Models of sex-ratio meiotic drive and sexual selection in stalk-eyed flies

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Summary

Hypertrophied sexually dimorphic eye stalks have evolved independently in several families of Diptera, with the eyespan of males exceeding their total body length in some species. These structures function in intermale contests for territories and in mate attraction, the classical mechanisms of sexual selection. In the family Diopsidae, species with extremely exaggerated eye stalks and marked sexual dimorphism in relative eyespan also usually have strongly female-biased sex ratios in nature caused by *X*-linked meiotic drive, whereas species with relatively small eye stalks have little or no sexual dimorphism, often lack meiotic drive and have even sex ratios. We investigate the possible connection between sexual selection and sex-ratio meiotic drive by analysing a three-locus model for the evolution of female choice for a male character associated with meiotic drive. Both meiotic drive and the male character are *X*-linked and the female preference is autosomal. Our model shows that suppressed recombination between meiotic drive and the male character, e.g. by inversion of the *X* chromosome, is necessary for sex-ratio selection to promote the origin of female mating preferences and exaggerated secondary sexual characters. With complete suppression of recombination, sexual selection reduces the frequency of meiotic drive, and may eliminate it. Very rare recombination, gene conversion or mutation, at rates characteristic of chromosome inversions in *Drosophila*, restores the meiotic drive polymorphism to its original equilibrium. Sex-ratio meiotic drive may thus act as a catalyst accelerating the origin of female mating preference and exaggerated male traits.

1. Introduction

Among the most bizarre morphological characters in the Diptera are the hypertrophied eye stalks and antlers that have evolved independently in several families, including the Drosophilidae, Diopsidae, Otitidae and Richardiidae (Wilkinson & Dodson, 1997). These structures generally are more exaggerated in males than in females and in some species the eye span of males exceeds their total body length (Fig. 1, Table 1). Even the monomorphic species of diopsids have relative eye spans much wider than most other Diptera (Grimaldi & Fenster, 1989; Wilkinson & Dodson, 1997). Eye span in diopsids therefore appears predisposed to function as an indicator of general body size and fighting ability when males face each other prior to combat (Wilkinson & Dodson, 1997). Exaggerated male characters often evolve as a result of both intermale contests and female mating preference, and in such cases it generally is difficult or impossible to distinguish which came first or whether they evolved together (Darwin, 1874; Fisher, 1958).

Artificial selection experiments on the Malaysian stalk-eyed fly *Cyrtodiopsis dalmanni* demonstrated that relative eye span (eye span/body length) in males has a realized heritability of 0·35 with a realized genetic correlation of 0·39 between male and female relative eye span (Wilkinson, 1993). After 14 generations of selection on male relative eye span, comparison of the mean phenotypes of F₁ males in reciprocal crosses between high and low lines showed that 14 % of the selection response was attributable to *X*-linked loci, and with continued selection to 32 generations *X*-linked loci contributed 32 % of the

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Fig. 1. Silhouettes of male stalk-eyed flies, *Cyrtodiopsis quinquegutta* (left) and *C. whitei* (right).

Table 1. *Sexual dimorphism in relative eye span* (*eye span/body length*) *and frequency of* X-*linked meiotic drive* (% *males with highly significant female-biased progeny sex-ratios*, $P < 0.01$) *in natural populations of stalk-eyed flies* (*Diopsidae*)

| | Mean relative eye span | | Meiotic drive | |
| | | | Males (%) | (Males[a], mean progeny[b]) |
Species	Male	Female		
Sphyracephala beccarrii	0·47	0·44	8%	(96, 80·8)
Teleopsis quadriguttata	0·67	0·65	0	(69, 37·9)
Cyrtodiopsis quinquegutta	0·61	0·60	1%	(103, 34·5)
C. dalmanni	1·20	0·89	10%	(93, 114·7)
C. whitei	1·25	0·84	33%	(64, 226·2)
Diasemopsis sylvatica	1·12	0·79	15%	(13, 44·9)
D. dubia	1·12	0·82	0	(108, 37·3)

From Wilkinson and Dodson (1997), Wilkinson *et al.* (1998b) and unpublished data.
All species have mean body length about 5–7 mm.
[a] Number of males tested for female-biased progeny sex ratio.
[b] Mean number of progeny per male.

total response (L. Wolfenbarger & G. S. Wilkinson, unpublished data). In controls and in lines selected for increased relative eye span in males, females preferred to mate with males with large eye span, but in lines selected for decreased relative eye span in males, females preferred males with short eye span (Wilkinson & Reillo, 1994). This demonstrates that female mating preference for male relative eye span is partly heritable and has a positive additive genetic correlation with male relative eye span, as predicted by population genetic models of sexual selection (because of assortative mating due to heritable variance in mating preferences: Lande, 1981; Kirkpatrick, 1982). Field observations on *C. dalmanni*

provided no evidence that mate choice affects female viability or fecundity (Wilkinson and Reillo, 1994).

Recent observations reveal that diopsid species with extremely exaggerated eye stalks and marked sexual dimorphism in relative eye span also often have strongly female-biased sex ratios in nature caused by X-linked meiotic drive similar to that first observed in *Drosophila pseudoobscura* (references in Powell, 1992). *Cyrtodiopsis dalmanni* males with X-linked meiotic drive produce a small percentage of sons, which have normal fertility (Presgraves *et al.*, 1997). In contrast, species with relatively small eye stalks and little or no sexual dimorphism tend to have even sex ratios and lack X-linked meiotic drive (see Table 1). Females of

the dimorphic species remate more frequently than females of the monomorphic species (Wilkinson *et al.*, 1998*a*), perhaps in order to select against meiotic drive by sperm competition since driving males produce less sperm than non-driving males (Wu, 1983*a*, *b*; Haigh & Bergstrom, 1995; Presgraves *et al.*, 1997). Furthermore, after 22 generations of artificial selection on male relative eye span in *C. dalmanni*, one of two low lines showed a significant increase in frequency of the *X*-linked meiotic drive, and both high lines showed significant increases in the frequency of suppressors of meiotic drive (Wilkinson *et al.*, 1998*b*). Backcrossing the high lines to controls showed that *Y*-linked genes do not affect male relative eye span as initially proposed (L. Wolfenbarger & G. S. Wilkinson, unpublished data), which is consistent with the degenerate *Y* chromosome in Diptera having relatively few functional genes (White, 1973; Charlesworth, 1991).

These observations suggest a possible causal connection between sexual selection and sex-ratio selection (Presgraves *et al.*, 1997). In a species with an established polymorphism for *X*-linked meiotic drive, *X*-linked alleles that increase male relative eye span might be used by females as an indicator of lack of meiotic drive or of meiotic drive suppression in males (Wilkinson *et al.*, 1998*b*). Female mate choice against male meiotic drive would help females achieve a more even (or male-biased) progeny sex ratio that is highly advantageous in a population with a strongly female-biased sex ratio (Fisher, 1958; Hamilton, 1967).

Such a mechanism would provide a compelling selective explanation for the origin of female mating preferences and the evolution of exaggerated secondary sexual characters in diopsids and other Diptera. In polygamous species, female mating preferences are thought to originate either as a correlated response to selection on male characters (Fisher, 1958; Lande, 1981; Kirkpatrick, 1982), by sensory bias – where the more conspicuous males are noticed more easily by females (Kirkpatrick & Ryan, 1991), or because of 'good genes' that chosen males pass on to their offspring. Wilkinson *et al.* (1998*a*) observed significant female preference for large male eye span in two sexually dimorphic congeners with exaggerated male eye span, *C. dalmanni* and *C. whitei*, but discounted the sensory bias hypothesis because they failed to detect female preference for male eye span in the primitive monomorphic species *C. quinqueguttata*. Despite their popular appeal as an adaptive mechanism for the origin and maintenance of female mating preferences, 'good genes' have rarely if ever been identified, and their ability to explain exaggerated secondary sexual characters appears to be limited (Kirkpatrick, 1996). Alleles that indicate lack or suppression of sex-ratio meiotic drive would be among the first concrete examples of 'good genes' utilized as cues for female mate choice (e.g. Lenington *et al.*, 1994).

Sex-ratio meiotic drive polymorphisms are common in Diptera (Lyttle, 1991; Jaenike, 1996), in part because Diptera possess special genetic mechanisms that facilitate reduced recombination. The developmental mechanism of meiotic drive operates by the destruction of non-driving sperm during spermatogenesis (Policansky & Ellison, 1970; Lyttle, 1991; Presgraves *et al.*, 1997). The underlying genetic mechanism depends on a distorter locus, with driving and non-driving alleles, and a responder locus, with susceptible and resistant alleles (Hartl, 1974). Distorter chromosomes do not destroy themselves because they are associated with resistant responder alleles, and suppressed recombination between distorter and responder loci is necessary to prevent meiotic drive elements from suicide (Charlesworth & Hartl, 1978). In dipteran species males usually lack recombination, and in females recombination is often locally suppressed by paracentric chromosomal inversions. Crossing-over is suppressed near the breakpoints of heterozygous inversions, so small inversions effectively suppress recombination within and around them (Roberts, 1976). Most types of crossovers within paracentric inversion heterozygotes produce major duplications and deletions that would be zygotic lethals, but these aneuploid products are shunted into the polar bodies rather than the egg nucleus during meiosis of female Diptera (Patterson & Stone, 1952; White, 1973).

The sex-ratio distorters in *Drosophila pseudoobscura* and several other *Drosophila* species, and the autosomal segregation distorter in *D. melanogaster*, are associated with inversions (Charlesworth & Hartl, 1978; Lyttle, 1991; Powell, 1992); however *D. neotestacea* and *D. simulans* are exceptions (Jaenike, 1996; Cazemajor *et al.*, 1997). Although there is no recombination between *X* and *Y* chromosomes in male Diptera, if a polymorphism initially exists for susceptible and resistant alleles at the responder locus on the *X* chromosome then the initial spread of *X*-linked meiotic drive requires restricted recombination between the drive and responder loci (either by tight linkage or inversion polymorphism on the *X*) to minimize the production of suicide combinations of driving and susceptible alleles. Inversions may also help to increase the efficiency of the drive mechanism, as suggested by the multiple inversion multifactorial drive mechanism in *D. pseudoobscura* (Wu & Bechenbach, 1983). Different mechanisms of sex-ratio distortion may be involved with sexual selection in other taxa, such as sex-reversal in poeciliid and cichlid fish (Orzack *et al.*, 1980; Seehausen *et al.*, 1999).

Here we investigate the possible connection between sexual selection and sex-ratio selection by analysing a three-locus model for the evolution of female choice

on a male character associated with sex-ratio meiotic drive. Both the drive locus and the male character locus are X-linked and the female preference is assumed to be autosomal. Reinhold *et al.* (1999) analysed a similar model with no male character locus, where female mate choice depends directly on the presence or absence of X-linked meiotic drive in males. Our model shows that restricted recombinations on the X chromosome, e.g. by a chromosomal inversion, is necessary for a meiotic drive polymorphism to promote the origin of female mating preferences and exaggerated secondary sexual characters. Our results demonstrate the importance of restricted recombination for the interaction of sex-ratio meiotic drive and sexual selection, and suggest a mechanism by which meiotic drive can act as a catalyst for the origin of female mating preferences and exaggerated male characters.

2. The model

In Diptera X-linked meiotic drive elements, X^D, typically cause driving males to produce nearly all daughters. In the absence of other selective forces, such a gene will rapidly increase in a population, causing it to become progressively more female biased, resulting in population extinction if the drive is sufficiently strong (Hamilton, 1967). With constant genotypic fitnesses, viability and fertility selection on males alone are not sufficient to maintain a polymorphism for driving and non-driving males, basically because selection on only two male genotypes cannot produce a stable equilibrium unless fitnesses are frequency dependent. A stable polymorphism for sex-ratio meiotic drive also requires selection on females, such as by a partially recessive, strongly detrimental effect of X^D in females to balance the intrinsic advantage of meiotic drive in males (Curtsinger & Feldman, 1980). In *Drosophila pseudoobscura* there is strong viability selection against X^D in both males and females (Curtsinger & Feldman, 1980). Driving males are fully fertile in single pair matings, but when females are multiply mated, driving males have a strong disadvantage in sperm competition (Wu, 1983 *a*; Jaenike, 1996). Selection against X^D by sperm competition in remated females is frequency dependent, but the magnitude of frequency dependence is rather small (Wu, 1983 *b*), so for simplicity we assume constant viability and fertility selection on each genotype at the meiotic drive locus, as in Table 2.

Inclusion of a diallelic X-linked locus with alleles b and B, affecting only male relative eye span, brings the number of X-linked genotypes to 4 in males and 10 in females including both coupling and repulsion double heterozygotes. There is no crossing-over in

males and the recombination rate between the drive locus and the B locus in females is r. Inclusion of an autosomal diallelic locus with alleles c and C affecting female mating preference based on male relative eye span, brings the total number of genotypes to 12 in males and 30 in females. There is no natural selection directly on the male character or the female preference. The fertility of a mating pair is the product of the individual male and female fertilities.

Using ordered sets of X-linked and autosomal genotypes as in Table 3, the full array of 42 three-locus zygotic genotypes can be represented in a particular order, clustering the autosomal genotypes within X-linked genotypes so that the first 12 genotypes are males and the last 30 genotypes are females.

Defining the viability and the frequency of the ith three-locus genotype as v_i and p_i respectively, the adult genotype frequencies after viability selection are:

$$p_i^* = v_i p_i \Big/ \sum_{j=1}^{42} v_j p_j. \tag{1}$$

Female choice operates through a preference depending on the male phenotype. The preference of female phenotype j for mating with male phenotype i is defined by the preference function $\psi_{ij} = \exp\{y_i z_j\}$ where the female phenotype y_i and male trait phenotype z_j are defined in Table 2. The actual mate choices made by females depend on both their preferences and the frequencies of the different male phenotypes. We assume that for each phenotype of female the probability of mating with a given male phenotype is proportional to the product of the female preference and the male frequency (Lande,

Table 2. *Viability, fertility and progeny sex ratio (proportion daughters) of* X-*linked meiotic drive genotypes, and phenotypes of* X-*linked male trait locus and autosomal female choice locus*

Drive locus	Males		Females		
Genotype	XY	$X^D Y$	XX	$X^D X$	$X^D X^D$
Viability	u_1	u_2	u_3	u_4	u_5
Fertility	m_1	m_2	m_3	m_4	m_5
Progeny sex ratio	1/2	k			
Male trait locus					
Genotype	b	B	bb	Bb	BB
Phenotype	z_1	z_2			
Female preference locus					
Genotype	$c-$	$C-$	cc	Cc	CC
Phenotype			y_1	y_2	y_3

Table 3. *Ordered genotypes of* X-*linked and autosomal loci, and ordered three-locus genotypes and their frequencies*

X-linked loci	Males				Females									
Genotype	XY b	XY B	$X^D Y$ b	$X^D Y$ B	XX $b\,b$	XX Bb	XX BB	$X^D X$ $b\ b$	$X^D X$ $b\ B$	$X^D X$ $B\ b$	$X^D X$ $B\ B$	$X^D X^D$ $b\ b$	$X^D X^D$ $B\ b$	$X^D X^D$ $B\ B$

Autosomal locus				
Genotype	cc	Cc		CC

Three-locus genotypes (first 12 male, last 30 female)

Genotype	$XY\ cc$ b	$XY\ Cc$ b	$XY\ CC$ b	...	$X^D X^D\ cc$ $B\ B$	$X^D X^D\ Cc$ $B\ B$	$X^D X^D\ CC$ $B\ B$
Frequency	p_1	p_2	p_3	...	p_{40}	p_{41}	p_{42}

For the X-linked genotypes columns represent chromosomes, with the fifth and sixth female genotypes being the coupling and repulsion linkages.

1981; Kirkpatrick, 1982). Thus the absolute mate choice

$$\psi_{ij}^* = \psi_{ij} \bigg/ \sum_{i=1}^{12} p_i^* \psi_{ij} \qquad (2)$$

guarantees that all female genotypes mate equally often on average since $\sum_{i=1}^{12} p_i^* \psi_{ij}^* = 1$ for all j. The Appendix describes the dynamics of the three-locus system.

In the figures, linkage disequilibrium is measured by its standardized value, D', ranging between -1 and $+1$. For example, between alleles X and B the absolute linkage disequilibrium is $D_{XB} = p_{XB} - p_X p_B$ where p_{XB} is the frequency of XB chromosomes and p_X and p_B are the frequencies of the X and B alleles. The standardized disequilibrium is (Lewontin, 1964)

$$D'_{XB} = \begin{cases} D_{XB}/\text{Min}[p_X(1-p_B),(1-p_X)p_B] \\ \qquad\qquad \text{if} \quad D_{XB} \geqslant 0 \\ D_{XB}/\text{Min}[p_X p_B,(1-p_X)(1-p_B)] \\ \qquad\qquad \text{if} \quad D_{XB} < 0. \end{cases} \qquad (3)$$

For convenience, all linkage disequilibria are measured in male zygotes rather than in gametes.

3. Results

As a standard to assess the influence of sex-linked meiotic drive on the evolution of the sexual selection system, we first examined the dynamics in the absence of meiotic drive. The autosomal female mating preference locus is polymorphic for a no preference (random mating) allele, c, and a low frequency of a preference allele, C. An allele increasing the male relative eye span, B, is introduced at a low frequency, simulating a new mutation arising on a single X chromosome in a small, geographically isolated population. Recall that the male character is subject to sexual selection by mate choice but no natural selection, and the female preference is selectively neutral. This model was iterated to determine the
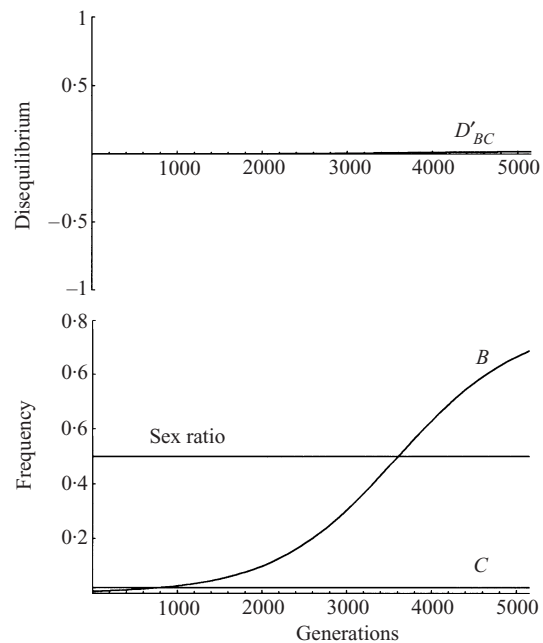

Fig. 2. Evolutionary dynamics of sexual selection in the absence of sex-ratio meiotic drive, so the sex ratio (proportion males) is 0·5. The X-linked allele increasing the male relative eye span, B, and the autosomal allele for female preference for it, C, are both initially rare. With no natural selection on B or C, the low frequency of female preference causes a gradual increase in B, with little change in the frequency of C because of the small linkage disequilibrium maintained by non-random mating with independent segregation.

evolutionary trajectories of alleles at the male trait locus and the female preference locus. Fig. 2 shows that in the absence of meiotic drive the frequency of the B allele gradually increases, but the frequency of female preference remains nearly constant. This occurs because non-random mating can maintain only a small amount of linkage disequilibrium between unlinked loci for the male character and female mating preference.
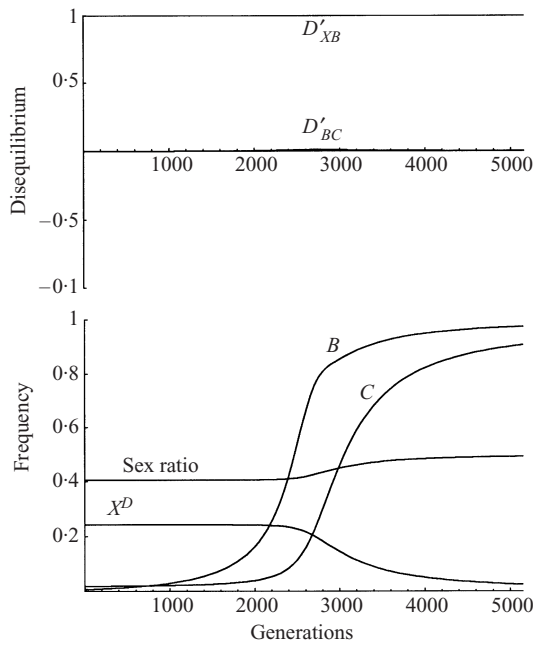
Fig. 3. The same as Fig. 2 but with a stable polymorphism for a sex-ratio meiotic drive allele X^D initially established. The B allele is introduced only on the non-driving X chromosome, with no recombination, $r = 0$. The C allele increases primarily because of sex-ratio selection favouring females that mate with non-driving males. The B allele increases mainly by sexual selection. Parameters from Table 2 are $k = 0.98$, $u = (1, 0.95, 1, 0.9, 0.5)$, $m = (1, 0.9, 1, 1, 1)$, $z = (0, 1)$, $y = (0, 0.1, 0.2)$, with no natural selection on B or C.

Fig. 4. The same as Fig. 3 but with a recombination rate $r = 0.01$ between the sex-linked loci for meiotic drive and the male character.

Now consider sex-ratio meiotic drive. The population initially is at a polymorphic equilibrium for an X-linked meiotic drive allele, X^D, with no variation at the X-linked male trait locus (and no sex dimorphism). The autosomal female mating preference locus is polymorphic for a no preference (random mating) allele and a low frequency of a preference allele. With no variation at the male trait locus, there is no basis for female choice and the population initially is random mating and in linkage equilibrium. An allele increasing the male trait is introduced at a low frequency, linked to either the X or the X^D allele, in strong linkage disequilibrium with the meiotic drive locus. The model was then iterated to determine the evolutionary trajectories of alleles at the male trait locus and the female preference locus, for different values of the recombination rate between the meiotic drive and male trait loci.

With no recombination on the sex chromosome, a rare allele for larger male relative eye span, B, initially coupled to the X allele, increases along with the female preference for it, C, until all non-driving X chromosomes contain the B allele. The major increase in frequency of C occurs by sex-ratio selection: in a population with a female biased sex ratio, females that mate with non-driving males produce more grand-
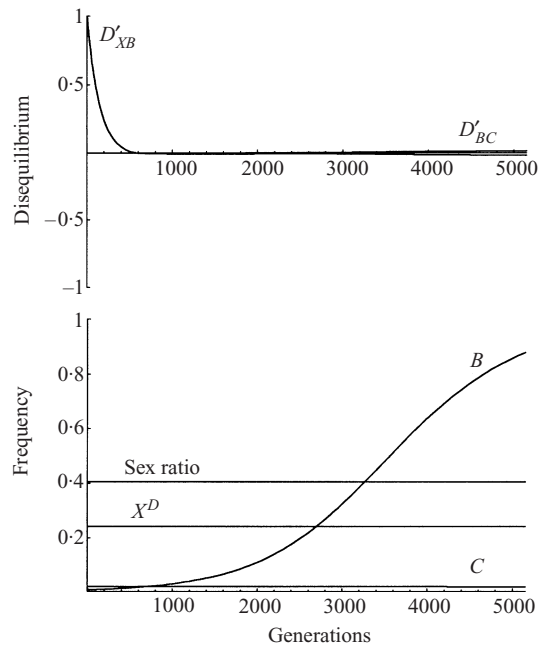
children than average because sons contribute a higher proportion of autosomal genes to the next generation than daughters (Fisher, 1958; Hamilton, 1967). The frequency of the B allele increases mainly by sexual selection. However, the female preference for non-driving XB chromosomes also decreases the equilibrium frequency of X^D. In the example of Fig. 3, allele B is eventually fixed, C becomes nearly fixed and X^D is lost from the population.

With no recombination on the sex chromosome, if the rare allele for larger male relative eye span, B, initially is coupled to X^D, the female preference for it, C, decreases until it is lost from the population, and B increases only slightly in frequency, with little transient and no permanent change in the frequency of X^D.

A small but appreciable amount of recombination, $r = 0.01$, between a rare allele for increased male relative eye span, B, and the X allele to which it is initially coupled, erodes their association before the female preference allele C can gain any significant advantage from sex-ratio selection. The gene frequency dynamics are nearly identical to those in the absence of meiotic drive (compare Figs. 4 and 2).

An extremely low rate of production of $X^D B$ chromosomes, e.g. by rare double crossovers in females heterozygous for an inverted $X^D b$ chromosome and a standard XB chromosome, or by gene conversion or mutation, produces a qualitative change in the dynamics (Fig. 5). The coupling disequilibrium between X and B persists long enough for female mating preference allele C to receive a substantial boost from sex-ratio selection. The enhanced mating
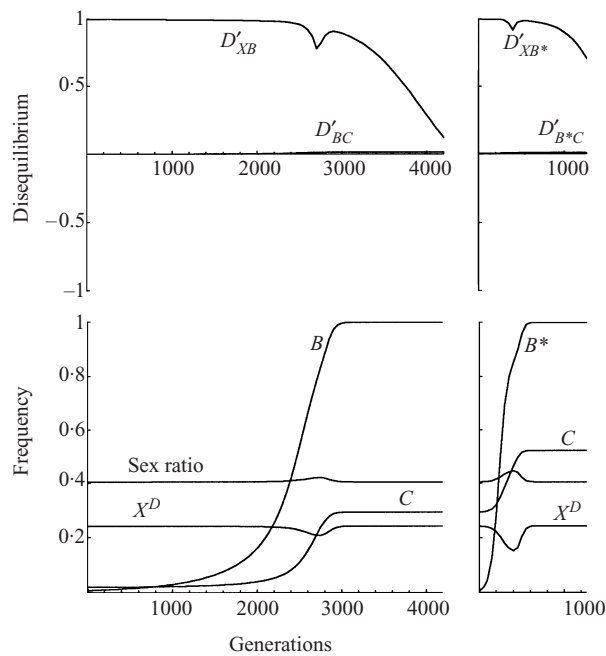
Fig. 5. The same as Fig. 3 but with a recombination rate $r = 5 \times 10^{-6}$ between the sex-linked loci for meiotic drive and the male character. Mutation B increasing the male character, initially coupled to the non-driving X, becomes fixed, female mating preference C increases substantially and the meiotic drive allele X^D is restored to its original equilibrium frequency, allowing the process to repeat (left-hand panels). Subsequently, a second mutation B^* further increasing the male character, initially coupled to the non-driving X, produces similar dynamics, but more rapidly (right-hand panels). The kinks in D'_{XB} are due to switching denominators in (3).

preference causes the initially rare $X^D B$ chromosomes to replace $X^D b$, so that the B allele becomes fixed in the population, removing the mating advantage of X over X^D, so the meiotic drive is rescued from extinction and returns to its initial equilibrium frequency, allowing the process to repeat with a new mutation increasing the male character (Fig. 5).

4. Discussion

Stable polymorphism for meiotic drive in Diptera requires tight linkage to maintain strong repulsion disequilibrium between driving and susceptible alleles at the distorter and responder loci composing the system, so that segregation distortion chromosomes do not destroy themselves (Hartl, 1974; Charlesworth & Hartl, 1978). The abundance of chromosome inversion polymorphisms, especially paracentric inversions, in Diptera (Patterson & Stone, 1952; White, 1973) therefore facilitates the origin of meiotic drive elements, and may also help to increase the efficiency of multifactorial drive mechanisms (Wu & Bechenback, 1983). These factors help to explain why sex-ratio meiotic drive is especially common in Diptera (Lyttle, 1991; Jaenike, 1996). In our model an inversion on X^D chromosomes that effectively suppresses recombination in $X^D X$ females is equivalent to no recombination of sex-linked genes ($r = 0$) since males do not recombine and only recombination in doubly heterozygous females (X-linked genotypes 5 and 6 in Table 3) alters linkage disequilibrium between the X-linked loci.

Our results show that a sex-ratio meiotic drive polymorphism with suppressed recombination greatly facilitates the establishment of autosomal female preference for increased male relative eye span associated with non-driving X chromosomes (compare Figs. 2 and 3). This occurs because suppressed recombination maintains the coupling linkage disequilibrium arising from the origin of allele B (for increased male relative eye span) by mutation on a non-driving X chromosome. The female preference allele C for mating with B males increases by sex-ratio selection: females mating with non-driving XB males leave more grandchildren than average because, in a population with a female-biased sex ratio, sons contribute a higher proportion of autosomal genes to the next generation than daughters (Fisher, 1958; Hamilton, 1967). Allele B for increased male relative eye span increases mainly through sexual selection by female mate choice.

The requirement for suppressed recombination is stringent, since a small recombination rate between sex-linked loci, $r = 0.01$, erodes the linkage disequilibrium between X and B, producing nearly the same dynamics as in the absence of meiotic drive, with little evolution of female preference (Fig. 4). With no recombination, strong female preference for males with large relative eye span could lead to loss of the meiotic drive polymorphism (see Fig. 3). However, once XB chromosomes have nearly replaced Xb, and female mating preference for B is established, $X^D B$ chromosomes could appear by rare recombination, gene conversion or mutation. Even if a paracentric inversion encompassed both the X^D and B alleles, rare recombinants can be produced by double crossovers within the inversion (Patterson & Stone, 1952; Krimbas & Powell, 1992). Gene conversion or mutation could also convert XB or $X^D b$ to $X^D B$. For many paracentric inversions in *Drosophila*, double recombination events are suppressed to the order of $r = 10^{-4}$ or less, comparable to the rates of gene conversion and mutation observed at loci encompassed by inversions (Powell, 1992). Some such event may have occurred during the evolution of sex-ratio meiotic drive elements in the sibling species *D. pseudoobscura* and *D. persimilis* because the standard non-driving X chromosomes in *D. pseudoobscura* are homosequential in salivary chromosome banding pattern to driving X^D chromosomes in *D. persimilis* (Wu & Beckenbach, 1983; Babcock & Anderson, 1996).

With strongly suppressed recombination, characteristic of that observed in *Drosophila* inversions, after alleles B and C have increased substantially, late-appearing $X^D B$ chromosomes produced by rare double recombination, gene conversion or mutation incur relatively strong sexual selection to replace $X^D b$, thereby fixing B in the population, undoing the mating advantage of X over X^D and restoring X^D to its original equilibrium frequency, allowing a similar process to occur repeatedly with new X-linked mutations increasing the male character (see Fig. 5). Thus, a sex-ratio meiotic drive polymorphism with suppressed recombination may act as a catalyst, accelerating the origin of female mating preference and exaggerated male traits, with only a transient change in the frequency of meiotic drive and sex ratio in the population. Once established, the female mating preference can further increase male relative eye span based on sex-linked and autosomal loci, as in the response to artificial selection for increased male relative eye span in *Cyrtodiopsis dalmanni* (L. Wolfenbarger & G. S. Wilkinson, unpublished data).

The present model assumes no natural selection on male eyespan or female mating preference. However, similar dynamics could occur if these assumptions were relaxed. Natural selection toward an optimum male phenotype would still permit the joint exaggeration of male eyespan and female mating preferences (Fisher, 1958; Lande, 1981; Kirkpatrick, 1982). In dimorphic species of diopsids there is ample opportunity for female mating preference as females remate frequently (Wilkinson *et al.* 1998a) and field observations on *C. dalmanni* suggest that female mate choice has little effect on female viability or fecundity (Wilkinson & Reillo, 1994). Weak selection directly on mating preference, coupled with strong sporadic sex-ratio selection via the mechanism described in our model, would nevertheless allow long-lasting evolutionary changes to occur in both male eyespan and female mating preference (Iwasa & Pomiankowski, 1995; Pomiankowski & Iwasa, 1997).

Appendix. Dynamics

The ordering of the 42 three-locus genotypes in Table 3 corresponds to a genotypic vector, g, that is the Kronecker product of the ordered vector of X-linked genotypes, $g^X = (g_1^X, g_2^X, ..., g_{14}^X)^T$, and the ordered vector of autosomal genotypes, $g^C = (g_1^C, g_2^C, g_3^C)^T$, where the superscript ^{T} denotes matrix transposition:

$$g = g^X \otimes g^C = \begin{pmatrix} g_1^X g^C \\ \vdots \\ g_{14}^X g^C \end{pmatrix}. \tag{A 1}$$

This genotypic ordering facilitates the derivation of the evolutionary dynamics.

Defining v^X as the vector of viabilities for the 14 X-linked genotypes, the full vector of viabilities for all 42 genotypes is $v = v^X \otimes (1, 1, 1)^T$.

The distribution of 42 progeny genotypes from each of the 360 possible matings can be constructed from the separate components for X-linked and autosomal genotypes, using Kronecker products to obtain the results for the whole genotype. Considering the two X-linked loci, for the ith paternal genotype in Table 3, construct a 14×10 matrix $M^X[i]$ in which each column contains the distribution of 14 progeny genotypes from a given maternal genotype (ordered as in Table 3). Considering the autosomal locus, for the jth paternal genotype in Table 3, construct a 3×3 matrix $M^C[j]$ in which each column contains the distribution of three progeny genotypes from a given maternal genotype (ordered as in Table 3). For example, with the paternal autosomal genotype Cc the progeny distribution of autosomal genotypes from each of the maternal autosomal genotypes is

$$\begin{array}{ccc} \text{Mother} & & \text{Father}:Cc \\ cc \quad Cc \quad CC & & \text{Progeny} \end{array}$$

$$M^C[2] = \begin{pmatrix} 1/2 & 1/4 & 0 \\ 1/2 & 1/2 & 1/2 \\ 0 & 1/4 & 1/2 \end{pmatrix} \begin{array}{c} cc \\ Cc \\ CC. \end{array}$$

For the three loci, the distribution of all 42 progeny genotypes from the $[3(i-1)+j]$th paternal genotype when mating with the 30 maternal genotypes is given by the 42×30 matrix

$$M[3(i-1)+j] = M^X[i] \otimes M^C[j] \quad \text{for}$$
$$i = \{1, 2, 3, 4\}, \quad j = \{1, 2, 3\}. \tag{A 2}$$

With only two male trait phenotypes and three female preference phenotypes (Table 3), the six distinct mate choice coefficients can be displayed in a 2×3 matrix from which the full 12×30 mate choice matrix can be composed:

$$\psi^* = \begin{pmatrix} 1 & 1 & 1 & 1 & 1 & 1 & 1 & 1 & 1 & 1 \\ 1 & 1 & 1 & 1 & 1 & 1 & 1 & 1 & 1 & 1 \end{pmatrix}$$

$$\otimes \begin{pmatrix} \psi_{11}^* & \psi_{12}^* & \psi_{13}^* \\ \psi_{21}^* & \psi_{22}^* & \psi_{23}^* \end{pmatrix} \otimes \begin{pmatrix} 1 \\ 1 \\ 1 \end{pmatrix}. \tag{A 3}$$

The zygotic frequencies in the progeny of the next generation (indicated by a prime) obey the recursion equation

$$p_h' = \frac{1}{\bar{w}} \sum_{i=1}^{12} f_i p_i^* \sum_{j=1}^{30} \psi_{ij}^* M[i]_{hj} f_{j+12} p_{j+12}^*, \tag{A 4}$$

where f_i is the fertility of the ith genotype and

$$\bar{w} = \sum_{i=1}^{12} f_i p_i^* \sum_{j=1}^{30} \psi_{ij}^* f_{j+12} p_{j+12}^*. \tag{A 5}$$

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