mol. Ecol.* 28: 1246–1262. <https://doi.org/10.1111/mec.14874>
- 872 Hurst, L. D., and A. Pomiankowski, 1991 Causes of sex ratio bias may account for unisexual sterility in hybrids: a  
873 new explanation of Haldane’s rule and related phenomena. *Genetics* 128: 841–858.
- 874 Hvala, J. A., M. E. Frayer, and B. A. Payseur, 2018 Signatures of hybridization and speciation in genomic patterns  
875 of ancestry. *Evolution* 72: 1540–1552. <https://doi.org/10.1111/evo.13509>
- 876 Kapopoulou, A., M. Kapun, P. Pavlidis, B. Pieper, R. Wilches, *et al.*, 2018 Early split between African and European popu-  
877 lations of *Drosophila melanogaster*. bioRxiv 340422. (Preprint posted June 06, 2018). <https://doi.org/10.1101/340422>
- 878 Knief, U., C. M. Bossu, N. Saino, B. Hansson, J. Poelstra, *et al.*, 2019 Epistatic mutations under divergent selection  
879 govern phenotypic variation in the crow hybrid zone. *Nat. Ecol. Evol.* 3: 570–576. <https://doi.org/10.1038/s41559-019-0847-9>
- 880
- 881 Langley, C. H., E. Montgomery, R. Hudson, N. Kaplan, and B. Charlesworth, 1988 On the role of unequal ex-  
882 change in the containment of transposable element copy number. *Genet. Res. (Camb.)* 52: 223–235.  
883 <https://doi.org/10.1017/S0016672300027695>
- 884 Larson, E. L., T. A. White, C. L. Ross, and R. G. Harrison, 2014 Gene flow and the maintenance of species boundaries.  
885 *Mol. Ecol.* 23: 1668–1678. <https://doi.org/10.1111/mec.12601>

- 886 Lawson, D. J., G. Hellenthal, S. Myers, and D. Falush, 2012 Inference of population structure using dense haplotype  
887 data. *PLoS Genet.* 8: e1002453. <https://doi.org/10.1371/journal.pgen.1002453>
- 888 Lenormand, T., and J. Dutheil, 2005 Recombination difference between sexes: a role for haploid selection. *PLoS Biol.* 3:  
889 e63. <https://doi.org/10.1371/journal.pbio.0030063>
- 890 Lindtke, D., and C. A. Buerkle, 2015 The genetic architecture of hybrid incompatibilities and their effect on barriers to  
891 introgression in secondary contact. *Evolution* 69: 1987–2004. <https://doi.org/10.1111/evo.12725>
- 892 Liu, K. J., E. Steinberg, A. Yozzo, Y. Song, M. H. Kohn, and L. Nakhleh, 2015 Interspecific introgressive origin of ge-  
893 nomic diversity in the house mouse. *Proc. Natl. Acad. Sci. U.S.A.* 112: 196–201. <https://doi.org/10.1073/pnas.1406298112>
- 894 Llopart, A., E. Brud, N. Pettie, and J. M. Comeron, 2018 Support for the dominance theory in *Drosophila* transcrip-  
895 tomes. *Genetics* 210: 703–718. <https://doi.org/10.1534/genetics.118.301229>
- 896 Mahajan, S., and D. Bachtrog, 2015 Partial dosage compensation in Strepsiptera, a sister group of beetles. *Genome Biol.*  
897 *Evol.* 7: 591–600. <https://academic.oup.com/gbe/article/7/2/591/630149>
- 898 Maheshwari, S., and D. A. Barbash, 2011 The genetics of hybrid incompatibilities. *Annu. Rev. Genet.* 45: 331–355.  
899 <https://doi.org/10.1146/annurev-genet-110410-132514>
- 900 Mallet, M. A., J. M. Bouchard, C. M. Kimber, and A. K. Chippindale, 2011 Experimental mutation-accumulation on  
901 the X chromosome of *Drosophila melanogaster* reveals stronger selection on males than females. *BMC Evol.*  
902 *Biol.* 11: 156. <https://doi.org/10.1186/1471-2148-11-156>
- 903 Mank, J. E., 2013 Sex chromosome dosage compensation: definitely not for everyone. *Trends Genet.* 29: 677–683.  
904 <https://doi.org/10.1016/j.tig.2013.07.005>
- 905 Mank, J. E., B. Vicoso, S. Berlin, and B. Charlesworth, 2010 Effective population size and the faster-X effect: empirical  
906 results and their interpretation. *Evolution* 64: 663–674. <https://doi.org/10.1111/j.1558-5646.2009.00853.x>
- 907 Manthey, J. D., and G. M. Spellman, 2014 Increased differentiation and reduced gene flow in sex chromosomes rela-  
908 tive to autosomes between lineages of the brown creeper *Certhia americana*. *J. Avian Biol.* 45: 149–156.  
909 <https://doi.org/10.1111/j.1600-048X.2013.00233.x>
- 910 Masly, J. P., and D. C. Presgraves, 2007 High-resolution genome-wide dissection of the two rules of speciation in  
911 *Drosophila*. *PLoS Biol.* 5: e243. <https://doi.org/10.1371/journal.pbio.0050243>
- 912 Matsubara, K., E. Yamamoto, R. Mizobuchi, J.-i. Yonemaru, T. Yamamoto, *et al.*, 2015 Hybrid breakdown caused  
913 by epistasis-based recessive incompatibility in a cross of rice (*Oryza sativa* L.). *J. Hered.* 106: 113–122.  
914 <https://doi.org/10.1093/jhered/esu065>
- 915 Meisler, R. P., and T. Connallon, 2013 The faster-X effect: integrating theory and data. *Trends Genet.* 29: 537–544.  
916 <https://doi.org/10.1016/j.tig.2013.05.009>



- 917 Muirhead, C. A., and D. C. Presgraves, 2016 Hybrid incompatibilities, local adaptation, and the genomic distribution  
918 of natural introgression between species. *Am. Nat.* 187: 249–261. <https://doi.org/10.1086/684583>
- 919 Muller, H. J., 1940 Bearing of the *Drosophila* work on systematics, pp. 185–268 in *The New Systematics*, edited by J.  
920 Huxley. Clarendon Press, Oxford.
- 921 Nei, M., and W.-H. Li, 1979 Mathematical model for studying genetic variation in terms of restriction endonucleases.  
922 *Proc. Natl. Acad. Sci. U.S.A* 76: 5269–5273. <https://doi.org/10.1073/pnas.76.10.5269>
- 923 Oswald, J. A., M. G. Harvey, R. C. Remsen, D. U. Foxworth, D. L. Dittmann, *et al.*, 2019 Evolutionary dynamics of  
924 hybridization and introgression following the recent colonization of glossy ibis (*Aves: Plegadis falcinellus*) into  
925 the new world. *Mol. Ecol.* 28: 1675–1691. <https://doi.org/10.1111/mec.15008>
- 926 Patterson, N., P. Moorjani, Y. Luo, S. Mallick, N. Rohland, *et al.*, 2012 Ancient admixture in human history. *Genetics*  
927 192: 1065–1093. <https://doi.org/10.1534/genetics.112.145037>
- 928 Payseur, B. A., D. C. Presgraves, and D. A. Filatov, 2018 Sex chromosomes and speciation. *Mol. Ecol.* 27: 3745.  
929 <https://doi.org/10.1111/mec.14828>
- 930 Phadnis, N., and H. A. Orr, 2009 A single gene causes both male sterility and segregation distortion in *Drosophila*  
931 hybrids. *Science* 323: 376–379. <https://doi.org/10.1126/science.1163934>
- 932 Pons, J.-M., C. Masson, G. Oliosio, and J. Fuchs, 2019 Gene flow and genetic admixture across a secondary contact  
933 zone between two divergent lineages of the Eurasian green woodpecker *Picus viridis*. *J. Ornithol.* 160: 935–945.  
934 <https://doi.org/10.1007/s10336-019-01675-6>
- 935 Presgraves, D. C., 2010 The molecular evolutionary basis of species formation. *Nat. Rev. Genet.* 11: 175–180.  
936 <https://doi.org/10.1038/nrg2718>
- 937 Presgraves, D. C., 2018 Evaluating genomic signatures of the large X-effect during complex speciation. *Mol. Ecol.* 27:  
938 3822–3830. <https://doi.org/10.1111/mec.14777>
- 939 Rafati, N., J. A. Blanco-Aguiar, C. J. Rubin, S. Sayyab, S. J. Sabatino, *et al.*, 2018 A genomic map of clinal variation  
940 across the European rabbit hybrid zone. *Mol. Ecol.* 27: 1457–1478. <https://doi.org/10.1111/mec.14494>
- 941 Ravinet, M., R. Faria, R. Butlin, J. Galindo, N. Bierne, *et al.*, 2017 Interpreting the genomic landscape of speciation:  
942 a road map for finding barriers to gene flow. *J. Evol. Biol.* 30: 1450–1477. <https://doi.org/10.1111/jeb.13047>
- 943 Roux, C., G. Tsagkogeorga, N. Bierne, and N. Galtier, 2013 Crossing the species barrier: genomic hotspots of  
944 introgression between two highly divergent *Ciona intestinalis* species. *Mol. Biol. Evol.* 30: 1574–1587.  
945 <https://doi.org/10.1093/molbev/mst066>
- 946 Sachdeva, H., and N. H. Barton, 2018 Introgression of a block of genome under infinitesimal selection. *Genetics* 209:  
947 1279–1303. <https://doi.org/10.1534/genetics.118.301018>

- 948 Satomura, K., N. Osada, and T. Endo, 2019 Achiasmy and sex chromosome evolution. *Ecol. Genet. Genom.* 13: 100046.  
949 <https://doi.org/10.1016/j.egg.2019.100046>
- 950 Schilthuizen, M., M. Giesbers, and L. Beukeboom, 2011 Haldane's rule in the 21st century. *Heredity* 107: 95–102.  
951 <https://doi.org/10.1038/hdy.2010.170>
- 952 Sciuchetti, L., C. Dufresnes, E. Cavoto, A. Brelsford, and N. Perrin, 2018 Dobzhansky-Muller incompatibilities, dominance drive, and sex-chromosome introgression at secondary contact zones: a simulation study. *Evolution* 72: 1350–1361. <https://doi.org/10.1111/evo.13510>
- 955 Sethuraman, A., V. Sousa, and J. Hey, 2019 Model-based assessments of differential introgression and linked natural selection during divergence and speciation. bioRxiv 786038. (Preprint posted September 30, 2019).  
956 <https://doi.org/10.1101/786038>
- 958 Sharp, N. P., and A. F. Agrawal, 2013 Male-biased fitness effects of spontaneous mutations in *Drosophila melanogaster*. *Evolution* 67: 1189–1195. <https://doi.org/10.1111/j.1558-5646.2012.01834.x>
- 960 Singh, A., and D. Punzalan, 2018 The strength of sex-specific selection in the wild. *Evolution* 72: 2818–2824.  
961 <https://doi.org/10.1111/evo.13625>
- 962 Sousa, V., and J. Hey, 2013 Understanding the origin of species with genome-scale data: modelling gene flow. *Nat. Rev. Genet.* 14: 404–414. <https://doi.org/10.1038/nrg3446>
- 964 Steinrücken, M., J. P. Spence, J. A. Kamm, E. Wicczorek, and Y. S. Song, 2018 Model-based detection and analysis of introgressed Neanderthal ancestry in modern humans. *Mol. Ecol.* 27: 3873–3888. <https://doi.org/10.1111/mec.14565>
- 966 Storchová, R., J. Reif, and M. W. Nachman, 2010 Female heterogamety and speciation: reduced introgression of the Z chromosome between two species of nightingales. *Evolution* 64: 456–471. <https://doi.org/10.1111/j.1558-5646.2009.00841.x>
- 969 Taylor, S. A., D. J. Anderson, and V. L. Friesen, 2013 Evidence for asymmetrical divergence-gene flow of nuclear loci, but not mitochondrial loci, between seabird sister species: blue-footed (*Sula nebouxi*) and Peruvian (*S. variegata*) boobies. *PloS One* 8: e62256. <https://doi.org/10.1371/journal.pone.0062256>
- 972 Taylor, S. A., R. L. Curry, T. A. White, V. Ferretti, and I. Lovette, 2014 Spatiotemporally consistent genomic signatures of reproductive isolation in a moving hybrid zone. *Evolution* 68: 3066–3081. <https://doi.org/10.1111/evo.12510>
- 974 Trier, C. N., J. S. Hermansen, G.-P. Sætre, and R. I. Bailey, 2014 Evidence for mito-nuclear and sex-linked reproductive barriers between the hybrid Italian sparrow and its parent species. *PLoS Genet.* 10: e1004075.  
975 <https://doi.org/10.1371/journal.pgen.1004075>
- 977 Trochet, A., E. A. Courtois, V. M. Stevens, M. Baguette, A. Chaine, D. S. Schmeller, and J. Clobert, 2016 Evolution of sex-biased dispersal. *Q. Rev. Biol.* 91: 297–320. <https://doi.org/10.1086/688097>
- 978

- 979 Turelli, M., and H. A. Orr, 1995 The dominance theory of Haldane's rule. *Genetics* 140: 389–402.
- 980 Turelli, M., and H. A. Orr, 2000 Dominance, epistasis and the genetics of postzygotic isolation. *Genetics* 154:  
981 1663–1679.
- 982 Turissini, D. A., and D. R. Matute, 2017 Fine scale mapping of genomic introgressions within the *Drosophila yakuba*  
983 clade. *PLoS Genet.* 13: e1006971. <https://doi.org/10.1371/journal.pgen.1006971>
- 984 Vicoso, B., and B. Charlesworth, 2009 Effective population size and the faster-X effect: an extended model. *Evolution*  
985 63: 2413–2426. <https://doi.org/10.1111/j.1558-5646.2009.00719.x>
- 986 Wang, L., K. Luzynski, J. E. Pool, V. Janousek, P. Dufková, *et al.*, 2011 Measures of linkage disequilibrium among  
987 neighbouring SNPs indicate asymmetries across the house mouse hybrid zone. *Mol. Ecol.* 20: 2985–3000.  
988 <https://doi.org/10.1111/j.1365-294X.2011.05148.x>
- 989 Wang, R.-X., 2013 Gene flow across a hybrid zone maintained by a weak heterogametic incompatibility and positive  
990 selection of incompatible alleles. *J. Evol. Biol.* 26: 386–398. <https://doi.org/10.1111/jeb.12057>
- 991 Wang, W., C. Dai, P. Alström, C. Zhang, Y. Qu, *et al.*, 2014 Past hybridization between two East Asian long-tailed  
992 tits (*Aegithalos bonvaloti* and *A. fuliginosus*). *Front. Zool.* 11: 40. <https://doi.org/10.1186/1742-9994-11-40>
- 993 Watson, E. T., and J. P. Demuth, 2012 Haldane's rule in Marsupials: what happens when both sexes are functionally  
994 hemizygous? *J. Hered.* 103: 453–458. <https://doi.org/10.1093/jhered/esr154>
- 995 Weir, B. S., and C. C. Cockerham, 1984 Estimating  $F$ -statistics for the analysis of population structure. *Evolution*  
996 38: 1358–1370.
- 997 Wilkinson-Herbots, H. M., 2008 The distribution of the coalescence time and the number of pairwise nucleotide differences  
998 in the Isolation-with-Migration model. *Theor. Popul. Biol.* 73: 277–288. <https://doi.org/10.1016/j.tpb.2007.11.001>
- 999 Wong Miller, K. M., R. R. Bracewell, M. B. Eisen, and D. Bachtrog, 2017 Patterns of genome-wide diversity  
1000 and population structure in the *Drosophila atabasca* species complex. *Mol. Biol. Evol.* 34: 1912–1923.  
1001 <https://doi.org/10.1093/molbev/msx134>

1002 **Table 1. Notation**

Parameters	Meaning
$L$	number of selected loci equally spaced on the genomic block
$c_F, c_M$	rate of recombination per generation between adjacent selected loci in ♀ (resp. ♂)
$\alpha$	map distance between the neutral marker and the nearest selected locus, relative to the interlocus map distance
$m_F, m_M$	fraction of females (resp. males) in the recipient species replaced by migrant females (males) in each generation
$s_F, s_M$	heterozygous (resp. hemizygous) selective effect per locally deleterious allele in females (males)
Quantities	Meaning
$P_F(y) ; P_M(y)$	fraction of females (resp. males) carrying a single introgressing block with $y$ locally deleterious loci X CHROMOSOME: $P_X(y) = \frac{P_F(y)+P_M(y)}{3}$ ; AUTOSOME: $P_A(y) = \frac{P_F(y)+P_M(y)}{2}$
$\bar{p}_X ; \bar{p}_A$	frequency of locally deleterious alleles on the sex chromosome (resp. autosomes) averaged over all selected loci
$\bar{u}_X ; \bar{u}_A$	frequency of the neutral marker at the end of the sex chromosome (resp. autosomes) averaged over all deleterious backgrounds
$b_X ; b_A$	strength of the sex chromosomal barrier to neutral gene flow (resp. autosomal)
Model extensions	Meaning
$h$	dominance coefficient of the locally deleterious alleles $h = 0.5$ : $\infty$ -dominance; $h = 0.1$ : partial recessivity; $h = 1.0$ : complete dominance
$s_{hom,F} = \frac{s_F}{h} ; s_{hom,M} = \frac{s_M}{h}$	homozygous selective effect per locally deleterious allele in females (resp. males)
$v_F(y, y') ; v_M(y, y')$	fitness of a female (resp. male) carrying $y$ locally deleterious loci in the heterozygous state and $y'$ loci in the homozygous state X CHROMOSOME: $v_F(y, y') = e^{-y s_F - y' s_{hom,F}}$ , $v_M(y, y') = e^{-y s_M}$ ; AUTOSOME: $v_F(y, y') = e^{-y s_F - y' s_{hom,F}}$ , $v_M(y, y') = e^{-y s_M - y' s_{hom,M}}$
$\beta$	fraction of selected loci that act epistatically, the remaining $1 - \beta$ acts multiplicatively
$\varepsilon$	direction and strength of epistasis $\varepsilon = 1$ : multiplicativity; $0 \leq \varepsilon < 1$ : negative epistasis

1009 Note that throughout we use  $s$  to denote the heterozygote selective effect (unlike the more standard notation, where  
1010  $s$  refers to the homozygous effect). Our choice of notation is motivated by the fact that to the first order in  $sL$  (where  $L$   
1011 is the number of selected loci on the block) introgression depends only on this single parameter and is independent of the  
1012 dominance coefficient,  $h$ .

1014 **Table 2. Sex-averaged effective parameters**

Parameters	Meaning
$s_X ; s_A$	effective heterozygous selection per locally deleterious allele on the X chromosome (resp. autosomes) X CHROMOSOME: $s_X = \frac{2s_F + s_M}{3}$ ; AUTOSOME: $s_A = \frac{s_F + s_M}{2}$
$m_X ; m_A$	effective migration on the X chromosome (resp. autosomes) X CHROMOSOME: $m_X = \frac{2m_F + m_M}{3}$ ; AUTOSOME: $m_A = \frac{m_F + m_M}{2}$
$c_X ; c_A$	effective recombination on the X chromosome (resp. autosomes) X CHROMOSOME: $c_X = \frac{2c_F}{3}$ ; AUTOSOME: $c_A = \frac{c_F + c_M}{2}$
$\theta_X ; \theta_A$	strength of coupling between locally deleterious alleles on the sex chromosome (resp. autosomes) X CHROMOSOME: $\theta_X = \frac{s_X}{c_X}$ ; AUTOSOME: $\theta_A = \frac{s_A}{c_A}$
$\frac{m_X}{s_X} ; \frac{m_A}{s_A}$	expected allele frequency when selected loci are perfectly linked on the sex chromosome (resp. autosomes)

1017 These effective parameters govern introgression assuming that  $sL$ ,  $cL$  and  $m$  are  $\ll 1$ .

1018

1019 **Table 3. Studies testing whether the sex chromosome is a stronger barrier to gene flow than autosomes**

XY SEXUAL SYSTEMS	INFERENCE		
	INTROG	A LOCUS	X LOCUS
<b>Insects</b>			
<b>Orthoptera</b>			
° <i>Gryllus pennsylvanicus</i> / <i>G. firmus</i> (1)	$X < A$	genome-wide	genome-wide
<b>Diptera</b>			
# <i>Drosophila yakuba</i> / <i>D. santomea</i> (2)	$X < A$	genome-wide	genome-wide
. <i>Drosophila melanogaster</i> EUR / AFR (3)	$X \geq A$	genome-wide	genome-wide
. <i>Drosophila melanogaster</i> / <i>D. simulans</i> (4)	$X \geq A$	genome-wide	genome-wide
. <i>Drosophila subquinaria</i> / <i>D. recens</i> (5)	$X \geq A$	19	7
. <i>Drosophila persimilis</i> / <i>D. pseudoobscura</i> (6)	$X \geq A$	genome-wide	genome-wide
1020 . <i>Drosophila subobscura</i> / <i>D. madeirensis</i> (7)	$X \geq A$	16	3
. <i>Drosophila athabasca</i> WEST / EAST (8)	$X \geq A$	genome-wide	genome-wide
<b>Mammals</b>			
	-	-	-
<b>Primates</b>			
# <i>Homo sapiens</i> / <i>H. neanderthalensis</i> (9)	$X < A$	genome-wide	genome-wide
° <i>Alouatta palliata</i> / <i>A. nigra</i> (10)	$X \geq A$	genome-wide	genome-wide
<b>Rodentia</b>			
° <i>Mus m. musculus</i> / <i>Mus m. domesticus</i> (11)	$X < A$	genome-wide	genome-wide
# <i>Mus m. domesticus</i> / <i>Mus spretus</i> (12)	$X < A$	genome-wide	genome-wide
<b>Lagomorpha</b>			
° <i>Oryctolagus c. cuniculus</i> / <i>O. c. algirus</i> (13)	$X < A$	genome-wide	genome-wide

1021

1022 **References:** (1) Larson et al. 2014; (2) Turissini and Matute 2017; (3) Kapopoulou et al. 2018; (4) Dalquen et al.  
1023 2017; (5) Ginsberg et al. 2019; (6) Fuller et al. 2018; (7) Herrig et al. 2014; (8) Wong Miller et al. 2017; (9) Steinrücken  
1024 et al. 2018; (10) Baiz et al. 2019; (11) Wang et al. 2011; (12) Liu et al. 2015; (13) Carneiro et al. 2010; Rafati et al. 2018;  
1025 (14) Backström and Väli 2011; (15) Dhimi et al. 2016; (16) Galla and Johnson 2015; (17) Taylor et al. 2013; (18) Oswald  
1026 et al. 2019; (19) Pons et al. 2019; (20) Bourgeois et al. 2018; (21) Carling et al. 2010; (22) Trier et al. 2014; (23) Hooper  
1027 et al. 2019; (24) Taylor et al. 2014; (25) Manthey and Spellman 2014; (26) Storchová et al. 2010; (27) Hogner et al. 2012;  
1028 (28) Chu et al. 2013; (29) Wang et al. 2014; (30) Knief et al. 2019.

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ZW SEXUAL SYSTEMS	INFERENCE		
	INTROG	A LOCUS	X LOCUS
<b>Birds</b>	-	-	-
<b>Accipitriformes</b>			
. <i>Aquila clanga</i> / <i>A. pomarina</i> (14)	$X < A$	36	15
<b>Anseriformes</b>			
. <i>Anas castanea</i> / <i>A. gracilis</i> (15)	$X \geq A$	17	7
<b>Galliformes</b>			
. <i>Tympanuchus cupido</i> / <i>T. pallidicinctus</i> / <i>T. phasianellus</i> (16)	$X < A$	4	5
<b>Pelecaniformes</b>			
. <i>Sula nebouxii</i> / <i>S. variegata</i> (17)	$X \geq A$	4	3
° <i>Plegadis falcinellus</i> / <i>P. chihi</i> (18)	$X \geq A$	genome-wide	genome-wide
<b>Piciformes</b>			
° <i>Picus v. viridis</i> / <i>P. sharpei</i> (19)	$X \geq A$	9	10
<b>Passeriformes</b>			
. <i>Zosterops borbonicus</i> LOW / HIGH (20)	$X < A$	genome-wide	genome-wide
. <i>Passerina amoena</i> / <i>P. cyanea</i> (21)	$X < A$	13	6
° <i>Passer domesticus</i> / <i>P. hispaniolensis</i> (22)	$X < A$	genome-wide	genome-wide
° <i>Poephila acuticauda</i> / <i>P. cincta</i> (23)	$X < A$	genome-wide	genome-wide
° <i>Poecile atricapillus</i> / <i>P. carolinensis</i> (24)	$X < A$	genome-wide	genome-wide
. <i>Certhia americana</i> SOU / NOR (25)	$X < A$	21	9
. <i>Luscinia luscinia</i> / <i>L. megarhynchos</i> (26)	$X < A$	8	4
. <i>Ficedula albicollis</i> / <i>F. hypoleuca</i> (27)	$X \geq A$	6	5
. <i>Carpodacus vinaceus</i> / <i>C. formosanus</i> (28)	$X \geq A$	25	10
. <i>Aegithalos bonvaloti</i> / <i>A. fuliginosus</i> (29)	$X \geq A$	3	3
° <i>Corvus c. corone</i> / <i>C. c. cornix</i> (30)	$X \geq A$	genome-wide	genome-wide

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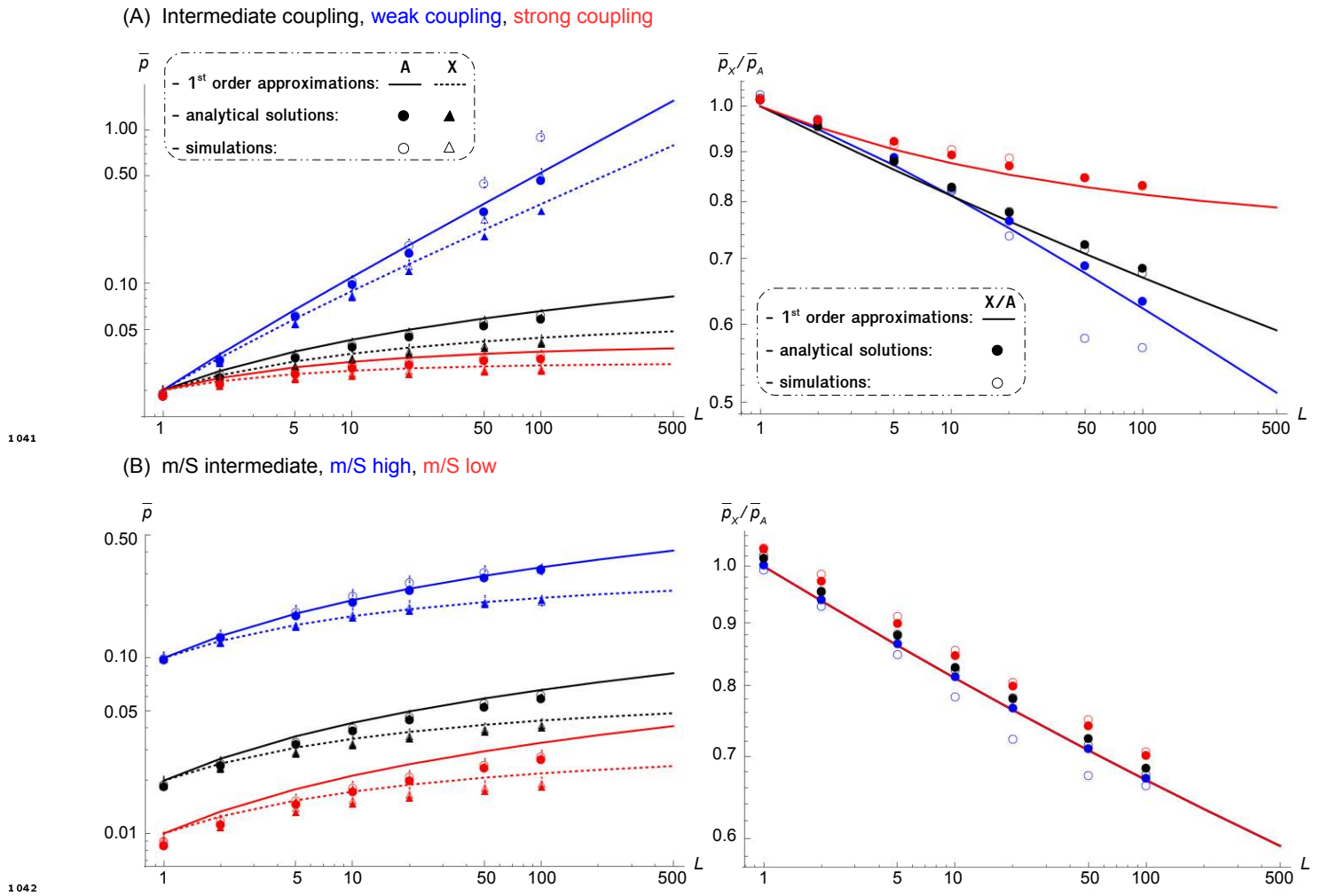
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Inferences are either based on (.) the migration rates under isolation-with-migration models (Sousa and Hey 2013), (#) the number of introgressed tracts (Lawson et al. 2012), or (°) the geographic/genomic cline widths (Barton and Hewitt 1985). We discarded studies only based on descriptive statistics such as  $F_{ST}$  (Weir and Cockerham 1984),  $G_{MIN}$  (Geneva et al. 2015) or  $f$ -statistics (Patterson et al. 2012). . **INTROG**: if  $X < A$  then the sex chromosome is a stronger

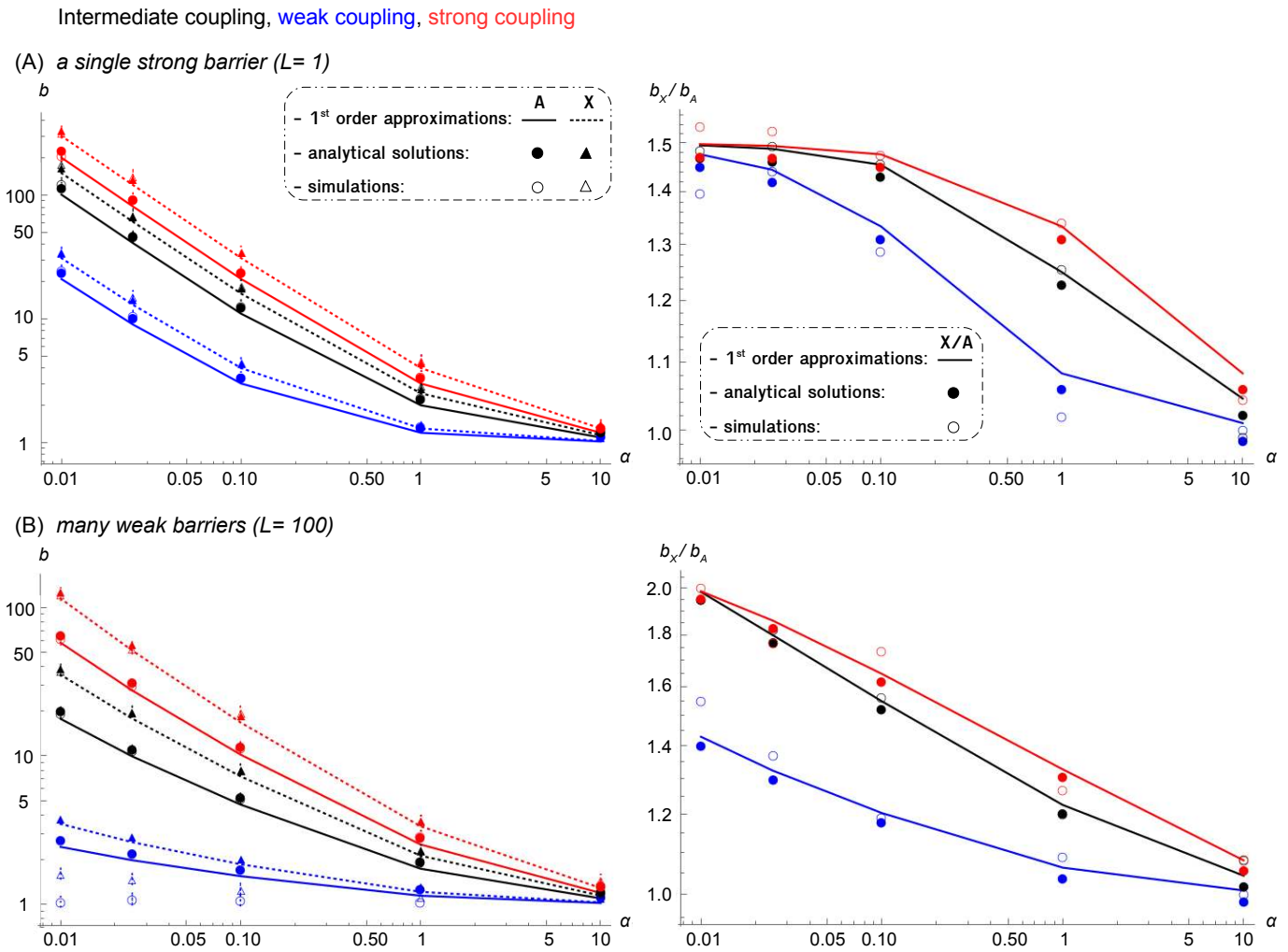
1036 barrier to gene flow than autosomes (either because it has a lower migration rate, a lower number of introgressed tract or  
1037 a steeper cline),  $X \geq A$  otherwise. **A LOCUS / X LOCUS**: number of autosomal/X-linked loci available for the inferences.  
1038 Redundant studies were eliminated by including only the more recent study with larger numbers of sampled individuals  
1039 and/or genetic markers. Studies with less than three loci available for sex chromosomes and/or autosomes were discarded.  
1040 Species with young sex chromosomes, as in amphibians and plants, were not considered.



1043 **Figure 1. Effect of  $\theta$  and  $\frac{m}{S}$  on average equilibrium frequencies**

1044 Average frequency of the deleterious alleles in the recipient species (Autosomal:  $\bar{p}_A$ ; X-linked:  $\bar{p}_X$ ; their ratio:  $\frac{\bar{p}_X}{\bar{p}_A}$ )  
 1045 plotted against the number of selected loci on the genomic block,  $L$ . The effect of two composite parameters governing  
 1046 introgression is shown: (A)  $\frac{m}{S}$  determines the expected allele frequency when selected loci are perfectly linked; (B)  $\theta = \frac{s}{c}$   
 1047 determines the extent of coupling between loci. Lines show the analytical expression in eqs. (4) and (12) (which are  
 1048 given in terms of composite parameters and are valid for  $sL$ ,  $cL$  and  $m \ll 1$ ); filled symbols show analytical expression in  
 1049 eqs. (11) and (16) (which are also valid for strong selection); empty symbols show results of individual-based simulations  
 1050 iterated for 10,000 generations and averaged over 100 replicates (error bars indicate the standard error of the mean).  
 1051 Colors stand for values of the composite parameters,  $\theta$  and  $\frac{m}{S}$ . Parameter values for intermediate  $\theta$  and  $\frac{m}{S}$  are:  $h = 0.5$ ,  
 1052  $m = 0.001$ ,  $cL = 0.05$ ,  $sL = 0.05$ ,  $m_X/S_X = m_A/S_A = 0.02$ ,  $\theta_X = 1.5$ ,  $\theta_A = 1$  and  $N = 10^5$  (simulations). Parameters for  
 1053 weak coupling:  $cL = 0.25$ ,  $\theta_X = 0.3$ ,  $\theta_A = 0.2$ ; strong coupling:  $cL = 0.025$ ,  $\theta_X = 3$ ,  $\theta_A = 2$ ;  $m/S$  high:  $cL = sL = 0.01$ ,  
 1054  $m_X/S_X = m_A/S_A = 0.1$ ;  $m/S$  low:  $cL = sL = 0.1$ ,  $m_X/S_X = m_A/S_A = 0.01$ . Note that the total selective disadvantage  
 1055 of the block ( $S = sL$ ) and its total map length ( $C = cL$ ) are kept constant as we vary the number of loci ( $L$ ; x axis).



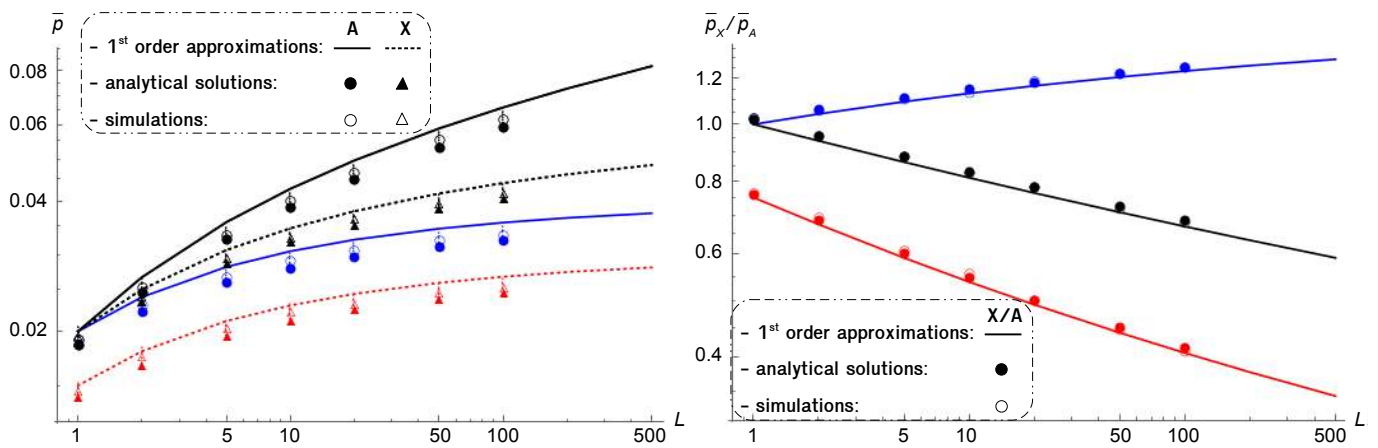


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1057 **Figure 2. Effect of  $\theta$  on barrier strength at the neutral marker**

1058 Barrier strength at the neutral marker (Autosomal:  $b_A$ ; X-linked:  $b_X$ ; their ratio:  $\frac{b_X}{b_A}$ ) plotted against its proximity to  
 1059 the nearest selected locus,  $\alpha$ . In (A) the neutral marker is linked to a single strongly selected locus ( $L = 1$ ); while in  
 1060 (B) it is linked to one hundred weakly selected loci ( $L = 100$ ). Lines show the analytical expression in eqs. (5) and (6)  
 1061 (which are given in terms of composite parameters and are valid for  $sL, cL$  and  $m \ll 1$ ); filled symbols show analytical  
 1062 expression in eqs. (23) and (30) (which are also valid for strong selection); empty symbols show results of individual-based  
 1063 simulations iterated for 10,000 generations after equilibrating at the selected loci; they are averaged over 100 replicates  
 1064 (error bars indicate the confidence interval of the estimate). Colors stand for values of the composite parameter  $\theta = \frac{s}{c}$   
 1065 at the selected loci. The recombination rate between the neutral marker and the nearest selected locus is  $\alpha Lc$ , therefore  
 1066 lower  $\alpha$  values means closer proximity. Other details match Figure 1.

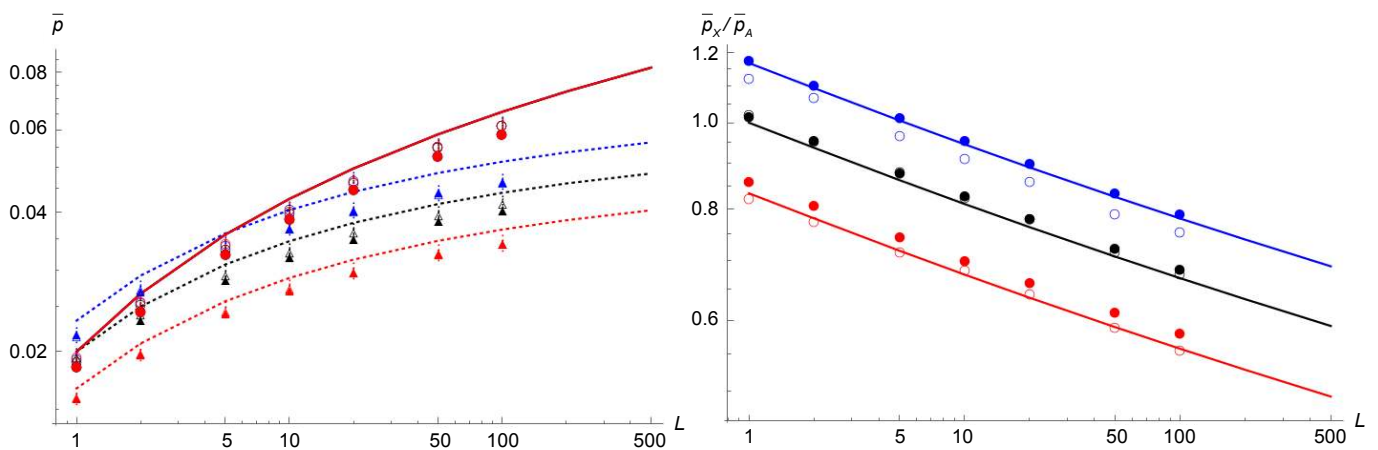
(A) Basic model, **achiasmy**, **dosage compensation**



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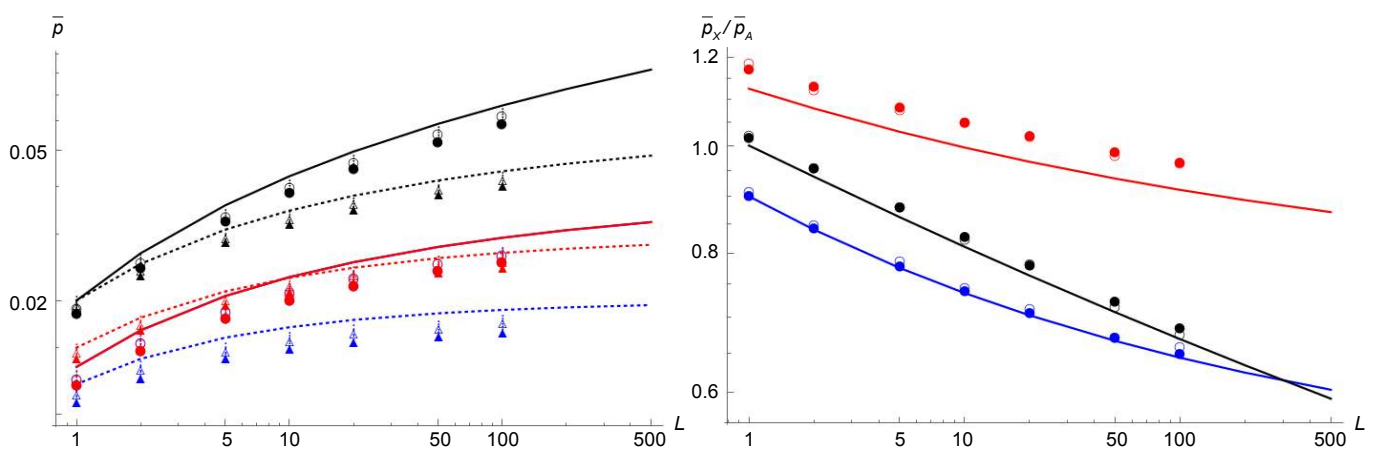
(B) Basic model, **♀<sub>XX</sub>-biased migration**, **♂<sub>XY</sub>-biased migration**



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(C) Basic model, **♂<sub>ZZ</sub>-biased selection**, **♂<sub>XY</sub>-biased selection**



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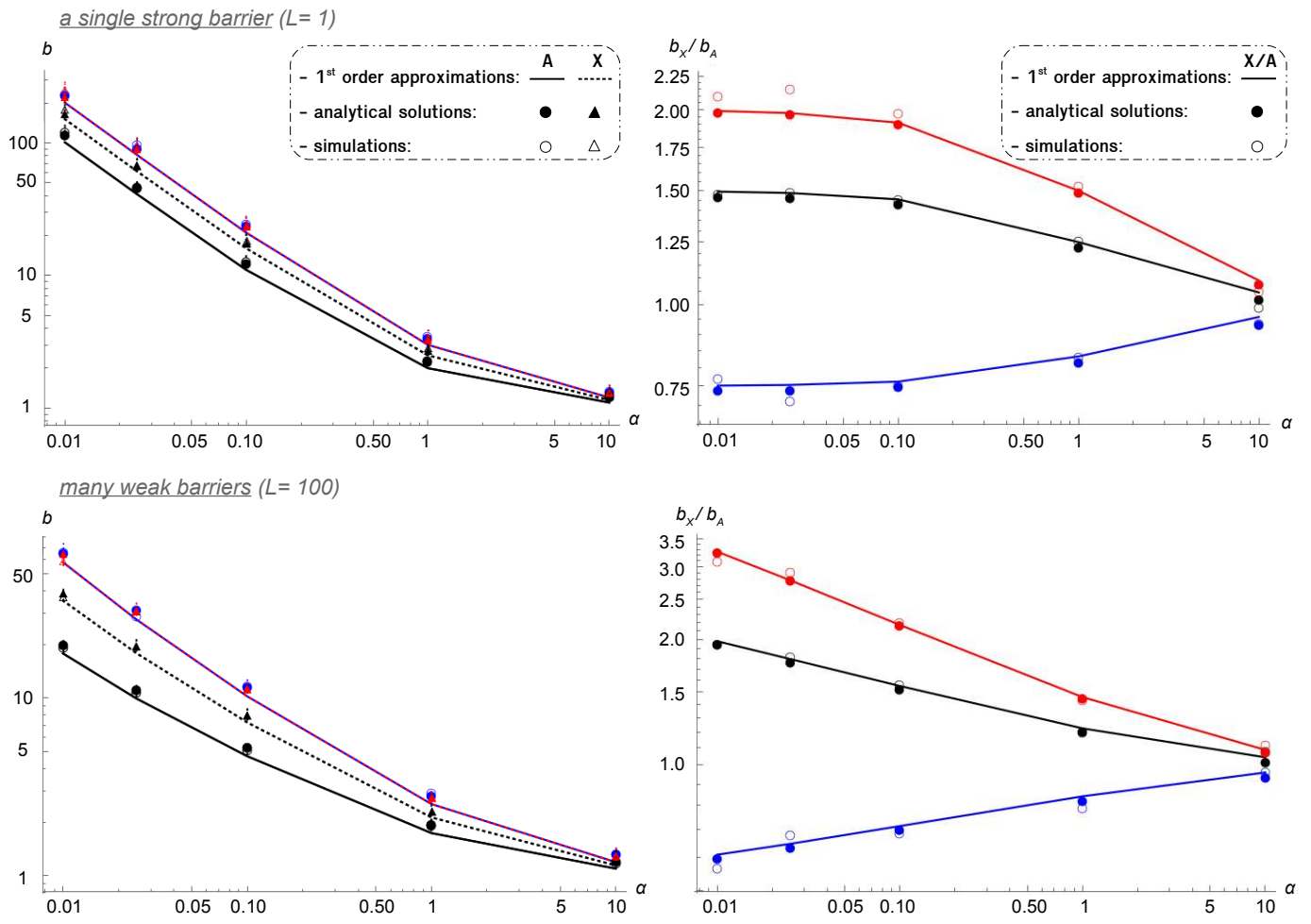
1072 **Figure 3. Effect of sex-specificities on average equilibrium frequencies**

1073 Average frequency of the deleterious alleles in the recipient species (Autosomal:  $\bar{p}_A$ ; X-linked:  $\bar{p}_X$ ; their ratio:  $\frac{\bar{p}_X}{\bar{p}_A}$ ) plotted

1074 against the number of selected loci on the genomic block,  $L$ . (A) Dosage compensation ( $s_M L = 2s_F L = s_{hom,F} L = 0.1$ )

1075 for sex-linked alleles,  $m_X/S_X = 0.015$ ,  $\theta_X = 2$ , red) and achiasmy in XY males ( $c_M L = 0$ ,  $\theta_A = 2$ , blue). **(B)** Sex-biased  
1076 migration: e.g., if males migrate three times more than females ( $m_M = 3m_F = 0.0015$ ,  $m_X/S_X \sim 0.016$ , red); or females  
1077 migrate three times more than males ( $m_F = 3m_M = 0.0015$ ,  $m_X/S_X \sim 0.023$ , blue). **(C)** Sex-biased selection: e.g. if  
1078 selection coefficient is twice as strong on XY males as on females ( $s_M L = 2s_F L = 0.1$ ,  $m_X/S_X \sim 0.015$ ,  $m_A/S_A \sim 0.013$ ,  
1079  $\theta_X = 2$ ,  $\theta_A = 1.5$ , red); or selection coefficient is twice as strong on ZZ males as on females ( $s_F L = 2s_M L = 0.1$  in our XY  
1080 notation,  $m_Z/S_Z \sim 0.012$ ,  $m_A/S_A \sim 0.013$ ,  $\theta_Z = 2.5$ ,  $\theta_A = 1.5$ , blue). The basic model is shown in black in all panels.  
1081 Parameter values for the basic model are:  $h = 0.5$ ,  $m = 0.001$ ,  $cL = 0.05$ ,  $sL = 0.05$ ,  $m_X/S_X = m_A/S_A = 0.02$ ,  $\theta_X = 1.5$ ,  
1082  $\theta_A = 1$  and  $N = 10^5$  (simulations). Other details match Figure 1.

(A) Basic model, **achiasmy**, dosage compensation

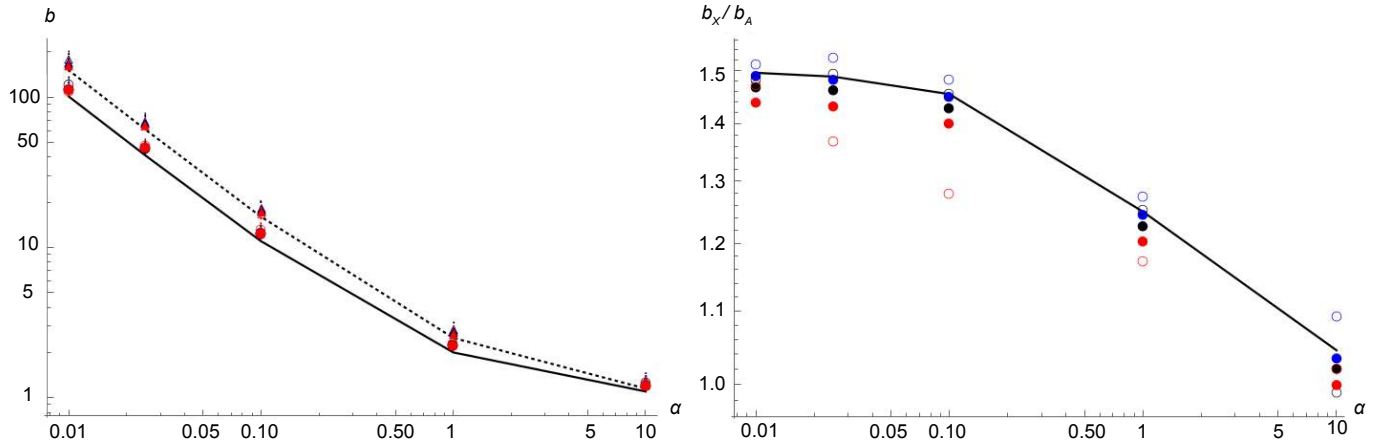


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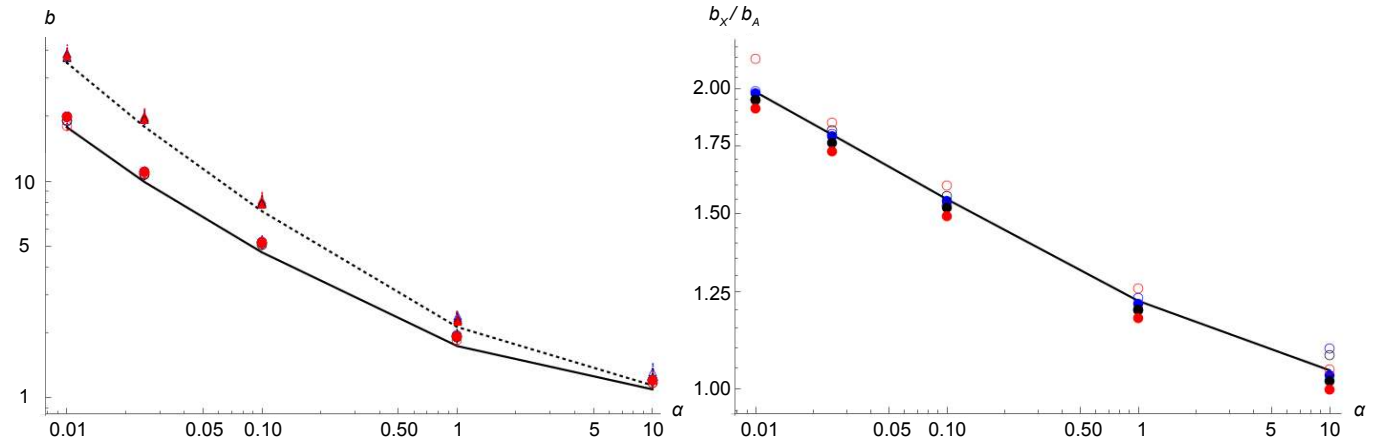
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(B) Basic model, ♀<sub>xx</sub>-biased migration, ♂<sub>xy</sub>-biased migration

*a single strong barrier (L = 1)*



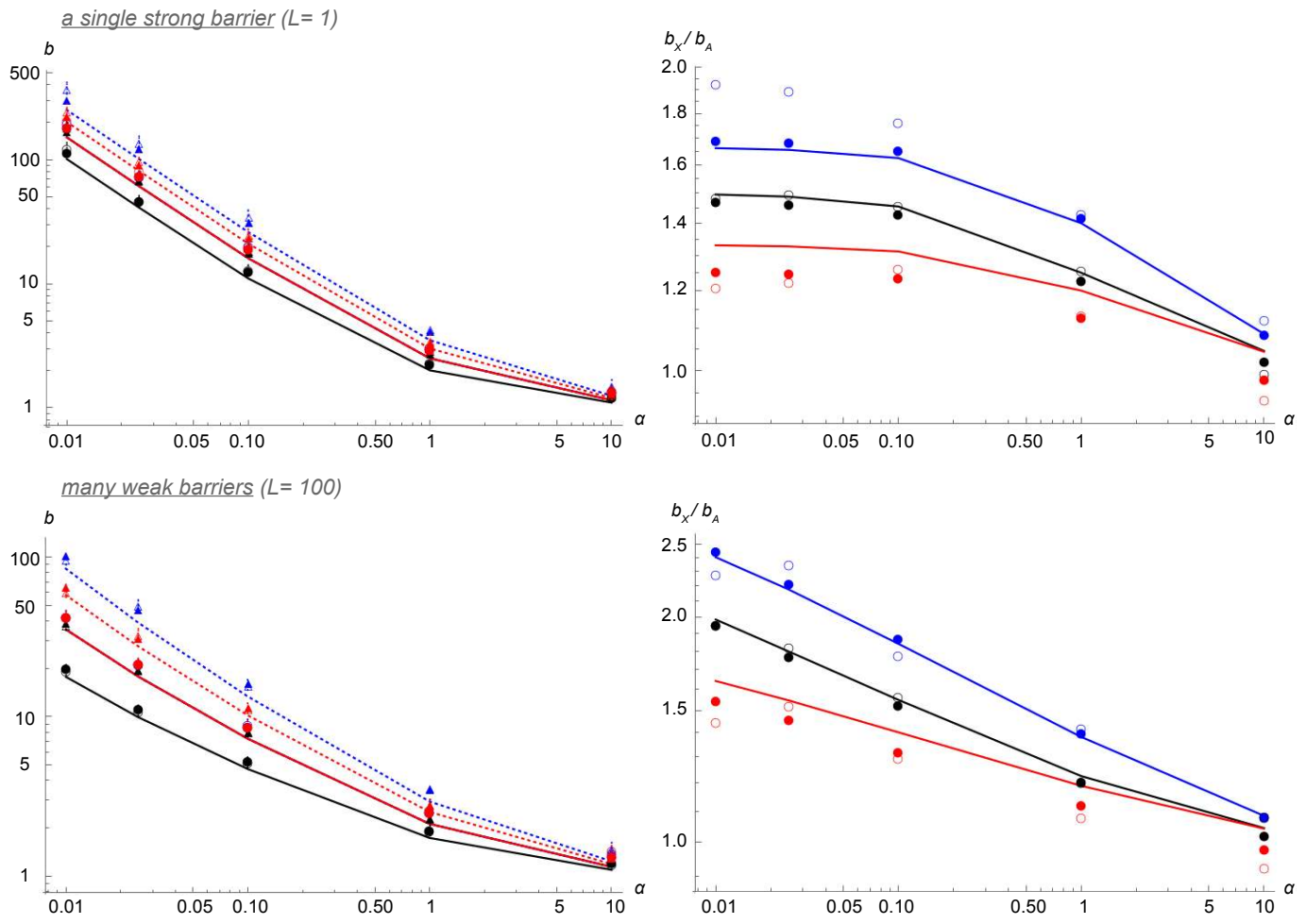
*many weak barriers (L = 100)*



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(C) Basic model, ♂<sub>ZZ</sub>-biased selection, ♂<sub>XY</sub>-biased selection



1087

1088 **Figure 4. Effect of sex-specificities on barrier strength at the neutral marker**

1089 Barrier strength at the neutral marker (Autosomal:  $b_A$ ; X-linked:  $b_X$ ; their ratio:  $\frac{b_X}{b_A}$ ) plotted against its proximity to the  
 1090 nearest selected locus,  $\alpha$ . (A) Dosage compensation (red) and achiasmy in XY males (blue). (B) Sex-biased migration:  
 1091 e.g., if males migrate three times more than females (red); or females migrate three times more than males (blue). (C)  
 1092 Sex-biased selection: e.g. if selection coefficient is twice as strong on XY males as on females (red); or selection coefficient  
 1093 is twice as strong on ZZ males as on females (blue). The basic model is shown in black in all panels. Other details match  
 1094 Figure 2 and 3.